THE NATURE OF RADIOACTIVE FALL-OUT AND ITS EFFECTS ON MAN

HEARINGS

BEFORE THE

SPECIAL SUBCOMMITTEE ON RADIATION

OF THE

JOINT COMMITTEE ON ATOMIC ENERGY
CONGRESS OF THE UNITED STATES

EIGHTY-FIFTH CONGRESS

FIRST SESSION

ON

THE NATURE OF RADIOACTIVE FALLOUT AND ITS EFFECTS ON MAN

JUNE 4, 5, 6, AND 7, 1957

PART 2

Printed for the use of the Joint Committee on Atomic Energy

UNITED STATES
GOVERNMENT PRINTING OFFICE
93299° WASHINGTON : 1957

JOINT COMMITTEE ON ATOMIC ENERGY

CARL T. DURHAM, North Carolina, *Chairman*
CLINTON P. ANDERSON, New Mexico, *Vice Chairman*

CHET HOLIFIELD, California
MELVIN PRICE, Illinois
PAUL J. KILDAY, Texas
JOHN J. DEMPSEY, New Mexico
STERLING COLE, New York
JAMES E. VAN ZANDT, Pennsylvania
JAMES T. PATTERSON, Connecticut
THOMAS A. JENKINS, Ohio
RICHARD B. RUSSELL, Georgia
JOHN O. PASTORE, Rhode Island
ALBERT GORE, Tennessee
HENRY M. JACKSON, Washington
BOURKE B. HICKENLOOPER, Iowa
WILLIAM F. KNOWLAND, California
JOHN W. BRICKER, Ohio
HENRY C. DWORSHAK, Idaho

JAMES T. RAMEY, *Executive Director*

SPECIAL SUBCOMMITTEE ON RADIATION

CHET HOLIFIELD, *Chairman*

MELVIN PRICE, Illinois
STERLING COLE, New York
JAMES E. VAN ZANDT, Pennsylvania
JOHN O. PASTORE, Rhode Island
HENRY M. JACKSON, Washington
BOURKE B. HICKENLOOPER, Iowa
JOHN W. BRICKER, Ohio

CLINTON P. ANDERSON, New Mexico (*ex officio*)
CARL T. DURHAM, North Carolina (*ex officio*)

HAL HOLLISTER, *Staff Technical Adviser*
GEORGE E. BROWN, Jr., *Professional Staff Member*
PAUL C. TOMPKINS, *Consultant*

CONTENTS

(Part 1 also contains statements by leading scientists, together with statements and papers submitted for the record.)

(A detailed subject index is now being prepared and will be published at a later date.)

[1] Each witness was asked to submit a short professional biography. Most, but not all, of the witnesses did, or, as in the case of some Government witnesses, their departments did. In a few cases the biographies came from American Men of Science.

In some cases the JCAE staff has abbreviated or deleted from the biography, for brevity, no matter what the original source. These biographies are for information only and should not be regarded as a formal statement.

THE NATURE OF RADIOACTIVE FALLOUT AND ITS EFFECTS ON MAN

TUESDAY, JUNE 4, 1957

CONGRESS OF THE UNITED STATES,
SPECIAL SUBCOMMITTEE ON RADIATION OF THE
JOINT COMMITTEE ON ATOMIC ENERGY,
Washington, D. C.

The special subcommittee met, pursuant to recess, at 10 a. m., in room P-63 of the Capitol, Hon. Chet Holifield, chairman of the subcommittee, presiding.

Present: Representatives Holifield, Durham (chairman of the Joint Committee), Cole, Van Zandt; Senators Anderson, Hickenlooper, and Bricker.

Also present: Professional staff members: James T. Ramey, executive director; George E. Brown, Jr., Hal Hollister, staff technical adviser, and Paul C. Tompkins, consultant.

Representative HOLIFIELD. The committee will be in order.

This morning we continue our hearings on the effects of radiation on man, and we get into the area of genetics this morning.

Our first witness, Dr. Crow, is professor of genetics and zoology at the University of Wisconsin.

STATEMENT OF DR. JAMES F. CROW, PROFESSOR OF GENETICS AND ZOOLOGY, UNIVERSITY OF WISCONSIN [1]

Dr. CROW. Mr. Chairman and members of the committee, my intention is to summarize briefly and nontechnically the present knowledge of the genetic effects of radiations in man. I want in particular to indicate the sources of the data on which our conclusions are based, and give some idea of the reliability of quantitative estimates of genetic damage.

Previous statements before this committee have dealt mainly with effects on the person who receives the radiation. My discussion will be restricted to effects on his descendants; that is, to inherited effects.

I believe I can best summarize the general information on this question by stating four well established principles that are necessary background for any discussion of possible genetic hazards to man.

1. All high energy radiations increase the rate of mutation. A mutation is a change in the hereditary material of the cell, a change in the genes and chromosomes on which our heredity depends. An

[1] Date and place of birth: January 18, 1916, Phoenixville, Pa. Education: A. B., Friends University, 1937; Ph. D., University of Texas, 1941. Work history: Instructor and assistant professor of zoology, Dartmouth College, 1941-48; assistant professor, associate professor, and professor of genetics and zoology, University of Wisconsin, 1948; associate managing editor of Genetics, 1951-56; member of editorial board of Genetics; member of board of directors, American Society of Human Genetics; member of the council, Society for the Study of Evolution. (Submitted by witness.)

example of a multational change is that producing the bleeding disease hemophilia, characterized by abnormal blood clotting. Mutations can occur in any part of the body, but from the standpoint of heredity the important ones are those that occur in reproductive cells and are transmitted to the children. Thus, to have an effect on future generations, radiation must reach the reproductive cells sometime between conception and reproduction.

2. Almost all mutations that have been studied have been harmful.

Representative COLE. Will you repeat what you have said?

Dr. CROW. I said that almost all mutations that have been studied have been harmful.

Representative COLE. What do you mean by a "harmful" mutation?

Dr. CROW. One that in some way impairs the individual that carries this mutation. He might have his fertility reduced. He might be more prone to an early death. He might have most any kind of abnormality.

In general, most of the mutations that have been studied by geneticists have been in some way or another harmful to the individual.

Representative VAN ZANDT. After exposure to radiation?

Dr. CROW. That is right. I am talking about subsequent generations.

Representative COLE. What is a mutation?

Dr. CROW. Maybe the difficulty is that I have used mutation in two different senses. I have used the mutation as a change in the gene itself. What I should perhaps have said is that the consequence of that mutation in future generations has been harmful. I want to say, though, this is to be expected on purely theoretical grounds for the reason that a mutation is essentially a random change in an organism that is already functioning reasonably well. A random change is much more likely to be harmful than beneficial.

Consider, for example, a random rearrangement of parts in your television set. It is much more likely to make it worse than to improve its operation.

A point I want specifically to make here is that the typical mutation is not a gross abnormality.

Representative COLE. Let us be sure when you say mutation we are talking about the same change. You have indicated that you have used the word in two different concepts. To me a mutation is the consequence of some abnormality that has occurred in the ancestral genes. The result is the mutation. You have used mutation this morning as describing a damage to the parent genes themselves.

Dr. CROW. Yes.

Representative COLE. When you say mutation, let be be clear what we are talking about.

Dr. CROW. I am clear in my own mind, but whether I can make it clear in general is another question. Let me say this:

The mutation is the change that occurs in the gene, in the reproductive cell.

Representative COLE. Of the parent.

Dr. CROW. Of the parent. This changed gene is passed on to subsequent generations causing this varying amount of harm to the individuals that possess that changed gene in future generations.

Representative COLE. You mean that a changed gene inevitably passes that change on to the descendant?

Dr. Crow. That is correct.

Representative Cole. That an altered gene could not reproduce a normal child?

Dr. Crow. That is correct as far as the gene is concerned; the gene in the future that descend from the altered gene are altered.

Representative Cole. Is that always an accepted rule of genetics?

Dr. Crow. Yes.

Senator Bricker. How do you explain the fact, then, that there are always mutations going on in human regeneration, some caused, no doubt, by the normal background of radiation?

Dr. Crow. We have a certain amount of harmful mutation with us all along.

Senator Bricker. There must be some that are beneficial or there would not be the evolutionary process taking place in life.

Dr. Crow. That is correct. I did not say all were harmful. I said the great majority were harmful.

Representative Holifield. The great majority of the damaged genes are harmful, but the amount of damaged genes in relation to the total number of genes is infinitesimal, is it not?

Dr. Crow. It is small.

Let me answer Senator Bricker's point in a little detail.

Representative Cole. Before leaving this difference between infinitesimal and small; that is a tremendous area.

Dr. Crow. I agree.

Representative Cole. I would like to have you indicate on the record just what you mean by small as against infinitesimal.

Dr. Crow. Yes. Let me be a little more thorough about what you would like to know. You would like to know what proportion of the genes actually in the population are harmful versus that proportion which is beneficial?

I do not think the geneticists can answer that question in general. I do not think we have that kind of information. I can say that the proportion of sperms or eggs carrying a newly arisen mutant gene in any one generation may be of the order of 5 percent or so, perhaps as much as 15 percent.

That is what I mean by small.

Representative Cole. Newly arisen?

Dr. Crow. Yes. Newly mutated genes. By "newly" I mean having occurred in that generation.

Representative Cole. And out of that 5 percent, how many are harmful?

Dr. Crow. Practically all of them.

Representative Cole. So then your testimony is that 5 percent of the mutated genes are harmful?

Representative Holifield. Let us see if the Chair can understand.

The 5 percent that you spoke of was a rise in the rate of mutated genes. It did not refer to a 5 percent of the total number of genes. Am I right on that, or would you say that 5 percent of the genes are damaged, and the rate has risen to the point where 5 percent of the total number of genes are damaged?

Dr. Crow. I think I have confused this a great deal more than I have helped it.

Representative HOLIFIELD. No, we are the ones who have confused it, Doctor. You go ahead and straighten it out if you can. We will do our best to help you.

Senator BRICKER. Will you come to my question next?

Dr. CROW. Yes. Let me answer Senator Bricker's question.

The implication of your question is that if I say the great majority of mutants that occur are harmful, why is it that the great majority of genes that now exist in the population are beneficial?

The reason for this is natural selection. The mutant genes that have occurred in the past have been weeded out by the process of natural selection so that the genes which now are part of the normal population are those which have been retained by this process of natural selection. Therefore, even though the great majority of mutants at the time they occur are such as to cause harmful effects to the descendants, the ones which cause the most harmful effects are eliminated by natural selection. The genes left in the population are the beneficial ones.

Senator BRICKER. You would not say that all mutants are deleterious.

Dr. CROW. No. I did not say that. I said the great majority.

Senator BRICKER. There are some that are beneficial.

Dr. CROW. That is correct.

Senator BRICKER. Those are the ones naturally selected through the generations by the process of evolution.

Dr. CROW. Yes. May we go on from here? I am sure I have left the previous point in a state of considerable confusion. I think I can straighten it out as I go along.

Representative HOLIFIELD. Go ahead.

Dr. CROW. I think the things I say subsequent to this will straighten it out. So will the following speakers. I want to emphasize one point, though, and that is that the typical mutation is not a gross freak. Here I must rely largely on information from fruitflies, and this information tells us that the typical effect of a mutation is not a gross effect on the fly that can be seen but is much more likely to be something leading to the death of the fly and still more likely to be something that leads to an increased probability of early death of the fly without the certainty of it.

So the most frequent class of mutation that we can study in fruitflies is a kind that causes a slight impairment in the survival of these flies, but no obvious external effect.

I suggest by analogy that most mutations in man would produce various body impairments leading to increased susceptibility to disease, lower life expectancy, increased embryonic death rate and similar things.

All of these are occurring anyhow due to all sorts of causes and therefore it is ordinarily impossible to tell whether a particular instance of impairment or death is or is not of mutational origin.

3. My third main point is that one might perhaps think that mutations that cause only a minor impairment are unimportant, but this is not so far for the following reason: Deleterious mutant genes are eventually eliminated from the population since they generally increase the death rate or lower the fertility of the person carrying them. A mutant that causes a great deal of harm is eliminated in a few generations. But one that causes only a small amount of harm will persist much longer, and thus affect a correspondingly larger

number of persons. On the average the larger number affected by a mild mutation roughly compensates for the lesser effect on the individual.

Since minor mutations in the long run can do as much harm as more drastic ones and occur much more frequently, it follows that most of the mutational damage in a population is due to the accumulation of individual minor effects. This means that an estimate of mutational damage that considers only obvious hereditary diseases and conspicuous abnormalities is probably a gross underestimate of the total damage. The effect of minor mutations, though intangible in the sense of ordinarily being indistinguishable from the other ills that we are beset with, is probably in the aggregate much more important.

4. Evidence from experimental animals, principally Drosophila, indicates that the number of mutations produced is strictly proportional to the amount of radiation received. These are departures from this straight-line relationship at high doses, but these are too high to be likely to be encountered in any ordinary human situation. It is technically impossible to test this relationship for the very lowest doses, but the straight-line relation holds down to the smallest amounts that have been studied. Also from purely physical considerations, one would expect linearity at low doses. I have in mind the kind of analysis Dr. Pollard made yesterday.

For these reasons a simple proportionality between the amount of radiation and the number of mutations is fully accepted by geneticists.

The proportionality between dose and mutation production holds irrespective of the intensity or spacing of the dose. That is, 100 roentgens given at one dose is exactly equivalent to the same total given as a series of small doses over a long period of time. Also from the standpoint of future generations, it makes no difference whether one person receives 10 roentgens or 10 persons receive 1 roentgen; the effect on future generations is the same.

The total harm to the population, as measured by effects on future generations, is strictly proportional to the total amount of radiation received by the reproductive cells of the population.

Senator Bricker. Regardless of the number of people involved?

Dr. Crow. Yes.

Representative Holifield. This, then, would establish as far as the majority of the geneticists are concerned the principle of linear progression in deleterious effects of radiation regardless of amount?

Dr. Crow. That is correct. A nonthreshold situation, to put this in yesterday's vocabulary.

This means that there is no such thing as a safe dose of radiation to the population. Any amount of radiation, however small, that reaches the gonads—testes or ovaries—of a person who may later reproduce, involves a risk proportional to that amount. The benefits of radiation must be considered in relation to this risk.

Senator Bricker. I do not see how you distinguish that rational analysis with the radiation background that we already have.

Dr. Crow. I would say, Senator, that the mutations that we now have that are produced by earth background and cosmic rays are themselves harmful. It is something we have been putting up as a population for a great many generations. But I do not say it is good.

Senator Bricker. This process of natural selection has overcome the deleterious effects?

Dr. Crow. That is correct.

Representative Holifield. Only to the extent that it has gradually by natural selection strengthened the race rather than allowing it to become weaker and weaker. To the extent that those harmful genes did produce weakened individuals, to that extent, speaking from an individual standpoint, there was no overcoming by natural selection.

Dr. Crow. Yes. Let me amplify that a bit.

The way natural selection usually operates is through death and disease and misery and the elimination of deleterious genes has been from our standpoint a cruel process. It is our concern with minimizing this that leads us to worry about the possibility of an increased mutation rate.

I would very happy if there were some way of decreasing the spontaneous mutation rate.

Representative Holifield. Let me go further, then, and ask if there is a tendency before fertilization or immediately prior to the fertilization of the ovum for a healthy gene to mate with a healthy gene, or is the tendency for a healthy gene to mate with one that is harmed by mutation?

Dr. Crow. I think I would say that the joining of sperm and egg is purely random and the sperm and egg is ignorant of its own content as far as the probability of fertilization is concerned.

Representative Holifield. Therefore, the fact that it is a random selection at that point results in either abnormalities or weakened individuals, is that correct?

Dr. Crow. That is correct.

Representative Holifield. You may proceed.

Dr. Crow. The statements thus far made rest on a great number of experiments on a large variety of experimental organisms—for example, viruses, bacteria, fungi, corn, insects, mice—and the results from one agree with those from others.

Furthermore, there is some direct, though fragmentary evidence for radiation effects in man from study of children of radiologists and of patients who had heavy therapeutic doses of radiation. Fortunately, biological effects of radiations have been extensively studied for many years and there is a large body of well established knowledge which we can make use of in the present situation.

Let me remark, parenthetically, that it is fortunate that the development of genetics preceded the discovery of nuclear energy. Two of the men who have contributed most of this development, Doctor Sturtevant and Doctor Muller, will follow me on this stand.

It was almost exactly 30 years ago that Muller first discovered that X-rays enhance the mutation rate in fruit flies. We now have a sufficient backlog of knowledge of radiation genetics to proceed into the atomic era with due caution for possible harmful genetic effects.

I want to say a little bit about the distribution in time of the effects of mutations that might be induced at the present time.

In the first generation after radiation there would be an increase in hereditary diseases. But, as emphasized earlier, most of the harm would be of a generalized sort, not overtly distinguishable from other human weaknesses. The total damage would be spread out very thinly over many generations, with the greatest amount the first generation and slowly diminishing amounts in succeeding generations.

Senator BRICKER. Does the same thing hold true with mental development as with the physical?

Dr. CROW. As far as we know.

Senator BRICKER. The same ratio?

Dr. CROW. I think so, sir, though nobody knows for sure.

Representative VAN ZANDT. Will this hold true regardless of the amount of the dose?

Dr. CROW. Yes.

Fruit fly data suggest that the mutations would probably persist for a long enough time, that half of the total damage would occur in 30 to 50 generations. I state this to give some sort of perspective in time as to what we are dealing with. In other words, I am dealing with something that is occurring over dozens of generations and therefore thousands of years.

Despite some uncertainty as to details, we can safely say this: The genetic damage from radiation is spread over a very long time in the future, with only a small fraction appearing in the first generation.

Representative HOLIFIELD. At this pont may I ask you this: If a person is damaged by radiation it will be entirely possible for that person to have normal children and those children not be damaged by the radiation, but the damage might appear in a grandson or great grandson?

Dr. CROW. Yes; or a great-great-great-grandson. Not only is that possible, I think that is the rule.

Representative HOLIFIELD. So then we cannot draw the conclusion that a person who has received a substantial dose of radiation, even though his children are normal, that there has not been damage done to the genetic pool.

Dr. CROW. That is correct.

Senator BRICKER. There has to be some radiation effect upon the genes entering into the first generation or it does not carry on.

Dr. CROW. That is correct, too. The genes themselves are damaged but they would not necessarily cause any damage in the person.

Senator BRICKER. The personality or the physical attributes of the first generation. If those genes are not affected in the first crossing then there is no deleterious effect?

Dr. CROW. In that particular generation?

Senator BRICKER. In that lot.

Dr. CROW. It is a little different point. We have gotten confused on the same point again. An individual that received from his parents a defective gene may or may not and usually will not shown any consequence of that particular gene. At the same time he has a certain probability of the effect showing up and some one of his descendants on the average will show the effect.

Senator BRICKER. But it is only a small minority of the genes in the parent that are affected.

Dr. CROW. That is correct.

Senator BRICKER. If the particular gene entering into the first generation is not affected deleteriously there is no effect upon subsequent generations.

Dr. CROW. That is correct.

Representative HOLIFIELD. But is it not true in the case of a female that at birth the female child has all of the supply of ovum that she will have for her whole lifetime?

Dr. CROW. That is correct, too.

Representative HOLIFIELD. So therefore somewhere in that chain of ova would be a possible harmed gene.

Dr. CROW. That is correct.

Representative HOLIFIELD. That is where it would come up in future generations. It would be that transmission of the total supply to each descendant which would carry forward the mutated genes as well as the normal genes.

Dr. CROW. Yes. What may appear as a contradiction between what Senator Bricker said and what the chairman has said is this point: Each time sperm and egg are produced there is a sampling process in which some of the genes are transmitted; some others are not. That means it is perfectly possible that a harmful gene a person carries will not be transmitted to the next generation. It is just as likely by chance that it will be transmitted twice as often to the next generation. I have ignored this fact since these chance effects cancel out in the long run.

Representative VAN ZANDT. Will you describe the nature of the effects on the succeeding generations?

Dr. CROW. I think the most important thing to say of the nature of such effects is that they are not of a specific sort. They are of the same sort of ills that you and I are already beset with. Decreased life expectancy and increased susceptibility to all sorts of diseases.

I want to make the point that genetic harm is not distinguishable in any way from other kinds of harm and that is what makes our problem so difficult.

Representative VAN ZANDT. Would it have a tendency to break down the resistance of the body to various types of diseases?

Dr. CROW. I think so.

Representative HOLIFIELD. For the benefit of the lay reader and the committee, could you not give us now on the board the chronological progression of the germ so that we could understand the different steps?

Senator HICKENLOOPER. Mr. Chairman, before he gets to that very complicated situation, I wonder if I could ask just one more question about this matter, which the chairman raised a moment ago, about damage to the ovum in the female.

Assuming that there is a radiation effect which might or might not damage the ova supply, would that tend to damage all of the ova or would it be selective after a fashion and only damage some?

Dr. CROW. I would say that the radiation acts completely blindly. Which particular future egg or sperm is affected as a consequence of radiation is entirely random.

Senator HICKENLOOPER. That is purely one of hazard or chance?

Dr. CROW. Yes.

Senator HICKENLOOPER. Do I understand you to suggest that under ordinary circumstances of exposure to radiation, not all of the ova or all of the sperm would suffer damage?

Dr. CROW. Yes.

Senator HICKENLOOPER. Let me ask you this as far as a female is concerned.

Dr. CROW. Let me make one statement first. This is where my first figure that I was mentioning a while ago comes in. I would suspect that in any particular generation from spontaneous normal causes

about 5 percent, perhaps more, of the sperms or eggs produced by an individual will carry a new mutation.

Senator HICKENLOOPER. Now we get down a little further to the practicalities of the situation. I presume your statistics and figures with regard to possible mutation or change or something of the kind that might enter in there would be based on an assumption that every ovum or every egg would be fertilized.

Dr. CROW. No.

Senator HICKENLOOPER. Out of that complete fertilization a certain percentage would be bad?

Dr. CROW. No.

Senator HICKENLOOPER. Which leads me down to the question of the whole supply of ova which a female may be born with and which she may produce during a life and how many of those in the normal course of human life are actually fertilized.

Dr. CROW. Only a small fraction, as you are pointing out.

Senator HICKENLOOPER. Does that alter or change your percentage?

Dr. CROW. No, I do not think it changes a thing, Senator. The particular ovum or sperm that succeeds in being fertilized or fertilizing is a random sample of all that are produced. The numbers are very large and I assume that we can use the kind of statistical analysis that one always uses with large numbers.

Senator HICKENLOOPER. Do I understand that you are suggesting that while a very small amount, percentagewise, of the ova that are produced are fertilized, that the percentage of injury to that small percentage that would be fertilized, would that be consistent as proportionate to the whole?

Dr. CROW. That is correct. The injury among those fertilized is exactly the same as those not fertilized.

Senator HICKENLOOPER. On the whole supply?

Dr. CROW. Yes.

Senator HICKENLOOPER. Thank you.

Dr. CROW. It should be emphasized that not all spontaneous mutations are due to radiations. Part are due to natural radiations—cosmic rays, ground radiations, radioactive isotopes in the body, et cetera—but many mutations, probably the majority, have other, mostly unknown, causes.

I am saying that most mutations are probably not caused by radiation at all, including natural radiation or manmade.

Spontaneous mutations that have occurred in the past probably, directly or indirectly, account for a substantial fraction of human physical and mental impairments. One way of attempting a quantitative assessment of possible radiation damage is to compare that due to manmade radiation with that from spontaneous mutation.

If I compare spontaneous mutation rates in man for those few genes that have been carefully studied with the radiation induced rate of mutation in mice, which is the best we can do because we have no quantitative data on man, one can estimate that it would require about 50 roentgens to produce by radiation a number of mutations equal to those that occur naturally.

I shall call this, as people have previously done, a doubling dose. That is, if your genes are as mutable as those of mice it would require about 50 roentgens to produce by radiation as many mutations as occur spontaneously.

Needless to say, this is subject to a considerable uncertainty. The National Academy of Sciences Committee on Genetics estimated 30 to 50 roentgens. The British Committee reached the same conclusion.

We, that is, the members of the Academy of Science Committee, have been told that the amount of radiation due to fallout at present rates would amount to something in the vicinity of one-tenth of a roentgen in a 30-year period. I shall choose a 30-year period because that is about the average age of reproduction. From a genetic standpoint this is the important aspect of the life cycle.

If a rate of one-tenth roentgen were to continue indefinitely, and the 50-roentgen doubling dose that I previously gave is assumed to be correct, this means that the population would eventually have an increase of one five-hundredth in genetic damage. Even if my estimate of 50 roentgens is much too high, the damage would still be a small fraction of the genetic damage due to other causes. The lowest possible value for this doubling dose would be of the order of 3 to 5 roentgens. That is, this would assume that only radiation can produce mutations and therefore all spontaneous mutations are due to background radiation.

Senator BRICKER. That is not always true.

Dr. CROW. No; I do not think it is true. I am trying to set a lower limit on the frequency of natural mutation or an upper limit on the consequences of fallout.

Representative COLE. Is it possible to distinguish the source of the cause of spontaneous mutations? They are not all caused by radiation.

Dr. CROW. No.

Representative COLE. Is it possible to segregate the causes of spontaneous mutations—those attributable to radiation and those due to unknown causes?

Dr. CROW. No; it is not. In any particular event one does not know whether this mutation is caused by radiation or something else.

As I said before, ordinarily one does not know whether a particular illness is caused as a consequence of mutation or not. In no case can we trace back a particular instance of disease to a radiation effect or to other kinds of mutational effect.

Representative COLE. By experimentation you can prove that radiation or induced radiation does result in mutations.

Dr. CROW. On a statistical basis; yes. Of course, in experimental animals the evidence is much more precise than this.

If I make this minimum assumption; that is, that all mutations that occur spontaneously are due to background radiation, and the fallout rate of one-tenth roentgen per 30 years, the mutational damage would eventually be increased by about 3 percent. That is, there would be a slow rise up to a point where the present damage due to mutation, whatever that be, would increase by a factor of about 3 percent.

Representative COLE. Doctor, how can you make that assumption, that all spontaneous mutations are the result of radiation?

Dr. CROW. I am trying to set a maximum on the effect of fallout. By setting a maximum on the effect of radiation I think we can set a maximum on the amount of fallout damage.

Representative COLE. In making that assumption you admit it is fallible.

Dr. Crow. Yes. It is a very unlikely assumption. I am trying to make an extreme assumption for the purpose of setting an upper limit. So I beli that we are completely safe in concluding that fallout at the present rates will increase the existing genetic damage by only a small fraction, even if continued indefinitely.

Representative Van Zandt. A body has 50 roentgens already.

Dr. Crow. Let me say the body already has a mutation rate equivalent to that produced by 50 r. of radiation.

Representative Van Zandt. Taking into consideration the present fallout, would it be increased by 3 percent over a period of 30 years?

Dr. Crow. My best guess was not 3 percent. My best guess was 1 part in 500. It might be as much as 3 percent.

Senator Bricker. But there is nobody who believes that all the mutational changes in human life and the characteristics of body and mind are due to radiation.

Dr. Crow. I do not believe anybody believes that.

Senator Hickenlooper. Possibly do some of these mutations occur from chemical processes?

Dr. Crow. Very likely. I wish we knew the causes of all mutations but don't.

Although the general conclusions so far given are well established and there is no disagreement among geneticists, the quantitative assessment of human mutational damage is much less certain. There are no quantitatively reliable human data on radiation-induced mutation, and the figures from different experimental organisms are not in quantitative agreement. About the best we can do is to take the animal nearest man, in this case the mouse, as an indicator of possible human effects.

I think at this point there are two alternatives. One can say, as some geneticists prefer, that our quantitative data are so uncertain that we had better make no statements at all of a quantitative nature. Alternatively, we can say, as other geneticists do, that a poor numerical estimate is better than none at all. I belong to the latter group, so I shall, therefore, proceed to make some estimates as to the genetic consequences of the amounts of radiation involved in fallout.

Senator Hickenlooper. I am sorry to keep interrupting you, Doctor, but, may I ask, do some mutations have a beneficial effect occasionally?

Dr. Crow. I think a very small minority of mutations probably have a beneficial effect.

Senator Hickenlooper. We have had experience in mutations in grain where exposed to radiation, and I think the record shows that while in the overwhelming number of those mutations the progeny is less desirable than the ancestor, yet in a certain small percentage the mutations produced are in many ways far superior to the ancestry. Is that your understanding?

Dr. Crow. I think that is very likely true in man as well, Senator, but neither you nor I would suggest——

Senator Hickenlooper. I am sorry; I understand that you talked about that on the record before I came in, and I do not want to go over the same ground.

Dr. Crow. I have a sentence I want to say, anyhow. I think neither you nor I are willing to suggest that we sacrifice 999 persons with inferior mutations in order to get 1 beneficial one.

Senator HICKENLOOPER. I think we agree that is not even a 50–50, 1 horse and 1 rabbit, but I only wanted to be assured on the point that, in effect, not all mutations are bad and that if we have to have mutations there might be some modicum of benefit that would come out of it.

Dr. CROW. A very small amount.

Senator BRICKER. Would you say the percentage is 1 to 10 as to good?

Dr. CROW. No. It is much smaller than that.

Senator BRICKER. It has been proved between 5 and 10 in some of the grain experimentation.

Dr. CROW. I do not think so. I think the proportion of beneficial mutations that actually occur in grain or anything else is hardly higher than one in a thousand and may be much less than that. The reason the grainman can make use of this very rare beneficial mutation is that he can afford to throw out 999 of 1,000 grains and save the one that is superior.

Senator BRICKER. That is what they do. I thought the percentage was a little higher.

Dr. CROW. I do not think so, Senator. I think it is much lower. I do not care too much whether it is 1 percent or one-tenth of 1 percent or one one-hundredth percent. It is very small.

Senator ANDERSON. Was that the experience at Brookhaven when they were testing grain?

Dr. CROW. I do not know the exact figures.

Senator ANDERSON. Would that not be interesting to know?

Dr. CROW. I think it would be interesting, but I do not think it is too important from this particular consideration. It is very small, and that is what I am concerned about.

Representative HOLIFIELD. Is it not true that Burbank made thousands and thousands of experiments of crossbreeding in the plant field before he found a superior berry or superior fruit?

Dr. CROW. That is correct.

Representative HOLIFIELD. And that the actual cross-pollination and selection of pollen went into a thousand experiments before he found what he wanted?

Dr. CROW. That is correct. That is the way the modern plant breeder works. He grows thousands and thousands of plants, selects the one plant that shows the traits he wants, and throws all others out.

Representative HOLIFIELD. Which, again, proves the general statement that mutuations, generally speaking, are in the high proportion harmful.

Dr. CROW. That is correct.

Representative HOLIFIELD. Or degenerating rather than strengthening the strain.

Dr. CROW. That is correct.

I want to emphasize in these figures that I am about to present the large errors of measurement and the uncertainty of the assumptions involved in these estimates. To be concrete, I have prepared numerical estimates. I have prepared numerical estimates of the actual number of cases of different kinds of genetic harm that might be expected.

I shall assume a population of 2 billion children whose parents received an average of one-tenth roentgen. To repeat, one-tenth roent-

gen is roughly the amount that one might expect from fallout, according to the figures that have been published in a 30-year period. This may be too high; it may be too low. But it will give us a rough idea of the general magnitude of the risk.

Two billion is roughly the number of children that will be born in the entire world in the next generation whose parents have been exposed to fallout in this generation.

Representative VAN ZANDT. You are basing your figures on present-day test rates.

Dr. CROW. Yes. I am using the figures given to the Academy Committee and which agree with those published by Dr. Libby in his report last spring. Let me put some of these figures on the board, please. I think that is perhaps the best way to do it.

(The material on the board was as follows:)

Kind of damage	Number		Proportion of total population affected	Fraction by which existing abnormalities would be increased
	First generation only	Total for future generations		
Gross physical or mental defect	8,000	80,000	1/250,000	0.0001
Stillbirths and childhood deaths	20,000	300,000	1/100,000	
Embryonic and neonatal deaths	40,000	700,000	1/50,000	

NOTE.—Plus a larger but unknown number of minor or intangible defects.

Dr. CROW. I have written here rough guesses, which means that is what they are. I hope nobody takes the numerical values as being worth very much as numerical figures. They may be five times too high or low or more. I want only to give some rough indication, as I said before.

I am assuming that the parents had an average of one-tenth of a roentgen and there are a total of 2 billion children. I am going to give the number of various kinds of abnormalities both in the first generation and the total for all time, and I am going to give these figures in absolute figures, because these will be large, and then I am going to give them as percentage figures, and they will be small. That will make my principal point, as a matter of fact; the numbers are large, but the percentages are small.

Senator ANDERSON. Doctor, can I go back to the statement of yours a minute ago that quantitative data are so uncertain that we better make no statements at all.

Is it not possible that by taking these assumptions of yours that parents are only going to receive one-tenth and there will be 2 billion children born and so forth, that you are going to come out with some figures that are very reassuring that there is no danger in these tests, but you have no figures to back it up.

You keep making a statement that they are all right and then you say it is based on figures that do not mean anything.

Dr. CROW. I have not been sending out statements that it is all right.

Senator ANDERSON. You are about to make one as to the number of children based on assumptions. If I assume that today is Sunday I can prove it is only a 5-day week.

Dr. Crow. Let me finish my statement, Senator, and then come back.

Senator Anderson. It is the assumption that you start with. If there are 3 people up here and if 2 go the place is practically deserted, but there are more than that.

Dr. Crow. I say I have made what seem to me to be reasonable assumptions. That may be wrong. There is a high probability that numerically they are wrong. I still hold to the idea that we are better off estimating very crudely what the numbers involved here are than not making any numerical estimates at all. That is all I want to proceed with. I do not want anybody to take these more seriously than they deserve.

Representative Van Zandt. Dr. Crow, as I understand it, you are here to give us the benefit of your thinking.

Dr. Crow. Yes. I do not want to speak for geneticists as a whole or commit myself to any certainty as to actual numerical values.

The first category I want to talk about is gross genetic disease. I include in this both physical and mental disease, known or suspected to be of fairly simple genetic origin.

I would estimate something like 8,000 such cases is the first generation, and a total of some 80,000 all told. This would make up a fraction of about 1 in 250,000 in the population.

I have in mind here the kind of diseases that I referred to earlier. These may be serious mental diseases, serious physical diseases, deformities, mostly things of a rather clear hereditary origin.

In the second line we have death rates in childhood, including stillbirths. I will call this stillbirths and childhood deaths. The number I would give to this is 20,000, or a total of 300,000 for all time, making up in any one generation a fraction of about 1 in 100,000.

Then the final category I want to mention are embryonic deaths and deaths around the time of birth. I am going to call this neonatal deaths. I would guess for that 40,000 the first generation, 700,000 total, or a fraction of something like 1 in 50,000.

I have one other figure I would like to note and that is that these collectively make up about 0.0001; that is, approximately 1/10,000 of the normal incidence of these conditions.

I want to add at the bottom "Plus a larger but unknown number of minor or intangible defects."

I do not want to tell you in detail how these estimates were gotten. I would be glad if you like, to insert in the record the procedures by which there were obtained so that they could be independently checked by others.

Representative Holifield. I think you should do this.

Dr. Crow. I shall be glad to do that.

(The information referred to follows:)

Procedures by Which Estimates of Genetic Damage Were Made

These estimates ignore the effects of completely recessive factors. Such factors would have a probability of expression of much less than 1 percent in any 1 generation unless the amount of consanguineous marriage were high, or there were a high frequency of the mutant allele already in the population. The latter is not likely to be the case except for a few genes (such as thalassemia in some populations) that are maintained by some sort of selective balance. There is substantial evidence in drosophila, and some in mice and humans, that recessive genes generally have some effect as heterozygotes and that this effect is

large enough to constitute the main effect of these loci on the population. The early evidence has been summarized by Muller (American Journal on Human Genetics 2; 111–176, 1950) and there are recent confirmations. It therefore appears likely that most "recessive" mutants are eliminated as heterozygotes before they ever have an opportunity to become homozygous. My estimates are based on this assumption. I think this is reasonable as a first approximation. To the extent that the assumption is wrong the damage would be spread over hundreds rather than dozens of generations with correspondingly less effect on each, though the total effect would be about the same.

The mehods used in arriving at these estimates are explained in an article soon to appear (Eugenics Quarterly, July 1957). Briefly, they are as follows:

The number of gross physical and mental defects was obtained this way. The number of abnormalities of fairly simple genetic origin is estimated as about 2 percent of all children born (see "Genetics and Disease" by Tage Kemp, p. 190; National Academy of Sciences report, p. 25). Assuming a 50 r. doubling dose (National Academy of Sciences report, pp. 23–4; Muller, Bulletin Atomic Scientists, 11: 336, 1955; Crow, Eugenics Quarterly, 3: 201–208, 1957), 0.1 r. continued over many generations would lead at equilibrium to an increase of 0.1/50, or 0.002, in these types of abnormalities. The total effect over all time of 1 generation exposure is equal to the effect of 1 generation at equilibrium after repeated exposure. Thus, assuming a stable population of 2 billion births each generation, the total affected from one dose of 0.1 r. would be $0.02 \times 0.002 \times 2 \times 10^9$ or 80,000. The National Academy Committee suggests that about 10 percent of the damage would appear the first generation, making 8,000 out of 2 billion, or 1/250,000 of the population. The normal incidence of such conditions, genetic and nongenetic, is about 5 percent, so the 0.1 r. would cause an increase of 8,000/100 million=0.00008, or approximately 0.0001.

The estimates on stillbirths and childhood deaths are based on an estimate from the increased death rate in children of consanguineous marriages (Morton, Crow, and Muller. Proc. National Academy Sciences 42: 855–863. 1956). This estimates about 8 percent as the proportion of stillbirths and childhood deaths at mutational equilibrium. Again assuming a 50 r. doubling rose, 0.1 r. would lead to an increase of 1/500, or 0.002. In a population of 2 billion this leads to a total number of $0.08 \times 0.002 \times 2 \times 10^9 = 320{,}000$ or approximately 300,000. Drosophila data suggest that the typical "recessive" mutant has about 4 percent dominance, and making a small allowance for inclusion of dominant factors, I estimate 6 percent of the damage the first generation. Six percent of 320,000 is 19,200 or approximately 20,000. The total death rate in these ages in the populations on which these studies were made was about 12 percent, hence the effect of 0.1 r. is 20,000/240 million=0.000083 or approximately 0.0001.

The data on embryonic and neonatal deaths come from Russell's data on radiated mice. When the father had 300 r., the litter size (counted at age 3 weeks) was reduced by about 3 percent (Proc. Intern. Conf. on Peaceful Uses of Atomic Energy. 2: 382–383). If both parents received 0.1 r., the effect would be 0.2/300 as great. Hence the estimated first generation effect is $0.03 \times 0.2/300 \times 2 \times 10^9 = 39{,}600$ or approximately 40,000. Again assuming 6 percent the first generation, the total effect is 40,000/0.06=670,000 or approximately 700,000.

Dr. Crow. Let me give a rough indication.

These are based on statistical data on the frequency of such diseases and the assumption that mouse provides us a good indication as the radiation induced mutation rate. I have to use Drosophila data. The animal I am talking about here is in a sense a composite of a man, mouse, and fruitfly. To whatever the extent the information from these other organisms is relevant, these figures are reliable.

The stillbirths and childhood deaths are based on human figures for death in these ages gotten in a very indirect way that I do not want to take time to describe from the frequencies of different kinds of deaths in children of parents who were related. Once again I have to make use of fruitfly data in order to complete this estimate.

The third row of figures is based on embryonic death rates in mice in Russell's data. These are deaths that occur in mouse embryos and postnatally up to the age of 3 weeks.

Note that the second and third rows are not exclusive. Stillbirths and infant deaths are included in both groups.

I hope I have indicated that these all depend on mouse and fruitfly data and if man is different, the figures are no good. I think these are about as good an indication as one can get at this time.

Representative HOLIFIELD. There is no doubt that the mammal case would be much closer to the man than the fruitfly.

Dr. CROW. I think so.

Representative HOLIFIELD. The only reason for using, or the chief reason for using, the fruitfly is the rapid span of life.

Dr. CROW. That is correct. And the tremendous backlog of information in the fruitfly that we possess as a consequence of a great many years of work by geneticists.

Let me say in general that I have used human data when I could. When I could not, I used mouse. When neither human or mouse data is applicable, I have used fruitfly data.

I have one summarizing paragraph.

Despite the quantitative uncertainty of these estimates, I believe they have enough validity to permit some definite conclusions. One is that with the present levels of fallout, the amount of genetic damage in future generations from this cause will be a very small fraction of the total human death, disease, and misery. On the other hand, the number of persons exposed to fallout is as large as the world population, and therefore we can be sure that several hundreds, or thousands, or tens of thousands, or perhaps more persons will be diseased, or deformed, or will die prematurely, or be otherwise impaired as a consequence of fallout if the present rates of testing continue. In my opinion, even one unnecessary individual tragedy is too many, and no increase in radiation for any reason should occur unless it offers some compensating benefit for mankind.

Representative HOLIFIELD. Thank you, Dr. Crow. I am sure there will be some questions.

Senator HICKENLOOPER. Doctor Crow, perhaps you mentioned this, and I am sorry I was a little late in getting in, but did you discuss or have you studied or examined any data over a substantial period of time with regard to the Indians who live in the monazite sand district of India and the genetic data with respect to those generations that have developed in that monazite sand area?

Dr. CROW. My understanding is that a study of this is contemplated. I am not aware of any data yet coming from that.

Senator HICKENLOOPER. In the record we had yesterday, I believe Dr. Warren gave the tremendous amount of radiation activity there as compared to normal where these people have been living for no one knows how many generations. Perhaps those studies have not gone far enough to produce any results as yet.

It would seem to me that with that tremendous overplus of radiation exposure in this monazite sand area, we might get at least some significant data as to what had happened generation after generation there, if anything.

Dr. CROW. We might. On the other hand, we might not. I want to point out some of the difficulties in this kind of a study.

One is that the numbers of persons are fairly large, but they are not very large with respect to what is needed for this kind of study.

The second is that one always has some doubt when he compares one community with another community that there is not something else that is different about these two communities besides just the radiation. I think it is a difficult question.

Senator HICKENLOOPER. If we are attempting to pinpoint this for at least the purpose of these hearings on radiation, then I presume it would depend on whether there is reliable data going back a number of years or what the history of that situation is. From the evidence given yesterday that this is an area in the world where the strength of the radiation is many many times any possible radiation that might come from tests and is a number of times the radiation force of the normal background radiation on the average over the country or over the world, it would seem to me that would be a very fertile field for seeing whether mutations of significance have actually occurred there.

Dr. CROW. I certainly hope such a study will be done, but it has not been done.

Representative HOLIFIELD. Are there any further questions?

Representative COLE. Mr. Chairman, I just want to make sure my understanding of the doctor's figures is correct. That is, the figures which he placed on the blackboard.

Do those figures indicate that it is your very very rough guess that over the next generation of the 30 years of the total number of people in the world who will be born with a gross genetic disease, the number of stillbirths out of that total, 1 out of 10,000 of that gross number can be attributable to the presently produced radiation from fallout?

Dr. CROW. That is my best guess; yes. To clarify one point, I am assuming continuance for 30 years of the testing at the present rate.

Representative COLE. Based on what has occurred?

Dr. CROW. Based on the 5-year rate; yes.

Representative HOLIFIELD. These figures that you give us are based on the present rate of testing, and if that rate of testing would increase tenfold or more as it has in the past 10 years from the original rate of testing in 1946, then these figures would have to be multiplied, would they not, by the increased rate of testing? And would it be a simple multiplication or would it have more effect or less effect than the figures involved?

Dr. CROW. For all practical purposes it would be a simple multiplication. If testing were increased by tenfold each of my values here would increase by tenfold.

Representative HOLIFIELD. This is also assuming that the one-tenth roentgen is an average distribution.

Dr. CROW. Yes, sir.

Representative HOLIFIELD. If it is not average, as we have had testimony is that there is no such thing as an average distribution of fallout, but that the fallout is heavier in the temperate zone and it is uneven in the temperate zone, then there would be a higher degree of radiation received upon some groups of individuals than the one-tenth, would there not?

Dr. CROW. That is correct.

Representative HOLIFIELD. To that genetic strain possessed by those individuals, this would have to be multiplied at this time.

Dr. CROW. That is correct.

Representative HOLIFIELD. Assuming that they received a roentgen in certain areas, this would be a 10 times factor, would it not?

Dr. Crow. Yes.

Representative Holifield. And if they received 50 roentgens, it would be a 500 times factor?

Dr. Crow. Yes.

Representative Holifield. We do know that condition does exist in the world. There are peaks and valleys of distribution of fallout from the present testing rate.

Dr. Crow. Yes.

Representative Holifield. So in considering this average we should also consider the fact that it is an average and not an extreme either way, because there will be people who will receive no radiation from these tests.

Dr. Crow. Yes. I do want to say one thing, Mr. Chairman. Most of the harm from this will show up several generations in the future. By that time I suspect that within small areas the effects of peaks and valleys will in a sense have disappeared because persons from peak areas will have married descendants of persons from valley areas and the children will be mixed up by that time. On the other hand, if there are large differences in large areas, if one continent has a heavier distribution than another continent, most of the descendants of persons from one continent will stay in that continent. In this case the difference would be important.

Representative Holifield. I do not think we should leave this without pointing up that these figures again are based on peacetime testing and not upon the utilization of hundreds of weapons in a possible war.

Would you care at this time to say what you think would happen to the genetic pool of the human race if a hundred 5-megaton bombs were dropped upon any country in the world by any other nation in the world?

Dr. Crow. I have not made any such calculation, but my guess is that it would be disastrous.

Representative Holifield. That it would be disastrous?

Dr. Crow. Yes.

Representative Holifield. I am speaking about the survival. We know that a lot of strains would be eliminated immediately.

Dr. Crow. Yes.

Representative Holifield. To the survivors it would be disastrous as far as the future of mankind is concerned.

Dr. Crow. Yes. I think a major nuclear war would be a serious genetic hazard as well as a serious hazard immediately.

Representative Holifield. That is the point I wanted to bring out.

Dr. Crow. It may be, Mr. Chairman, that some of the people who will follow me have done some calculations on this point. I simply have not.

Representative Van Zandt. Dr. Crow, is this your best estimate?

Dr. Crow. Yes.

Representative Van Zandt. Have you taken into consideration the estimate of some of your colleagues in this highly specialized field?

Dr. Crow. Yes. Other people have made some of these same estimates. In particular the first estimate would agree with that one made by the National Academy of Sciences Committee.

Representative VAN ZANDT. Would you say your estimate is a general opinion of you and your colleagues?

Dr. CROW. I think so. You will have a chance to ask my colleagues later on.

Senator HICKENLOOPER. Dr. Crow, perhaps you have covered this, but have you discussed whether or not there are degrees of deleterious effects of mutations? In other words, would some mutations be by hazard or otherwise worse than others, or would some mutations merely result in a slight alteration that would in the long run be not very serious at all?

Dr. CROW. The answer is, I think, "Yes" and "No" to your two statements. Certainly mutations range from very serious to very mild. I did make the point earlier and I would like to repeat it now, that we cannot afford to ignore mild mutations because the milder mutations by virtue of being milder are less likely to cause the sterility or death of the person who possesses them and therefore more likely to persist in the population and therefore more likely to affect a larger number of persons.

So to some extent the mildness of the mutation is compensated for by the greater number of individuals it affects. For example this minor list I put at the bottom I do not regard as negligible at all. I think it may be the major part of the damage. It is so intangible that it is very difficult to measure or even to discuss.

Representative HOLIFIELD. Dr. Crow, I asked you a question a few minutes ago and it was not quite a fair question when I asked you about the number of weapons—one hundred 5-megaton weapons—so let me put it in a different way, which would possibly be more within your field of consideration.

If there was an accumulation of 500 roentgens over a period of 5 or 10 years by an average of the population, as might be expected from a massive attack such as I described, then would your answer be more explicit?

Dr. CROW. If I take the one-tenth of an r. figure that I have used here and convert that to 500 r., that means simply that every figure on this is multiplied by 5,000. I think it would be serious.

Representative VAN ZANDT. Dr. Crow, does this tear down the resistance in the body?

Dr. CROW. Yes.

Representative VAN ZANDT. I am thinking of the diseases spread over the world from time to time, like polio, and the virus situation in the Far East at the present time. Would it not add to the problem considerably from the standpoint of deaths?

Dr. CROW. Yes. I think what the nature of most genetic mutations is, as you said a few minutes ago, to lower the resistance so that the disease is more likely to be fatal than it would be otherwise.

Representative VAN ZANDT. Therefore we would be faced with additional hazards and other diseases that are taking a heavy toll today?

Dr. CROW. Yes.

Representative VAN ZANDT. It would greatly aggravate the problem.

Representative COLE. Before the doctor leaves, I am intrigued by his answer that 500 r. up in the atmosphere we would multiply all of his figures by 5,000. That would conclude that in the next 30 years, out of every 10,000 injurious effects on living persons or unborn persons, 5,000 would be due to 500 r. in the atmosphere.

Dr. CROW. One way of saying it is that I would expect that the number of such effects we now have would be increased by approximately 50 percent.

Representative COLE. What do you calculate would be the needed number of roentgens in the atmosphere to make the equation 10,000 equal 10,000? Would it be 10, or 100? It would be 1,000, would it not?

Dr. CROW. If I say one-tenth——

Representative COLE. If you say 1,000 r. is in the atmosphere then your fraction would be 10,000 out of 10,000 cases attributable to these 1,000 r.

Dr. CROW. I think that is right; 1,000 r. would, on these assumptions, about double the numbers for many generations in the future.

Representative COLE. That would be ten thousand times more radiation than presently exists?

Dr. CROW. Yes. Ten thousand times my assumed fallout level.

Representative HOLIFIELD. Thank you very much.

We are going to ask all of you gentlemen to remain until the end of today when we will have a roundtable discussion on this subject. We will do it at the end of the morning if we can. If any of the witnesses do have previous engagements they will be excused.

Our next witness is Dr. Bentley Glass. Dr. Glass is professor of biology at the Johns Hopkins University. He has a notable record. We are glad to have you with us.

STATEMENT OF DR. BENTLEY GLASS, PROFESSOR OF BIOLOGY, THE JOHNS HOPKINS UNIVERSITY [2]

Dr. GLASS. Mr. Chairman and members of the committee, like the preceding witness and those who will follow me this morning, I served as a member of the National Academy of Sciences Committee on the Genetic Effects of Radiation, and I would like to have it clear on the record that the opinions I express this morning are my own opinions and not those of the Committee as such, although I believe that the Committee would be in agreement with them.

---

[2] Department of biology, Johns Hopkins University, Baltimore, Md. Genetics. Born: Laichowfu, Shantung, China, January 17, 1906. A. B. Baylor, 1926; M. A., 1929; Ph. D. (genetics), Texas, 1932. Teacher, high school, Texas, 1926–28; teaching fellow, Baylor, 1928–29; National Research Council fellow, genetics, Oslo, Kaiser-Wilhelm Institute and Missouri, 1932–34; instructor, zoology, Stephens College, 1934–38; assistant professor biology, Goucher College, 1938–42; associate professor, 1942–46; professor, 1946–48; associate professor, Johns Hopkins, 1948–52; professor, 1952–; research associate, bureau of education, research in science, Teachers College, Columbia, 1936–37; Baltimore Rh blood typing laboratory, 1947–52; consultant, U. S. Department of State, Germany, 1950–51; governing board, Institute Biological Science, 1951–53, chairman, 1954–; assistant editor, Quarterly Review Biological, 1944–48; associate editor, 1949–; editor McCollum-Pratt Symposia, 1949–; Survey Biological Progress, 1954–; biology editor, Houghton Mifflin Co., 1946–; editorial board, Science, Science Monthly, 1948–; acting editor, 1953. Delegate International Union Biological Sciences, 1953, 1955. A. A.; Genetics Society, Society Physical Anthropology; Society Study Evolution; Human Genetics Society, Genetics of Drosophila; human genetics; history of genetics; suppressor genes; Rh blood types. (From American Men of Science, 1955.)

I hope that what I have prepared to say will answer some of the questions that have already been raised by the members of the committee, if they have not already been adequately answered to your satisfaction.

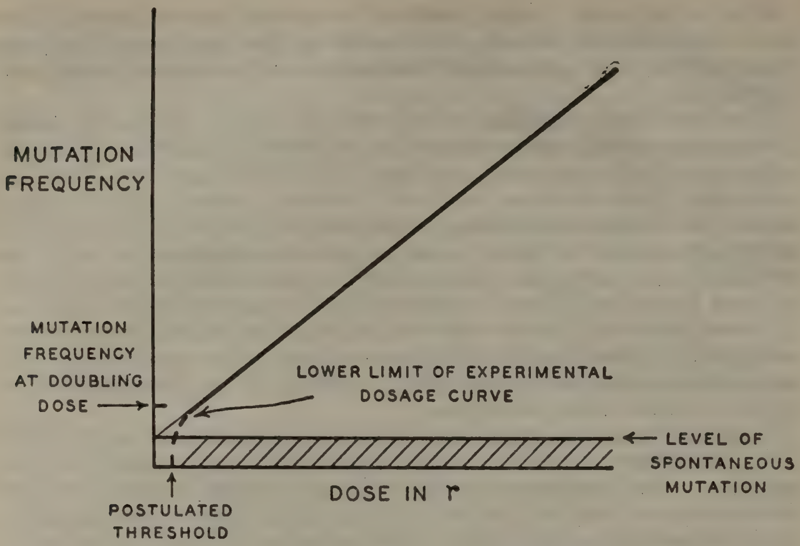
The hereditary material of all cells is located in bodies called chromosomes which are contained in the nuclei of the cells. This material is divisible into genes, perhaps 10,000, 20,000, or even 40,000 in each cell. This explains why—as I believe, Senator Hickenlooper asked—the matter that a large proportion of ova are not fertilized is not really a matter of prime concern, since we are concerned with the probability of mutation of the individual gene and there are so many of these in each cell that the probability that a mutation of some one of those genes will occur after any sizable dose of radiation is considerable. There would hardly be an ovum or a sperm cell that would not carry a mutation of some gene or other if the dose of radiation were more than a few roentgens.

Each of these genes regulates one important biochemical step in the living machinery, or controls some other significant property of protoplasm. The biochemical nature of the material is now identified as being deoxyribose nucleic acid (DNA), which forms a major part of the chromosomes. Permanent genetic changes, that is to say, mutations, consist then of modifications of the chromosomes and specifically of the DNA.

The hereditary material not only regulates the synthesis of enzymes and thus controls all life processes extrinsic to itself; it also has the unique ability to duplicate itself so that every new cell coming into existence has a replica of the genes and chromosomes of the parent cell. This at once explains why on the one hand damage done to other parts of cells can, if not too extensive, be repaired by replacing the lost or damaged machinery with newly synthesized parts, but on the contrary damage to the genes and chromosomes is irreparable. Such damage constitutes a defacement of the mold, the model, the die by means of which the new hereditary stuff is replicated, and which therefore inevitably bears the replica of every flaw in the original. Only by an exact reverse mutation may the damage done to the hereditary material by a mutation be erased. As long as it is transmitted in heredity to offspring it is permanent.

Microscopically visible fractures of chromosomes are produced by all sorts of ionizing radiations. In general, only those alterations that involve two or more breaks of chromosomes in the same nucleus, followed by reunions in new arrangements, can be inherited, for single breaks result in losses which are promptly fatal. Hence these gross mutations increase as the square or some higher power of the radiation dose.

I would like to duplicate a graph that Professor Pollard presented to the committee yesterday, plotting dose against mutations produced. We might elaborate on his diagram a bit by putting in a kind of a substratum to represent the level of spontaneous mutation. I draw it all the way across, since no matter how much of a dose of manmade radiation is applied, this would be the proportion of spontaneous mutations.



Now the gross chromosomal mutations which are microscopically visible show an increase with dosage like that first curve which Professor Pollard drew for you. These gross mutations are generally very harmful but because they occur mostly only at high doses and chiefly in spermatozoa—rather than ova—which may be eliminated if the male is rendered temporarily sterile by the high dose of the radiation, they are not very significant in the overall picture of mutation and the damage that mutation does.

Of chief importance are the so-called point mutations, in which the lesions in the hereditary material are submicroscopical in size. These increase characteristically in direct, linear proportion to the dosage, and, like the gross mutations are producible by all ionizing radiations, such as cosmic rays, gamma rays, and X-rays, and by ionizing particles such as beta particles, alpha particles, and neutrons.

These are the mutations, then, that show a linear proportionality to dosage even up to a very high dosage in all the organisms that have been studied experimentally. And although we cannot work on this region where the dose is very small the direction of that dosage curve points to this level of spontaneous mutation as its origin, which implies that even down to the very smallest doses each quantum of radiation is capable of producing a mutation.

As Professor Pollard said, it would be very difficult on any kind of physical ground to suppose that a threshold exists, say, at this level of radiation, (see figure) and you had a steeper slope to the curve right in this low dosage range than anywhere else.

Representative HOLIFIELD. Doctor, at that point, will you tell us where the amount of the dose begins in terms of roentgens?

Dr. GLASS. Yes.

Representative HOLIFIELD. I mean the amount of discernible calculation which you have in your linear field.

Dr. GLASS. The data are most extensive for the fruitfly and the lowest dose that has actually been studied is 25 r.

Representative HOLIFIELD. But the fruitfly is not as susceptible as the mammal.

Dr. GLASS. No, that is correct.

Representative HOLIFIELD. So you would have to raise that for the mouse or the man, would you not?

Senator ANDERSON. It would be lowered.

Dr. GLASS. The only part of the curve which has actually been studied—I think Dr. Russell will testify about this—is the range between 300 and 600 roentgens in the mouse. But in some plants and in bacterial studies part of this curve has been extended down to 5 roentgens or thereabouts.

Representative HOLIFIELD. In my previous question I should have said a lower dose, because I knew it took more to affect the fruitfly.

Dr. GLASS. Yes.

Because the changes in the genes are irreparable except by exact reverse mutations, the effects of doses of radiation administered at different time are cumulative; that is to say, the mutation rate is proportional to the total dose administered, irrespective of whether it is given in small doses over a long period of time or in large doses of high intensity as in the case of X-rays.

Because a mutation can be produced by a single ionization in the right place, there is no threshold below which the amount of radiation is too small to produce mutations—that is, every dose produces mutations with a probability equal to its magnitude.

This is to repeat what Dr. Crow said, that there is no safe dose of mutation. This curve continues down without any threshold until it hits the zero point at the level of the spontaneous mutation in the population. Because the genes control important, often essential biochemical steps in the living system, a mutation of any one of them is practically always harmful, and would often be lethal, were it not that each cell, being lineally descended from the union of sperm and egg at the beginning of the individual life, has two representatives of each gene, the one maternal in origin and the other paternal. We have a kind of life insurance, so to speak. Two genes of every sort, so that if something goes wrong with one of them we can still carry on. This is very important to us. It is why most of the mutations that are produced, although they would be very harmful if we had 2 doses of them, have a much milder effect when we have only 1 mutation of that kind.

Thus a mutation is often not obviously harmful at once, unless its effect is dominant over the normal gene of the same sort which is still present. But the recessive mutations are nonetheless harmful, and in many cases kill or severely handicap those persons in the population who eventually happen to inherit the same mutation from both parents. This may occur only after many generations—as Dr. Crow said, 30 or 50 generations—have elapsed since the mutation originated. Consequently, harmful mutations can accumulate and be carried in a population without visible damage until at length an equilibrium is reached at a frequency where the mutation rate that produces each particular kind of harmful gene is balanced against the elimination of that gene from the population either by the death or by the failure to reproduce of some person who gets a double dose of the gene.

There is also evidence that even in single dose most mutant genes do harm, although it is of an intangible sort, affecting general resistance, shortening the life span, or reducing the fertility of its carrier. I think Dr. Crow has spoken sufficiently to that point. From studies on the fruitfly, which are the only ones on any animal sufficiently extensive, it appears—and I believe these are the figures you were asking for a while ago—that about one-fourth of all mutations are lethal or semilethal, 15 to 20 percent produce sterility in one or both sexes, and nearly all of the remainder, whether producing visible changes or not, reduce the vitality. Less than 1 in 100 mutations—probably nearer 1 in 1,000—is definitely advantageous under existing conditions, although some of the subvital ones might become neutral or even advantageous under altered circumstances.

Two other things I would like to emphasize. First—and this is an important point which I have not seen previously emphasized in these hearings—most, if not all, of the mutations produced by radiation act as if they were truly losses of a part of the hereditary material. Some of these losses are big enough to see in the microscope. Others cannot be seen, but are probably losses, because they cannot be made to revert to the original condition. For this reason, mutations produced by radiation are probably as a class much worse in nature than those which arise spontaneously.

Secondly, there is no known way of directing mutation and of producing mutations of just the particular gene, a mutation of which may be desired. Suppose there is one mutation in a thousand that is beneficial. We cannot produce just the mutation of that one sort we desire. We have to produce a thousand mutations and pick it out. Radiation acts blindly, and that is why the deleterious nature of the vast majority of mutations is so important. By means of several hundred r. of radiation, it might indeed be possible to increase the probability of obtaining a desirable mutation in a spermatazoa or egg cell to a chance of 1 per 1,000. At the same time, the probability of getting a lethal mutation of some gene would have risen to 1 in 4, and the probability of getting a mutation with some degree of harmful effect would have become a virtual certainty. That is why we must wait for the slow processes of evolution to sort out the advantageous changes.

The breeder, of course, can produce the advantageous mutations along with the many deleterious ones and discard the latter. But in the human population we cannot do that.

It would be a grave mistake to think that mutations of the hereditary material are confined to the reproductive cells or germ line. They can unquestionably occur also in the somatic cells of any tissue—kidney, brain, liver, skin, everywhere—but at present we have too little knowledge of what consequences may follow. In case the mutations are recessive, perhaps little damage would result because of that insurance principle I spoke of.

More significant would be dominant or partially dominant effects upon essential metabolic or biochemical processes, which might as a result be impaired. The loss of damaged cells would presumably do little harm, since in most tissues undamaged cells could take their places and repair would follow. But recent suggestions made here yesterday that leukemia and shortening of the life span, when induced by radiation, may increase linearly with the dose and show

no sign of a threshold at the lower dosage rate, may imply that those effects, too, result from the induction of mutations by radiation, and their accumulation in the somatic cells.

I know that Dr. Lewis would not commit himself to that point of view, but I think he and others would agree that this is a possibility that must be recognized. Possibly cancer, in general, may arise through the same cumulative effect, which does not at all exclude the intervention of other types of agents (viruses, nutritive factors, or chemical agents) in the final outburst of malignancy.

I just want to emphasize here that contrary to what many people seem to think, mutations are not limited to the reproductive cells. They can occur to any part of the body. If they are damaging in the reproductive cells we would suppose that they were damaging in the other cells, too. It is only because the body has the ability to replace damaged cells that we can escape some of these effects.

I shall now try to appraise the current exposure of the United States population to nuclear radiations, with special reference to fallout. According to the views of most, though not all geneticists, the genetic effects of exposure to radiations can best be weighed in relation to the magnitude of the spontaneous mutation rate, which is currently responsible for a certain amount of tangible genetic defect in the population, and a certain load of wholly or partially hidden mutations in individuals who carry only a single does of any particular mutant gene.

If one could confidently assume that all spontaneous mutation was attributable to the background radiation of the environment, the problem would be fairly simple. Unfortunately, this cannot be done, since in most organisms the spontaneous mutation rate is demonstrably higher than could possibly be caused by the background. Many years ago Professor Muller, who may want to speak to this point later, pointed out that for the fruitfly not more than about one-thousandth of the spontaneous mutation could possibly be caused by the background radiation. For longer-lived animals a greater fraction may well be due to the background, since the overall mutation rate per generation in different species holds fairly constant, that is, within about one order of magnitude—10 times, say—although the exposure to background radiation increases far more than that, enormously, with length of life.

If the low-level radiation of the background in fact causes a proportionate amount of mutation, then in a species that lives a thousand times as long as the fruitfly all the spontaneous mutation would be caused by the background, and some eminent geneticists have argued that that is the case. Man lives about 365 times as long as the fruitfly, for their reproductive lifetimes are of the order of 30 days and 30 years, respectively. Thus, while it may not be very likely that for man the "doubling dose of radiation"—that is, as Dr. Crow defined it, the dose that would double the total spontaneous mutation frequency, is as small as the amount of the background radiation, it is quite possible that it may be no greater than 3 times the background, or about 10 r. The doubling dose is the dose that would raise the level of spontaneous mutation in my figure to just twice the original level. Here would be the mutation frequency caused by a doubling dose, and the doubling dose would be the corresponding dose right here [indicating

on the blackboard], that would lift the spontaneous mutation frequency to a level just twice as high.

Representative COLE. Doctor, while you are at your diagram, I do not understand why your linear line does not start at the corner of your chart, rather than upward, since there are mutations which occur from spontaneous radiation.

Dr. GLASS. Yes; it starts from the level of spontaneous mutation. This becomes effectively the corner right here. At all doses there is a certain fixed proportion of spontaneous mutation.

Representative COLE. You and Dr. Crow said that there are mutations resulting from spontaneous causes.

Dr. GLASS. Yes. Those are the mutations that are indicated in this blocked-in part at the floor of the diagram.

Representative COLE. But your diagram would indicate that mutations occur only from induced radiation and not from spontaneous.

Dr. GLASS. No; only that all mutations due to extra radiation are proportional to the added dose of radiation. For example, let us say the doubling dose represents 40 roentgens. At 40 roentgens the percentage of mutations below the horizontal line is the spontaneous frequency, and from that level up to this level is the frequency induced by the radiation. So at every dose—at large doses as well as at low doses—to the frequency of mutations induced by the radiation there must be added a small percentage of mutations that is occurring spontaneously in the population.

Senator BRICKER. Are there any data, Doctor, showing that the ratio of mutations due to background radiation is twice as much at Denver as it is at Washington, D. C.?

Dr. GLASS. No. I believe no data of that kind exist.

Senator BRICKER. You would naturally conclude that the rate would be higher?

Dr. GLASS. The increase in radiation at Denver is, of course, very small. Studies were done many years ago in an effort to see whether cosmic radiation, which is greater in amount at high elevations above sea level, produces a proportional amount of mutation. Fruitflies were placed on top of Pikes Peak and compared with fruitflies kept at sea level. This was a very large and laborious experiment, but it never added up to anything conclusive, because the difference in the amount of cosmic radiation at sea level and on top of Pikes Peak is so low in terms of roentgen units over a short period of time that you just would not get any perceptible change in the percentage of mutations.

Perhaps if the experiment could have been done with bacteria where you could work with much larger numbers, the effect could have been demonstrated. But certainly for the human population I know of no evidence at all that would show that the mutation rate varies with altitude.

Representative COLE. Doctor, would you indicate on your chart where the zero dose of radiation would occur?

Dr. GLASS. Manmade radiation? Zero would be right here [indicating on blackboard the level of spontaneous mutation].

Representative COLE. I was wondering about zero rate of radiation irrespective of source, whether spontaneous or induced, since radiation you have said does cause mutation. Therefore, mutation starts with

the occurrence of radiation. Would you indicate on your chart where is the zero of radiation irrespective of source?

Dr. GLASS. It is on this axis (the ordinate) at this particular point [indicating the intersection of the level of spontaneous mutation with the ordinate].

Representative COLE. Where is the zero mutation frequency?

Dr. GLASS. The zero mutation frequency would be at the bottom of the figure.

Representative HOLIFIELD. From radiation but not from other effects.

Dr. GLASS. From any source whatever. The zero mutation from added radiation would be approximately here [indicating the spontaneous mutation level]. It might be a little below that.

Representative COLE. Put a circle there so I can see it. Zero mutation frequency.

Dr. GLASS. Zero mutation frequency is right here.

Representative COLE. And zero radiation is there also?

Dr. GLASS. Yes.

Representative COLE. Including spontaneous radiation.

Dr. GLASS. Yes. I think the thing that is troubling is that perhaps if a sizable part of this spontaneous mutation frequency is due to background radiation, then the point at which this curve should be projected is not right here [intersection of ordinate at the level of spontaneous mutation] but at some point between the bottom and the spontaneous mutation level, at a point which corresponds to the proportion of the spontaneous mutation produced by radiation. If that is only one-thousandth of this spontaneous amount, as it is in the fruitfly, then it is so close to this point of origin that I could not draw it separately on the graph. If, on the other hand, as may be in the human species, according to Haldane's argument, perhaps all, or at least a third of the spontaneous mutation was produced by the background radiation, then the dosage curve would not point at that origin on the spontaneous mutation level, but would point at a lower origin. But we do not have the data for human population, only data from the experiments on animals.

Representative COLE. Thank you, Doctor.

Dr. GLASS. It is quite possible, then, that the amount of the doubling dose may be no more than 3 times the background or about 10 roentgens—that is, on the basis of the argument that the reproductive lifetime of man is about 365 times as long as that of the fruitfly and that a proportionally larger amount of radiation is received during that lifetime.

I have left out of this account the probability that the human genes are more sensitive to radiation than the fruitfly genes. At least we can base the argument on the analogy with the genes of the mouse. The mouse genes seem to be more sensitive to radiation than the fruitfly genes and therefore, if that should apply to human genes, it would also work toward an effective lowering of the doubling dose, the dose of radiation that would double the spontaneous radiation rate.

In the most recent estimate made by the consultants of the National Academy of Sciences committee—Drs. John Laughlin and Ira Pullman have estimated the average exposure of the population of the United States to background radiation as amounting to a dose to the reproductive organs over a 30-year period of 3.1 rem. (Cosmic radia-

tion, 0.78 r.; earth and housing, 1.59 r.; atmospheric radioactivity, 0.06 r.; internal radioactivity, mainly the beta radiation from potassium-40, 0.69 r.)

To correct something that was said yesterday, according to their data the amount from earth and housing is about double that from the cosmic radiation. This is one of the things that makes the experimental answer to the question that was posed a moment ago about the effects of cosmic rays on the mutation rate a very difficult one to work out experimentally. As Dr. Crow said, the lowest conceivable doubling dose would be 3.1 r. to the gonads over a period of 30 years.

Three times the average background would amount roughly to 10 roentgens, which is the permissible limit for the general population recommended by the National Academy Committee last year. A more probable range for the doubling dose is 30 to 50 r., but that expectation, which was adopted by both the American and the British committees reporting last year, is based on experimental evidence from fruitflies and mice, which have much shorter reproductive lives than human beings. That assumption, I feel, may not be sound.

Preliminary studies in my own laboratory with normal human cells growing in tissue culture—cells derived from kidney—and exposed to radiation of X-rays, indicate that doses of even 50 or 25 r. produce significant increases in the number of microscopically visible—gross—chromosome mutations, and from that fact it may be deduced that an even greater increase of submicroscopic mutations is to be expected.

These preliminary studies make me wonder whether our committee last year was not oversanguine in estimating that the 10 r. permissible level we recommended amounted to no more than one-third or one-fourth of the doubling dose. If it should actually constitute a doubling dose, then geneticists will certainly want to reconsider all their recommendations. For a doubling dose means that, after a lapse of generations, the frequency of all kinds of hereditary defects in the population will be doubled.

Right now this frequency amounts to at least half of all tangible defects not caused by accident or infectious disease. For example, about 5 percent of births are marked by some congenital defect, and at least 2 percent of these, according to the estimate of the National Academy Committee, are of a simple hereditary nature. Doubling just these, to say nothing of those less hereditary defects which Dr. Crow has emphasized, and those that arise later in life, would mean eventually about 2 million more defective babies per generation than now, assuming a hundred million babies per generation in the United States.

The 30-year gonadal dose from fallout amounts, according to our estimates, to about one-tenth roentgen if extrapolated on the basis of the average fallout for the past 5 years, or two-tenths roentgens at the rate of testing during the 2 most active years in that period. This is small indeed compared to the estimated background radiation or to the amount received on the average from medical and dental diagnosis and therapeutic uses of X-rays, radium, and radioisotopes, which amount to about 4.6 r. reproductive dose during a 30-year reproductive lifetime.

A safety factor here—that is, in relation to fallout—is the very fact that strontium 90 is accumulated in bones and radioiodine in the thy-

roid, and consequently they provide only a negligible amount of radiation to the gonads. We are not so sure about the localization of cesium 137, however. Nonetheless, if the gonadal dose from fallout which constitutes the genetic hazard is at the present time only 1 or 2 percent of the permissible limit for the general population, that is no reason to be complacent, in my opinion, about its rise. The increasing tempo of weapons-testing in 1957 will certainly make a reevaluation necessary.

Senator BRICKER. Is the half life of cesium 137 the same as strontium 90.

Dr. GLASS. Yes, sir, about 30 years.

Representative HOLIFIELD. It is deposited in the muscles.

Dr. GLASS. Yes, in the muscles and soft tissues, generally. It is possible that it might even concentrate in the reproductive organs. This is something that I think should be very urgently looked into in our experimental studies. I have heard a rumor to that effect. That is all I can say.

Representative HOLIFIELD. Is the power of emissions stronger than the strontium 90?

Dr. GLASS. I think it is about the same. I am not certain.

Representative VAN ZANDT. Doctor Glass, this increased tempo of weapons testing in 1957, did take into consideration the British testing?

Dr. GLASS. That is what I mean, the British and Russian testing plus our own.

Representative VAN ZANDT. Looking to 1958 and 1959, did you include any other nations participating in these tests?

Dr. GLASS. No, I do not know that any other nation is ready to begin weapons-testing, but I think that unless some international agreement to limit weapons-testing, or to eliminate it, is reached, it will be only a matter of a few years until other nations will be testing weapons, too.

Representative VAN ZANDT. In 1958–59 do you anticipate that the tempo of the schedule of 1957 tests will be stepped up?

Dr. GLASS. I certainly do, unless we can reach some international agreement.

Representative VAN ZANDT. In other words, you anticipate an annual increase of tests.

Dr. GLASS. Yes.

Senator BRICKER. The Russian tests up to the present produced about one third of the total of radiation effects through fallout.

Dr. GLASS. Others can speak about that better than I. I think that is about the correct fraction.

I must emphasize, too, that because I estimate fallout to be at present a negligible hazard to the genes we must pass on to future generations, in comparison with other factors, I by no means feel that the accumulation of strontium 90 is a negligible hazard in other respects. The evidence that has been presented to this committee would make me think otherwise. These are simply two different questions, and I am speaking to the genetic question only.

When we consider that at present our population is receiving almost half of the 10-roentgen allowance per generation from manmade sources, and that exposure is certain to increase as wastes from the development of peaceful uses of atomic energy multiply, a fivefold in-

crease in fallout from weapons testing would add to a grave problem.

There are clearly many uncertainties in the evaluation of these questions. I would therefore like to conclude by stressing something said in the final part of our National Academy of Sciences Committee report.

> The present state of advance in atomic and nuclear physics on the one hand, and in genetics on the other hand, is seriously out of balance. We badly need to know much more about genetics * * *

There are critical problems in this area about which answers are urgently needed, for example, the exact magnitude of the doubling dose.

> Our society should take prompt steps to see to it that the support of research in genetics is substantially expanded, and that it is stabilized.

I think that this remark, which I had prepared before I heard Dr. Pollard yesterday, strongly reinforces what he said to the committee.

The program of the Atomic Energy Commission exemplifies this unbalance between emphasis on the physical aspects of atomic energy and on the biological counterpart, its effects on living beings. Without wishing in any way to reflect on the competence within their own fields of our sincere and able Atomic Energy Commissioners, I think it safe to predict that this unbalance is likely to continue until the genetic and other biomedical problems and points of view are appropriately represented on the Commission itself.

Representative Holifield. Would you not say that a more desirable goal would be to have a completely independent study of this made, so that there would be no justification on the part of those making the study to attempt by the results of their study to justify an administrative policy which may or may no be correct?

Dr. Glass. That is an interesting question, Mr. Chairman. I would say that, in my own personal opinion, if at the beginning it had been set up that way, I would answer yes. But we have a going concern, and the Atomic Energy Commission has a large program of support for research in the biological sciences at the present time. I have indicated that I don't think it is by any means adequate, but it is large. I have great fear that if there was some attempt to shift all of the biological, genetic, and medical support of research in the atomic energy area to some other agency, there would be such disruption and chaos for a period of several years, that we would lag sadly, even more than now, in our program.

Representative Holifield. Any attempt that could be made within the program to achieve complete independence by freeing of such an advisory group or research group from administrative policy would be desirable, would it not?

Dr. Glass. Yes, I think so.

Representative Holifield. We had an Advisory Committee on Reactor Safeguards.

Dr. Glass. Yes.

Representative Holifield. In one instance their advice was not taken, and this is the problem that you run into with all advisory groups, that is, that their advice sometimes is not taken. I am not saying that it should be taken at all times. It should be considered in relation to the other problems which the administrator has. That is why I bring up the question.

Representative COLE. On that point, is it not true that Dr. Glass himself is a member of the Advisory Committee?

Dr. GLASS. I am a member of the Advisory Committee to the Division of Biology and Medicine of the AEC, yes.

Representative COLE. With further reference to your feeling that the program of the Commission is out of balance, it was indicated to me that the effort of the Commission in the field of biology and biological aspects of atomic energy has been neglected. I do not think you intend to imply that, do you?

Dr. GLASS. No, not neglected in an absolute sense. It is a matter of emphasis. It is a relative balance that is important.

Representative COLE. Since you are on the Advisory Committee in this field, while dollars are not a true yardstick in determining the degree of effort, could you tell us the amount which the Commission is spending annually in this field in which you think there should be greater emphasis?

Dr. GLASS. I will have to rely on my memory here. I believe it is currently around $38 million. Dr. Dunham is here.

Representative HOLIFIELD. Dr. Dunham, could you respond to the question?

Dr. DUNHAM. The question was the overall biology and medicine budget. About $31 million.

Representative COLE. That represents an effort on the part of how many individuals? I am speaking of scientists.

Dr. GLASS. I am afraid I cannot answer that.

Dr. DUNHAM. This material will be available to you by tonight. We are trying to figure that out. It is very difficult to sort out the projects and add up the number of scientific man-years.

Representative COLE. I was going to say it is my recollection, Dr. Dunham, at the beginning of these hearings you indicated of the order of 500 individuals were engaged in this research.

Dr. DUNHAM. I think it is a considerably higher figure now that we have gone back and looked at the record. We will have this for you.

Senator BRICKER. Most of this work is done by contract with the various universities and research centers.

Dr. DUNHAM. It is by contract with universities, independent laboratories and with our national laboratories.

Senator BRICKER. It would be practically impossible to know exactly the number of scientists working in the various fields, because this is determined by the local organizations.

Dr. DUNHAM. It is very difficult because you get people working part time on these projects. It will be an educated guess at best as to the figure we will come up with.

Senator ANDERSON. Did you say that the general overall biology and medical budget was $31 million?

Dr. DUNHAM. That is correct.

Senator ANDERSON. That includes a whole lot of things besides genetics.

Dr. DUNHAM. That is correct, sir.

Senator ANDERSON. Could you give us any idea when you give us a report how much money is being spent to study this question of genetics which has interested Dr. Glass and so greatly interested a great many people in the United States?

Dr. DUNHAM. This figure will be broken out separately.

Senator ANDERSON. At the same time, have you any idea how much the Atomic Energy Commission is spending in research in physics?

Dr. DUNHAM. I do not recall their exact budget this year, but it is in the $40 million or $50 million range.

(NOTE.—This figure was later checked by Dr. Dunham, and the budget for current fiscal year for the Research Division is $59,523,000 excluding cost of equipment.)

Senator ANDERSON. They are spending that much on a few reactors alone.

Dr. DUNHAM. Very definitely so. That is in a different part of the budget.

Senator ANDERSON. So we have plenty of money to find out how a reactor is going to react, but we do not have too much money to find out about genetics.

Dr. DUNHAM. It is one way to look at it.

Senator ANDERSON. It is a way that a great many mothers and fathers and even some grandfathers are interested in looking at it.

Representative VAN ZANDT. Dr. Glass, you say "appropriately represented on the Commission itself." What are your recommendations?

Dr. GLASS. I would say that the balance might be restored, possibly adequately, by one representative of the biological sciences. Perhaps a geneticist, perhaps not.

Representative VAN ZANDT. Serving as an AEC Commissioner?

Dr. GLASS. Serving as a Commissioner. I would also like to see either the present Advisory Committee to the Division of Biology and Medicine serving as a general advisory committee to the Atomic Energy Commissioners, or else I would like to see the present General Advisory Committee revised to include geneticists and biologists.

Representative VAN ZANDT. At any time during the past several years, have you made these recommendations to the Commission?

Dr. GLASS. No; I have not.

Representative HOLIFIELD. Dr. Dunham is asking for permission to speak.

Dr. DUNHAM. I would like to state that the Advisory Committee on Biology and Medicine is advisory to the Commission, and it was so established by the first Chairman of the Atomic Energy Commission.

Representative HOLIFIELD. But it does not have a geneticist or biologist on it.

Dr. DUNHAM. No. The Advisory Committee on Biology and Medicine, of which Dr. Glass is a member, is advisory to the Commission, not just to the Division of Biology and Medicine.

Dr. GLASS. This is technically correct. At the same time I think it is correct, is it not, Dr. Dunham, to say that it has a ranking somewhat lower than that of the General Advisory Committee?

Dr. DUNHAM. It is different in that the exchange of correspondence between Mr. Lilienthal and the Academy at the time the committee was set up indicated a definite wish that it be the advisory group on all biomedical problems to the Commission.

Representative COLE. That would indicate that there is no biologist on the General Advisory Committee.

Dr. DUNHAM. That is correct.

Representative COLE. But there is a special committee whose responsibility is exclusively in the field of biology.

Dr. DUNHAM. That is correct.

Representative VAN ZANDT. Mr. Chairman, may I ask Dr. Glass this question? Just what advantages would be enjoyed if we had a biologist sitting as a Commissioner on the Atomic Energy Commission?

Dr. GLASS. May I read my last three sentences which I think perhaps answer that?

Representative VAN ZANDT. All right.

Dr. GLASS. The geneticist cannot help feeling frustrated when physical scientists, religious leaders, and common people everywhere raise a loud outcry for some answer to their fears of genocide by radiation, and at the same time the available funds for research are actually in danger of reduction. Some of the research required is expensive, but in comparison with the cost of a bevatron for physical research or a nuclear reactor in the program it is of course minor. It is vital to redress the balance.

Senator ANDERSON. You say you have been on this Advisory Committee. How long have you been on it?

Dr. GLASS. Two years.

Senator ANDERSON. Dr. Dunham pointed out this is an advisory committee directly to the Atomic Energy Commission. In those 2 years, how many times have you been called before the Atomic Energy Commission to give a report of what the Advisory Committee had been doing?

Dr. GLASS. There has never been any formal call before the Commission as a whole.

Senator ANDERSON. Have you ever been called into a meeting to discuss this directly with the Atomic Energy Commission?

Dr. GLASS. Not personally, no. There have been occasions when various Commissioners—Admiral Strauss, Dr. Libby, Mr. Murray, and I remember Dr. von Neumann, too, before he became too ill—sat in with our committee for one or more sessions as individuals. I do not believe there was ever a time when more than one of them was present at the same time.

Senator ANDERSON. I thought Dr. Dunham made quite a point that this is an Advisory Committee which reports directly to the Commission. How many times have you been in with the Commission for the discussion of this problem in which so many people are interested? None at all?

Dr. GLASS. None at all.

Senator HICKENLOOPER. Have you made reports to the Commission from time to time periodically as to your recommendations and findings?

Dr. GLASS. Yes. This is what I wanted to state. Our minutes, our conclusions and recommendations are always submitted to the Atomic Energy Commissioners in writing. A full report is made and often special letters by way of report are made. This has not been neglected.

Representative COLE. How does your committee operate? Is it by meetings of individuals or by exchange of views by paper?

Dr. GLASS. By meetings. We meet regularly.

Representative COLE. During the past 2 years how many times has your committee met?

Dr. GLASS. About 12 times.

Representative COLE. That would indicate once every 3 months.

Dr. GLASS. Every 2 months, except for the summer, when we sometimes skip a meeting. There are special meetings called fairly frequently, too.

Representative VAN ZANDT. Dr. Glass, you have recommended to this committee that a biologist be appointed to the Atomic Energy Commission. Would you go a step further and recommend to President Eisenhower that one of the vacancies that exist now as far as the Commission is concerned be filled by a biologist?

Dr. GLASS. This is my personal feeling of what is wise.

Senator ANDERSON. If you get as far with your recommendation as a majority of this committee got with its suggestion, you will not get very far.

Dr. GLASS. There is no harm in trying.

Senator HICKENLOOPER. May I ask Dr. Glass if he would recommend that a physician or a medical doctor be appointed to the Commission, and if he would recommend that a physicist be appointed to the Commission? Where do we stop? One of the difficulties is that when 1 very important branch of this very ramified science is appointed to the Commission, it has a tendency to create a little friction on some of the other equally important branches involved, and there are only 5 Commissioners. So while it is desirable to have the best scientific competence available in one way or another to the Commission, I believe it was the theory of the original act that the General Advisory Committee and the other advisory groups would bring the professional competence in substantial numbers in the various fields for the benefit of the Commissioners, and it would to some extent stop the friction which occasionally unfortunately may exist between the various branches as to their relative importance. So if one starts putting one branch representative on the Commission there will literally be no stopping within any reasonable numbers as to the other branches of this science which might feel they too were equally justified in having representation.

Dr. GLASS. I realize, of course, that one cannot have representation of all the sciences and the specialized branches. I do think, however, that the biological considerations here are so important and require such urgent attention that it is advisable that the Atomic Energy Commission include at least 1 physical scientist and 1 biological scientist.

Senator ANDERSON. Doctor, the fact that there is a great deal of discussion in Japan over the testing of British bombs right now, and that the British consulate has been subjected to a little activity by students in the past few days, would indicate that there is some interest in these biological effects of the bomb, would it not?

Dr. GLASS. It certainly would.

Senator ANDERSON. And the fact that 2,000 scientists have joined in suggesting that there are some biological ramifications to this story would suggest that this is an extremely important facet. We have representation of fine men from industry on the Commission who are greatly interested in the reactor program. I think it is fine that there may be people interested in that field. One of the things which touches the hearts of the people has been the possibility that this might have some future implications for our children, and our children's children, and on down through succeeding generations. Is not that the consideration that prompts your suggestion?

Dr. GLASS. It certainly is.

Chairman DURHAM. Dr. Glass, is any substantial amount of research being carried on by any other group except the Commission at the present time in the field of genetics?

Dr. GLASS. Yes, indeed. There are considerable numbers of projects in this area which are supported by research grants from the National Science Foundation and also from the National Institutes of Health. There are some, not too many, I think, which are supported by the Office of Naval Research, because their interest is mainly physiological.

Chairman DURHAM. I thought that was true and that is why I asked the question. Could you hazard a guess as to the amount of money being spent by those outside of the Commission at the present time?

Dr. GLASS. Very roughly, I would say at the present time the Atomic Energy Commission and its Division of Biology and Medicine is supporting about half the work that is receiving governmental support in genetics. It may be even more than that.

Chairman DURHAM. Would that particularly apply to radiation?

Dr. GLASS. Much of the genetic work that is supported by the National Science Foundation of the National Institutes of Health is not concerned with radiation. If you limit it to radiation genetics, I would say that the Atomic Energy Commission is supporting at least 90 percent.

Senator HICKENLOOPER. I just wanted to ask Dr. Glass this question about the availability of competent personnel. Is there a substantial reservoir of competently trained personnel in genetics that could use greatly stepped up amounts of money over and above what is being used in total or that is devoting its time total in the country in both private and public activity?

Dr. GLASS. I think it would be a mistake to try to triple or quadruple the amount going into this area in one year or overnight. But over a period of years, a stepped-up increase could be absorbed. Perhaps the principal difficulty at the present time is in the selection and training of adequate young people in this area. They are not being attracted into it in large enough numbers because of lack of support.

Senator HICKENLOOPER. In order to get down to specific cases, suppose the Atomic Energy Commission said today we will double the amount for the next year for genetics activity; would that have a significant effect in the immediate future, or would it take a substantial period of time before you could train the personnel and get the equipment and so on to utilize that extra money? Are they available in the United States at the present time?

Dr. GLASS. It would take a rather careful survey, I believe, to answer that question precisely. I am not sure that doubling it in 1 year would be at all advisable. But there are certainly some large programs which would require a large annual budget which could be initiated then and that now cannot be attempted. I believe that an increase by 50 percent could probably be absorbed in 1 year.

Senator BRICKER. Is it not true that there are many, many programs beyond those which are being conducted by the Government, either by the Atomic Energy Commission or defense research or the National Science Foundation, by private institutions in the various universities of the country?

Dr. GLASS. There is a lot of genetics of that kind going on, yes. As I said a moment ago, I believe about 90 percent of the genetic

problems relating to radiation effects is being supported by the Atomic Energy Commission.

Representative COLE. Mr. Chairman, in response to a question by Senator Anderson, Dr. Glass indicated that these activities in Japan at the British Embassy indicated an interest by the Japanese people in the biomedical aspects of the weapons testing. Similar activities occurred at the American consulate 1 or 2 weeks ago. If that was the interest which inspired these people to become active at those two consulates, why have they been silent with respect to the testing by the Russians?

Dr. GLASS. I do not know that they have been silent. Have they?

Representative COLE. So far as I am aware, there has been no appreciable degree of representation in front of the Russian Embassy in Tokyo as there has been with the American and British.

Dr. GLASS. That may be true. I know that at the time of the last Russian tests, when fallout occurred over Japan, there was a considerable stir and a good deal of reporting of the Japanese concern about this in our newspapers. I do not know that it took the form of a vocal demonstration.

Representative COLE. There must be some other motiviation on the part of the Japanese than interest in the biomedical aspects which prompts them to voice themselves with respect to weapons testing.

Dr. GLASS. Yes, I am sure there must be political motives involved in this.

Senator ANDERSON. We all recognize that the Russians may have done a little better propaganda job than we have. They have offered to stop tests over and over again. We do not like the circumstances under which they offered it. That may be one of the things which led the Japanese people to believe they will stop the tests. It is that the British and ourselves will not stop for all practical purposes, so we have gotten ourselves in a bad position internationally.

Representative COLE. I would like to have Dr. Glass indicate for the record the extent, if any, to which the genetic aspect is under constant survey by the United Nations Committee on the Radioactive Hazard.

Dr. GLASS. It is a very important part of that study. The United Nations Committee, of which Dr. Shields Warren is the Chairman, I believe, has met repeatedly over the course of the last 2 years to consider these problems. I am confident that our own Atomic Energy Commission has given it every degree of cooperation possible in supplying whatever data were requested.

Senator HICKENLOOPER. Mr. Chairman, may I ask Dr. Glass one more question?

Representative HOLIFIELD. The Chair would like to make a statement at this time.

I am sure every member of the committee thinks that this problem is so important that we are not going to curtail questioning of witnesses even though it disturbs our schedule. I hope the members will agree with me on that. This is a very important part of our hearings, and they should not be curtailed in any way as long as we have valuable testimony to be given or questions which the members wish to ask.

Senator HICKENLOOPER. Dr. Glass, this occurred to me a while ago when you were talking about desirable mutations. If a desirable mutation is had, do the desirable qualities of that mutation continue in the succeeding generations, or do they deteriorate?

Dr. GLASS. They would be just as permanent, we think, as the effects of a harmful mutation.

Senator HICKENLOOPER. So that once a desirable mutation is acquired, its characteristics would probably continue.

Dr. GLASS. Would be transmitted indefinitely; yes.

Chairman DURHAM. By radiation, you are speaking?

Dr. GLASS. Yes. There was a big "if" there, if I may answer your question, because I did earlier stress the fact that as a class of mutations produced by radiation are much more likely to be losses of the genetic material than are mutations that occur spontaneously. Consequently, I think the probability that a desirable mutation will occur as a result of radiation is even less than the probability that it might arise spontaneously.

Senator BRICKER. I do not think there is a member of this committee or anyone advised that does not realize that there is political propaganda being utilized by the Communists and the left wing groups generally in this field, and we may be giving some credence to it. On the other hand, it is an essential investigation that must be made. I think the public is entitled to all the information they can get.

Do you, as a scientist, or does your group of advisers, have any information as to what Russia is doing in this field of biological effects?

Dr. GLASS. We have very little information with regard to the state of genetics in Russia at the present time. I think Professor Muller would be better qualified to answer that question than I am. Perhaps I should postpone it.

Representative HOLIFIELD. We want to give Professor Muller plenty of time. When we get him on the stand we will ask him to testify on this point.

Thank you very much, Dr. Glass, for your very fine presentation this morning. I hope you will be able to stay for the roundtable conference, whenever we have it. I am afraid we will not be able to get at it at noon. We would like to put on this morning another notable, Dr. A. H. Sturtevant from the California Institute of Technology. Dr. Sturtevant, we are happy to have you with us this morning. You may proceed.

STATEMENT OF DR. A. H. STURTEVANT, PROFESSOR OF GENETICS, CALIFORNIA INSTITUTE OF TECHNOLOGY [3]

Dr. STURTEVANT. Mr. Chairman, the nature of the genetic effects and the quantitative estimates have been presented by Dr. Crow and Dr. Glass, so I shall not go over that ground. There is one point

[3] Date of birth: 1891. Education: Bachelor of arts, Columbia University, 1912; doctor of philosophy, 1914; honorary doctor of science, Princeton, Pennsylvania, Yale. Thomas Hunt Morgan professor of genetics, California Institute of Technology. Past president, American Society of Zoologists, Genetics Society of America, Pacific division, American Association for the the Advancement of Science. Kimber Medal for Genetics, National Academy of Sciences. Member, National Academy of Sciences Committee on the Genetic Effects of Atomic Radiation. Member, American Philosophical Society, National Academy of Sciences. (Submitted by witness.)

I should like to indicate, however, that sometimes has led to some confusion.

If there is an increase in the amount of radiation, within the range with which we are here concerned, there will be more mutations induced, but there will not be more serious mutations. Individually they will have the same range of effect as will those which were produced by the smaller amount of radiation.

Many geneticists have been disturbed by statements from the Atomic Energy Commission implying that there is no reason for any concern about possible damage to man arising from bomb tests. Recent statements by Commissioner Libby suggest that there is now an area of agreement on which discussion may be based. We are agreed that there is at least a possibility of damage to a small percentage of the population. We are, I think, also agreed that it is not now possible to present very exact estimates as to just how small that percentage is; and, further, that it is important to make studies that will improve the accuracy of such estimates.

Such efforts to improve the estimates must come largely from further research, both on the physical side (as to the nature and distribution of fallout), and from the biological side (as to the genetic and pathological effects of fallout). I should like here to express my opinion that, at least in the field of genetics—which is where I am competent to judge—the AEC has sponsored an excellent program, both in its own laboratories and in its grants to other laboratories. These projects seem to me to have been well chosen and well administered. In particular, there is, so far as I know, no pressure for the expression of conclusions or opinions that may be agreeable to the AEC.

I would like to add here that I should not like this statement read to mean that I feel that more money could not be used effectively. In other words, I am not in general in disagreement with Dr. Glass' plea for more support in this field.

However, there remain areas in which some geneticists are still disturbed by the AEC position. This, as has often been pointed out, arises in part from a difference in attitude concerning very small percentages. Various methods of calculating damage from fallout result in estimated numbers of affected individuals ranging from a few hundred to perhaps tens of thousands or even millions, depending in part on what assumptions one makes about the rate of future bomb testing. The highest of these numbers remains a very small proportion of the total population, and to some people this means that it is relatively unimportant. It is probably unimportant for the survival of the race, and it is relatively unimportant as an economic burden to society, though these could become serious matters if the rate of fallout should increase by a large amount. But hundreds or thousands or tens of thousands or more of individual human beings are involved, and to me it is not acceptable to say that they are unimportant, no matter how small a percentage of the total they make up.

Another point that some of us find disturbing is the insistence that the risk from fallout is much less than risks that we voluntarily

take repeatedly—such as those involved in riding in an automobile or going for a swim at the beach. While the risk is less from fallout, the essential point is that it is one over which the individual has no control. It has been argued that the risk from fallout is not very different from that of wearing a wristwatch with a radium painted dial. Even if this comparison is accurate, it still leaves out of account the fact that some of us do not wear such watches, and would complain loudly if anyone tried to insist that we and our children must do so.

There are biological risks from bomb testing. They are small—so small that no individual should be seriously concerned about the danger to himself or his immediate descendants. Nevertheless, when the entire population of the United States, or of the world, is exposed, it must be expected that there will be many individuals damaged, both in the exposed generation and in following ones.

I should like to lay special emphasis on the fact that every bomb test adds to the biological hazard. It follows that the most effective way to reduce future hazards would be not to test any more bombs. A geneticist cannot assume special competence in evaluating all the arguments for or against such a cessation, since the decision has to take into account many factors that lie outside his special field. But I should like to point out that biologists have made a serious effort to present an objective statement of the biological hazards, and it therefore seems to me reasonable to ask for as detailed and objective a statement of the reasons for continued testing as is consistent with security considerations. Some physicists have questioned the desirability of testing, on the ground that it gives almost as much information to other nations as it does to those making the test. Many nonscientists in this and other countries have argued that the tests worsen rather than improve, the international situation. These arguments, and those of the biologists, have never been publicly discussed seriously and in detail by those responsible for carrying out the tests.

Representative HOLIFIELD. Thank you very much, Dr. Sturtevant. Are there any questions?

Senator BRICKER. Of course, it would be impossible for us to do it unilaterally in light of what Russia is doing at the present time, and that may be the reason for your last conclusion.

Dr. STURTEVANT. That will have the same effect.

Senator BRICKER. That leads to your last conclusion that that is the reason there has not been serious discussion of cessation until we can get the international situation solved. Until that is done, there can be no effective steps taken by the United States alone.

Dr. STURTEVANT. I think the discussion should be as frank as possible in view of the security considerations involved. I was careful to add that.

Senator ANDERSON. Did you hear the statement that Dr. Langham made the other day when we were considering the possibility of limiting tests in some fashion where he suggested that we set a sort of ceiling of ten megatons of fission products as a reasonable limit of how much might be put in the atmosphere each year. Would you feel that something of that general nature is desirable until we are able to reach

some other step in this process. In other words, I think his thought was if we could limit to maybe 10 megatons the amount of fission products going into the atmosphere, there is a certain deterioration of strontium 90 and the decay of that material, and therefore we should not be putting more into the atmosphere than might decay out. Do you think it would be desirable to have some sort of limitation of that nature?

Dr. STURTEVANT. Speaking as a geneticist, anything which will decrease the amount is desirable. Speaking as a citizen, I realize that there are other considerations that have to be balanced against this.

Senator ANDERSON. His statement was based on some sort of international agreement. It was not to be done unilaterally by this country. He did feel it was desirable to make sure no more was going into the atmosphere than was falling out of the atmosphere each year. I think geneticists generally are in sympathy with that.

Dr. STURTEVANT. I would prefer to see less going in than was coming out, if possible.

Senator ANDERSON. I think he would, too.

Representative VAN ZANDT. Dr. Sturtevant, in the closing part of your statement, you say these arguments and those of the biologists have never been discussed in public seriously with those responsible for carrying out the tests. Would you recommend a public forum for this discussion, keeping it within the bounds of security?

Dr. STURTEVANT. That has been suggested recently as a possibility. I would rather see myself a statement by people who were responsible for it. In other words, by the AEC.

Representative VAN ZANDT. These hearings should constitute a forum where the biologists and those responsible for carrying out the tests can sit down and discuss the overall problem.

Dr. STURTEVANT. I think that would be very desirable.

Representative HOLIFIELD. Are there any further questions? If not, we will recess at this time until 2 o'clock.

(Thereupon at 12:35 p. m. a recess was taken until 2 p. m., the same day.)

AFTERNOON SESSION

Representative HOLIFIELD. The committee will be in order.

We are honored to have before us this afternoon Dr. Muller from the University of Indiana. It is my pleasure to announce to our audience today that Dr. Muller is a Nobel Prize winner.

As the Chair looks around and sees the group of children on our left here and realizes the importance of this testimony to them and future generations, it brings to my mind the gravity of the purpose of this committee, which is to bring out the scientific facts in this field.

Dr. Muller, you may proceed.

STATEMENT OF DR. HERMANN J. MULLER, PROFESSOR OF ZOOLOGY, UNIVERSITY OF INDIANA [4]

Dr. MULLER. Thank you, Mr. Chairman.

The subject of the genetic effects of radiation is, in my opinion, so closely bound up with that of the effects on the exposed individual himself that I feel it necessary, to begin with, to touch on that subject first.

I feel this is especially necessary because there has been a curious official silence concerning findings showing that the main damage to the exposed individuals themselves by small or moderate exposures to radioactive substances or X-rays consists of an insidious weakening of the body's resistance to the onset of infirmities and diseases of all kinds, expressing itself in a shortening of the length of life, and also consists in a long delayed production of certain specific disorders of which the most important are leukemia and some other malignant conditions.

Still less publicized has been the increasing evidence that the amount of these effects is simply proportionate to the total dose of radiation received, even when this has been given in tiny bits scattered over long periods. That is evidence for the conclusion that there is no threshold, some of which evidence you heard presented by Dr. Lewis.

I may say that I first heard of this principle of the shortening of life in amounts proportionate to the total dose received, no matter in how many small bits, in a very scholarly address given by Dr. Robert D. Boche at Oak Ridge in April 1948, and again at the Argonne Laboratories in November 1948. These were not classified meetings. The principle that he explained to them was based on work done by a number of different investigators during the war, working mainly at Rochester, N. Y., under the Manhattan project.

I mentioned these conclusions of Boche in a series of lectures given under the auspices of the Society for Sigma Xi, that were published 2 years later. But I was unable to refer to Dr. Boche's work except as addresses because I waited in vain to see publications along those lines.

Representative HOLIFIELD. Where were these addresses given?

Dr. MULLER. One was at Oak Ridge and the other at Argonne. They were open meetings. That is to say, it was not classified. They

---

[4] Date and place of birth: December 21, 1890, New York City, N. Y. Education: A. B., Columbia University, 1910; M. A. (1911), Ph. D. in zoology, physiology and biochemistry, (1916); D. Sc., Edinburgh University, 1940. Work history: Teaching fellow in physiology at Cornell Medical College, 1911–12; assistant in zoology at Columbia University, 1912–15; instructor in biology, Rice Institute 1915–18; instructor in zoology, Columbia University, 1918–20; associate professor (1920–25); professor of zoology, University of Texas, 1920–36; guest investigator (1937–38); lecturer in animal genetics, Institute of Animal Genetics, Edinburgh University, 1937–40; resident associate (1940–42); visiting professor of biology, Amherst College, 1940–45; professor, (1945–53); distinguished service professor of zoology, Indiana University, since 1945. Subsidiary appointments: Guggenheim Memorial Foundation fellow at Institut fur Hirnforschung, Berlin, 1932–33; senior geneticist, Institute of Genetics of Academy of Sciences of U. S. S. R., Leningrad and Moscow, 1933–37; civilian consultant to MED, 1943–44, to AEC, 1946–48; member of National Aiademy of Science, American Association of Advanced Science (fellow), American Society of Naturalists (vice president, 1932; president, 1943), American Philosophical Society, American Academy of Arts and Sciences, American Society of Zoologists, Genetics Society of America (president, 1947), American Genetics Association, Society for Study of Evolution, American Society of Human Genetics, Society of Experimental Biology and Medicine, American Humanist Association (president, 1956–57), Sigma Xi, Phi Beta Kappa, Alpha Epsilon Delta. Honors received: Annual $1,000 award of American Association of Advanced Scientists, 1937; Nobel Prize in physiology and medicine, 1946, for the production of mutations by radiation; presidency of Eighth International Congress of Genetics, Stockholm, 1948; honorary D. Sc. from Columbia University, 1949; Kimber award in genetics of National Academy of Sciences, 1955; Virchow medal of Rudolph Virchow Medical Society of New York, 1956. (Submitted by witness.)

were not published and they were not open to the press so far as I know, so I only knew about the matter verbally.

Representative HOLIFIELD. Were they in the form of prepared addresses?

Dr. MULLER. Prepared addresses, and with charts of data; yes.

Representative HOLIFIELD. Who did you expect to print those?

Dr. MULLER. I expected he would have an article on the subject. Later I found that there was a classified article of his in 1946 on the subject. It was later declassified, but I am not sure when. It did not get out into the general literature. It was not spoken about to any extent. In fact, although I do not know whether it had any relation to that silence or not, there was an attempt made to prevent the publication of that part of my own lecture which contained this material. No special reason was given. I do not want to go into the details of that because it may have been accidental, but it is a very curious incident.

Representative HOLIFIELD. You said there was an attempt made?

Dr. MULLER. The editor had agreed to publish the whole article. It was presented in 2 parts because it was too long to go in as 1 article. Both parts were to have been published. After the one part had been published I was told that they did not want to publish the other part, that that was enough. The first part concerned itself with the effects on future generations and the second part with the effects on the exposed generation itself.

Representative COLE. Mr. Chairman, would Dr. Muller give the time when this incident occurred?

Dr. MULLER. The lectures were given in 1948. They were published first in 1950. So that occurred in 1950.

Representative VAN ZANDT. Dr. Muller, did the thinking at that time fit in with the problem of today?

Dr. MULLER. Yes. It was not as acute because we did not have hydrogen bombs yet.

Representative VAN ZANDT. Were there any predictions in those papers?

Dr. MULLER. I called attention to these points; yes. We did not know much about fallout yet.

Representative HOLIFIELD. Dr. Muller, what type of a meeting was this? Who set the meeting up, and where was it?

Dr. MULLER. One of them at Oak Ridge was set up by the Director of Research, Dr. Hollaender, a very eminent biologist and geneticist. The other was called under the auspices of the Argonne Laboratory on the problem of the effects of low doses of radiation.

There were many interesting papers there. For example, one which showed the lessening of the number of white blood cells in workers at Los Alamos who had received only 25 r. of radiation over the whole year. Nevertheless, by taking accurate statistics of the whole group it was possible to show the effect. One would not have been able to see it in one individual because it fluctuates too much.

Representative COLE. Mr. Chairman, I am curious to know, Dr. Muller, is it your feeling that there was a deliberate attempt on the part of the responsible officials of the Government, whether of the Atomic Energy Commission or the Manhattan Engineering District, to suppress the expression of your views on this subject?

Dr. MULLER. I would not like to express an opinion about that. It may have been a coincidence. But I was very surprised that no one else said anything about the matter in any semipopular or popular publication. This was a semipopular publication.

Representative VAN ZANDT. Dr. Muller, do you feel that had the papers been made public at that time that it would have eased this acute problem we are faced with today?

Dr. MULLER. Yes. I think that any such knowledge is to the good because the public should be prepared to know the facts and if they find out later that something has been withheld from them—I will touch on that later—they are apt to exaggerate the importance of the facts. I think that there was a feeling probably amongst many people who felt that these facts needed further verification, that it was unwise to let the public know about them until they were proved up to the hilt.

To me it seemed that the main principles were already far enough established to warrant a public airing, since if true, they were of such grave concern.

Senator ANDERSON. Doctor, this morning there was some reference made to Russia, and it was suggested that one of the witnesses testify about the situation in Russia. Here we have the situation in this country today where the foremost geneticists of the country are testifying before an open hearing. Is that the situation in Russia, or are geneticists in somewhat different circumstances over there?

Dr. MULLER. There are few geneticists left. I would rather postpone questions on Russia until we get through this because we might go on indefinitely. I think we can draw lessons from Russia.

Senator ANDERSON. I knew you had worked in Russia.

Dr. MULLER. Yes.

Senator ANDERSON. And therefore have some opinions. I hope you give them when you finish this afternoon.

Dr. MULLER. I would be happy to answer to the best of my ability any such questions as you might have.

These facts, which I think Dr. Hardin Jones and Dr. Russell will take up in their talks, about the shortening of the length of life seems to me to be extremely important. I think it can be shown that they and the long delayed malignancies such as leukemia constitute by far the greatest damage of all the effects of radiation on the exposed individual himself. That is, of moderate and small doses. I am not speaking of the radiation sickness and death that occurs from very high exposures such as direct atomic bombing.

Therefore, I thought it was very curious that these effects were not discussed more and were not in fact investigated more. It seemed to me if there was an uncertainty then it should have been pushed to find out is it true or not.

We did finally in 1954 have papers on this work published in declassified form in a highly technical volume edited by Dr. Blair almost 10 years later. Since then the effects have been talked about to some extent. They were mentioned in the Pathology Committee report of the National Academy of Sciences. So far as I can remember in the National Committee on Radiation Protection—I happened to belong to its Subcommittee on External Dose for some years—there was very little mention of them. I doubt very much that they were gen-

erally realized by workers in the field of radiology, for example, or by biologists in general.

In respect to the fact that probably there is no threshold, that these effects are proportional to the dose, in this respect these effects of radiation—and also the leukemia—on the exposed individual himself resemble those produced by the radiation in weakening descendants.

You have heard Dr. Glass and Dr. Crow say that geneticists are convinced that there is no threshold for the genetic effects and that others, too, now accept that principle for the genetic effects.

If this is true of these other effects, and it is certainly time we knew whether it was—I think the evidence is convincing that it is—then this important resemblance between the effects on later generations and on the exposed generation is probably not an accidental resemblance. For there is growing reason to infer that this shortening of life and the other long delayed damage done to an exposed individual have their basis in damage done to the genetic material—the chromosomes and their contained genes—of the body's ordinary cells, those of the blood, skin, glands, and so forth, similar to the damage done in his reproductive cells that is passed on to later generations.

In the body's own cells, however, we have good reason to infer that more of the damage that becomes expressed is done by breaks of chromosomes and in the reproductive cells by mutations of genes.

Let me use the blackboard a moment.

Here is a cell, and there is its nucleus, and in it there are these long threadlike bodies which can become condensed into sausage-shaped forms, called chromosomes, that consist essentially of strings of minute particles called genes, each of which, as Dr. Glass explained to you, has some specific effect in furthering the functioning of the body.

Radiation has two main kinds of effects on this genetic material. Let me again say that every cell of the trillions of cells in the body has this genetic material the same as the reproductive cells, and radiation can affect the genetic material in the body cells the same as in the reproductive cells.

There are two main kinds of effects on the genetic material. One is a break in a chromosome. Two, a change in the inner composition or arrangement of parts of the gene itself.

The breaks in the chromosomes have more effect on the exposed individual himself when they happen in the somatic cells for this reason. If this cell in the diagram is one that is going to later reproduce itself in the body to form more cells, or to multiply, as our skin cells must—as skin is always sloughing off and has to be replenished by multiplication of the cells in the lower part of the skin, and it is the same with many other parts of the body—then these chromosomes have to reproduce themselves, as Dr. Glass explained, forming duplicates of themselves. Then they are drawn apart by the process of cell division or mitosis, forming 2 groups of chromosomes in the 2 daughter cells. But if the chromosome is broken, it may kill the whole cell at the time or shortly after division by this mechanism, or it may result in very abnormal cells.

There is a point on each chromosome, which becomes attached to a fiber that pulls the 1 daughter chromosome to 1 pole and the other to the other pole, so that when you get the cell dividing into 2 daughter cells, one daughter cell gets one representative and the other gets the other representative of that duplication process, and they thereby come

to have like material. The chromosome gets pulled at that particular point. But the part beyond the break won't be pulled in because it has nothing to be pulled by. It gets left behind, and the cell is left without all those genes.

Moreover, where the chromosomes have broken, there is a sort of stickiness which causes them to join together at their raw ends if they happen to touch, which is very often the case, and then, when they get pulled apart, they form what we call a bridge between the two cells. They can't, in that case, get apart. That chromosome bridge, then, connects the two cells, which often die in consequence. Therefore, as a result of the chromosome break, we often have a cell death.

Dr. Puck, of Denver, studying human cells in tissue cultures, has found that, with doses of radiation given at a high dose rate, it takes only about 90 r. to cause the death of about 50 percent of the cells. In other words, that happens to about 50 percent of the cells in the tissue culture. It will only happen to cells which attempt to undergo division.

Your brain cells, your kidney cells, as Dr. Warren said yesterday, are not affected much by radiation. On this interpretation the reason is easy to see, because they have stopped dividing for life. You have the same cells of these kinds for life, whereas your skin cells, the cells of the intestine, et cetera, have to keep replenishing themselves by division. It is only when they come to replenish themselves by division that these tangles take place that result from the breaks in the chromosomes.

On that basis it is easy to understand the rule that Dr. Warren referred to yesterday, sometimes known as the law of Bergonié and Tribondeau, established about 1906, that it is the tissues with rapidly dividing cells that are the most damaged by radiation. The damage does not take place in the cell until division occurs. When that happens in a germ cell, those cells generally die and seldom take part in the production of offspring. That is why this is not important for the later generations. But it is important for the generation itself. It takes place both in the germ cells and body cells, but it is in the body cells that the main damage of this kind is done.

I will give you an illustration of that. In the people that were bombed a long time ago at Hiroshima you can see a lot of little opaque points in many of them in the lens of the eye where evidently cells have been badly damaged. That damage did not appear until much later when the cell probably undertook to divide. The reason you can see it there is because the lens is a transparent tissue. I think there is reason to believe that this happens in all the tissues of the body that contain cells that are subject to division. These tissues would, therefore, be weakened.

It is true that other undamaged cells would tend to replenish the damaged places. But that replenishment or regeneration, it is to be expected, will not be complete and perfect. Therefore, there is a certain amount of damage left. That damage, you can see, would be a generalized damage all over the body, wherever there are these dividing tissues. Therefore, it would be expressed as a weakening of resistance to disease and infirmities of all kinds, somewhat like what occurs in aging, since then, also, our resistance to bodily ills decreases as the functioning of the cells or tissues weakens.

I do not say by any means that is the proved explanation, though we do know this phenomenon of chromosome breakage occurs, and, to me, having examined the evidence, it seems by far the most reasonable explanation. That is, the bodily damage in general is to be explained in this manner rather than by some damage to the other materials of the cells.

There is a good deal of other evidence showing that it is this damage to the genetic material, to chromosomes and genes, that is by far the most important damage, and that you have to have the radiation strike at or in the close neighborhood of the chromosome or gene before you can damage it. For example, experiments done in Chicago by Zirkle and Bloom show that when they were able to direct a minute beam of radiation (protons) through the cell, it was only when they actually hit the chromosome or right close to it that you then got chromosomal damage. If you hit right there, when the cell divided you had the bridges form.

Other experiments similarly show that if you only hit the rest of the protoplasm you do relatively minor damage compared to what happens when you hit the chromosomes or genes.

You can readily see, I think, that since we know through experiments in geentics that the frequency of these breaks, like the frequency of the mutations of the genes, is linearly proportional to the dose of radiation used, no matter in how small bits it is divided, then you might expect a derived effect, such as the decreased resistance to disease and consequent shortening of the life span also to be linearly proportional.

It is true that at high dose rates of radiation you sometimes have two chromosomal breaks near together and then you can get entanglements which would not happen if you have low dose rates. At low dose rates you therefore expect the effect to be proportional but at the high dose rates to go up even more steeply.

I think it is also very probable that even the very pronounced effects of heavy exposure, such as you find in radiation sickness, such as the nausea, the drastic lowering of the count of white blood cells, the bleeding internally, are also due to this genetic damage to the somatic cells.

For example, in a very careful investigation, Dr. Quastler showed that the intestinal injury that leads to death after high doses is caused by the failure on the part of the cells of the intestine that normally replenish the rest every 3 or 4 days to carry out this task. Owing to being so badly damaged, they fail to survive the process of cell division in normal condition.

These effects of heavy doses do have thresholds, not because there is a threshold in the production of chromosome breaks, but only because you do not see clinical symptoms unless you have damaged a certain number of cells.

If this point of view is correct, then I think it is to be expected that even the damage to the exposed individual could be better investigated by people who have the genetic point of view about it. They would be the ones who would be more likely to look for an effect that has no threshold. I think it is not at all surprising, therefore, in view of the probable mechanism of these effects, that it has taken geneticists, notably Boche, in the case of life span, and Lewis, in the case of the

effect of leukemia—both, by the way, *Drosophila* geneticists originally—to uncover the evidence for the cumulative nature of the damage to exposed individuals. Moreover, I think the research should be continued along those lines.

Doctor Friedell said yesterday that the important thing to know is the mechanism involved. I do not say we know it. But I think we have more than a pretty good hunch. And we have to follow these hunches, even if they lead to conclusions that are distasteful to some people, such as that there is no threshold. But if we calculate (as Dr. Hardin Jones will calculate with you) the amount of effect of a given dose on the length of life, we find that the dose which is now the maximum permissible dose for occupationally exposed workers, namely, 50 r per 10 years, would lead in 40 years of their work to 200 r. and would thereby deprive them—we may want to change this estimate; we can't now say exactly—of some 4 years of their life. Perhaps 1 year of their life lost for each 4 years they work, or something like that.

My main point here is that if there is no threshold, then the loss is of a sizable amount and we had better pretty soon find out how much. We meanwhile have to act on the supposition that it exists. Nevertheless, this effect on the length of life of the exposed generation is not as great as the effect in damaging future generations.

Representative HOLIFIELD. Why is that, Doctor? Will you explain that?

Dr. MULLER. Yes. Let me now speak of the damage to future generations. I said that not much of it was caused by the chromosome breaks but most of it by the mutations of the genes. The reason the mutations of the genes do not cause much damage usually to the exposed individual is because most of them are what we call recessive. There is a normal gene from the other parent that has a dominating effect, although it may very well be the case that in leukemia we have a dominant gene. But mutant genes that are decidedly dominant are not the usual ones.

As for later generations, the chromosome breakage cases are largely weeded out by the cells dying before they get to the next generation. The mutations of the genes, however, persist and are handed down as mutant genes. These mutant genes also are usually recessive, as Dr. Glass explained. The person usually gets a normal gene from the other parent and that has the dominating effect. But the dominance is not quite complete usually, and that slight deviation from completeness is very important.

Suppose it only reduces by 5 percent the chance of an individual surviving to maturity; that is a chance of death of 1 in 20. It does so by handicapping him in some way. It is usually a slight handicap that he hardly realizes is there. He takes it in his stride because he has had it perhaps from birth, though it may not have expressed itself always, and it is mixed in with his other infirmities. All of us have some. No one is perfect because there is no such thing. But by it his biological survivability is reduced by just that much, and he hands that weakness on down to the next generation, and after awhile it will take its toll by happening to come in a combination of circumstances where it will kill or prevent reproduction. So the thing will finally die out.

As Dr. Crow explained, if it gives a chance of 1 in 20 of causing death in 1 individual, it tends to pass down to 20 individuals before it takes its toll. So it hampers correspondingly more persons than a gene that killed outright. Therefore, as Dr. Crow explained, the slight mutation is as bad in the end as the mutation with the big effect. Moreover, we are all of us full of these defects that come from the past. A hundred things, each of which does a one-hundredth of as much harm, are together as bad as one thing that does that much harm.

Representative HOLIFIELD. Before you leave that, is it not true that if a general population receives a dose of radiation, as would occur in the general raising of the rate in the atmosphere, that there would be a greater chance for the mating of recessive genes?

Dr. MULLER. If both people had been exposed, no, the increase in the chance would be insignificant, contrary to a common misconception. Because the chance is so slight that it should have been the same gene that was effected in both parents. There are more than 10,000 genes, we believe, and if each parent had an affected gene, therefore, the chance would be only 1 in 10,000. So that is virtually ruled out.

Senator ANDERSON. May I go back one step? Did I understand you to say that if, for example, the exposure was 50 r. every 10 years, and a man worked for 40 years, making a total of 200 r., his life might be shortened as much as 1 year for every 4 years of work?

Dr. MULLER. No; I am sorry. I made a mistake. I means 1 for every 10. Dr. Hardin Jones will give you the latest information.

Senator ANDERSON. You said 1 for every 4.

Dr. MULLER. Yes. I was thinking of 4 times 10, and I got the 4 instead of 10.

Senator ANDERSON. So that would be 4 years shortening.

Dr. MULLER. Yes. That would be the provisional estimate I made a long time ago and we may have better data now. I think this is probably a conservative estimate.

Dr. JONES. A very reasonable estimate.

Senator ANDERSON. Thank you.

Dr. MULLER. These effects then, being slight, are usually not recognized as such. Certainly you could not tell which mutation was caused by radiation, if radiation had been received by the parent, and which was not. The induced mutations are like those already existing in the population but are added to them. The only way you can tell they have been produced is by means of a very exact statistical study on large groups, comparing those that have been irradiated and those that have not.

Through work on the fruitflies where we have the most exact knowledge to date, unless Dr. Russell has more exact knowledge on mice now, we can get a kind of minimum estimate of the amount of damage to the children by a given amount of irradition of the parents. Although there would not be time to show you here the way the calculations are done—and they do have a considerable error—I think it is possible to show that the amount of damage to the offspring of parents that had received a certain amount of radiation to their whole body, in the case of fruitflies, would be something like the amount of damage in the parents themselves.

A comparison, however, would show that, when, X-rays or gamma rays were used on fruitflies, the damage to the offspring would be somewhat less than to the parents. You may think that is contradicted

by what I said before when I said the damage to future generations is greater than to the parents. There is no real contradiction because we are here referring only to the first generation of offspring. But they hand the damage down to their offspring, and so on.

As Dr. Crow explained to you, the damage does not die out to half its value for probably scores of generations. So you have to multiply this damage to the first generation of offspring by scores, maybe by 50, to find out what the total damage to future generations is of just the 1 exposure to 1 generation. When you have done that you have obtained a figure of damage to future generations that is far greater than what is done to the exposed generation itself.

I think most people would not be impressed with the weaknesses caused in future generations, even though future generations would feel them. Therefore, it is my contention that it is a very good thing that people's own life span is shortened; that is, that there is a demonstrable effect on the generation itself that is exposed, because they will take notice of this effect on themselves, if they are allowed to know it. They will then take precautions that will save future generations from a lot more damage than it saves themselves.

The prolonged official reluctance, at least until a year or two ago, to give information in popular form regarding these major types of radiation damage, that in the exposed individual himself expressed in the shortening of life and long delayed malignancies and that expressed in the descendants, and the reluctance to give information regarding the conclusion of some of those who have worked most directly in the field that even the tiniest doses add up accurately to determine the amount of these effects without any threshold, has, I think, undermined the confidence of large numbers of well-intentioned people in the judgment and the intentions of the responsible governmental authorities, because the facts have, after all, leaked out or have been suspected by the public, and they wonder why nothing has been said about it.

As I said before the National Academy of Sciences 2 years ago:

> So many of the public are already aware of the genetic damage produced by radiation that their morale is weakened and their apprehensions are increased when they see that the damage is denied by prominent sponsors of our national defense. Thus the door is opened for their acceptance of the defeatist propaganda which alleges that even the tests are seriously undermining the biological integrity of mankind. In this situation the only defensible or effective course for our democratic society is to recognize the truth, to admit the damage, and to base our case for continuance of the tests on a weighing of the alternative consequences.

Now when we do this, we conclude that the number of lives that will be seriously curtailed or injured throughout the world in future generations, as a result of the tests already held—supposing that they continue at this rate for perhaps 10 years longer—is in all probability in the hundreds of thousands or millions, and is therefore enormous. We should recognize that.

Despite all the uncertainties in regard to the exact figures, I think it was not possible to make clear to you how much careful work these estimates were based on, and the fact that although there may be an error of what we call a factor of 2 or 3, that is, that the true figures may be 3 times as much or only a third as much, nevertheless it is very unlikely that they should be less than, I would say, a third as much. In other words, the values given to you by Dr. Crow are those which are most likely in the light of present knowledge.

So I think we must recognize that the number of lives that would be seriously curtailed or injured will be in the hundreds of thousands or millions, and is therefore, enormous.

Senator ANDERSON. Doctor, you say that a number of lives seriously curtailed or injured from tests already held?

Dr. MULLER. Yes. I should modify that. I should say from tests held at the rate at which they have been held.

Senator ANDERSON. As they are now going?

Dr. MULLER. Yes.

Senator ANDERSON. That does make some difference.

Dr. MULLER. Yes.

Senator ANDERSON. That would damage, you think, hundreds of thousands or perhaps millions, and you say that is within a probability factor of 3?

Dr. MULLER. Yes.

Senator ANDERSON. A third as much or three times as much?

Dr. MULLER. I would put it at that.

Senator ANDERSON. Then it would be a very substantial number of lives if it is on the smaller side and an enormous number of lives on the larger side.

Dr. MULLER. Yes.

Senator ANDERSON. Doctor, do you believe that a number of geneticists agree with you in that point of view?

Dr. MULLER. I do, yes. They might differ as to where to put the factor. Some might say 2. Some might say 5 or 6.

Senator ANDERSON. This would sort of imply that there is no threshold.

Dr. MULLER. Yes.

Senator ANDERSON. That it starts immediately.

Dr. MULLER. Yes. That is because of the mechanism that Dr. Pollard explained. The thing strikes or it doesn't strike. If it strikes it does the damage even if there was only that one strike. He made the comparison with a lightning strike occurring in a large space and a long time. in which however that strike would be just as effective.

Senator ANDERSON. I ask that question because of the discussion we had this morning of having some group to come in and discuss these figures with a responsible group such as the Commission itself. You do feel that the geneticists could make a strong case in support of that?

Dr. MULLER. Yes, especially those who have worked in the field.

Senator HICKENLOOPER. Dr. Muller, in your prepared statement I notice you said with respect to that, and I quote from the prepared statement as follows:

> As I stated before the National Academy of Sciences 2 years ago, it has caused—

that is, certain conceptions or misconceptions—

> people to lend too ready an ear to the alarmists who declare that the genetic material of the human race is seriously endangered by the fallout from the test explosions themselves.

I do not know whether you got to that or whether you intended to do it or not.

Dr. MULLER. No. I read a more detailed statement on the subject, taken from the discussion that I gave before the National Academy

of Sciences. I read a part of that discussion. But I also stand by the statement as I have it in the text that you have just read.

Senator HICKENLOOPER. With respect to these numbers which you refer to as hundreds of thousands or millions, do I understand the connotation of that to be that that in fact is extremely minute as compared to all the human beings for ensuing years?

Dr. MULLER. Yes.

Senator HICKENLOOPER. Although it is large in numerical value standing by itself.

Dr. MULLER. That comes in the next sentence, you will see, where it says:

Nevertheless these injuries, being scattered over the whole earth and through hundreds of years—

I should have said thousands—

are relatively very few, in comparison with those due to other causes, including natural mutations. Moreover, the suffering to be entailed, although enormous in absolute terms, must be very small relatively to that which might follow from any serious mistake in the conduct of international relations.

Senator HICKENLOOPER. Therefore, Doctor, it becomes a matter of relative value in this particular field.

Dr. MULLER. Yes.

Senator HICKENLOOPER. Some things we may have to do, such as getting into war where we do not want to kill people, whether by bullet or disease or anything else, but we have to balance security and necessity against the hazard and strike a balance as to our conduct.

Dr. MULLER. Yes. I do not mean that when we strike that balance—I am not trying here to say which way the balance will be struck.

Senator HICKENLOOPER. I understand. You are making the point that someone must exercise judgment and determination in the light of all the circumstances.

Dr. MULLER. Yes. I would agree with Doctor Sturtevant that one life is a serious matter.

Senator HICKENLOOPER. Yes, without doubt.

Representative HOLIFIELD. You are really making a plea that all the facts be known before the decisions on policy are made.

Dr. MULLER. Yes.

Representative HOLIFIELD. And no facts be repressed.

Dr. MULLER. Yes.

On the other hand, the consequences of a full-fledged war, with its heavy irradiation of large numbers of people on both sides, would be inordinately more serious in its effects on the human genetic heritage as well as in its more direct effects. It is this consideration which, in my opinion, makes a continuation of test explosions a monstrous mistake of policy for both sides. Of course it would be absurd to expect one side to stop without the other. But a continuance by both sides would tend to lead the world nearer to a war that even with present techniques would result in the cataclysmic ruination of humanity in general.

May I add that the means of destruction are now so advanced on both sides that further advances by one side alone could not save it in the case of a war from becoming itself destroyed. By war I mean an atomic war, because I do not think you can have a world war any-

more without a thermonuclear or atomic war. I do not think it is realistic to suppose that you can.

Senator HICKENLOOPER. This illustration, I realize, is not exactly on all fours with the situation we have here today, but we are concerned with the danger and propriety of continued tests in the world.

Dr. MULLER. Yes.

Senator HICKENLOOPER. I presume all of us would earnestly hope that we never had to test atomic weapons. That perhaps would be the ideal. There are certain political factors that enter into those decisions, but by the same token I presume that we want to save thousands of lives in this country every year and we could just abolish the manufacture of automobiles and go back to riding horses. It seems to have struck a balance in the minds of people that transportation is important and we keep making automobiles, people keep getting killed by the thousands on the highways every year. We are all sad about that.

The point I was attempting to understand in my own mind is that there is a balance which someone must determine as to the ultimate good to either us or the world in this atomic field and whether or not we continue.

Dr. MULLER. I would accept that, except that I would rather say, "which every one has to help determine."

Senator HICKENLOOPER. Yes, indeed.

Dr. MULLER. I might add in this connection that in my talk to the National Academy 2 years ago I stated it to be my belief at that time that a continuation of the nuclear tests was necessary. I still think that this was the case at that time but I think the situation has changed since then. But there I am not speaking as a geneticist.

Senator HICKENLOOPER. I do not know whether I understand or not, but may I ask you, are you advocating that the United States stop testing weapons unless we get reliable agreement that other nations in the world would stop also?

Dr. MULLER. Of course not; no. The more that we can get people of the world to recognize the terrific damage that nuclear war will bring to them all, I think the more they will see the light on that point.

Senator BRICKER. Are you going to discuss later, Doctor, the answer to the question I asked this morning in regard to the experimentation that is going on in Russia?

Dr. MULLER. Yes. Just a little more here—I am on the last page on a somewhat different topic, but it is all related.

In order that the grave biological effects of radiation may receive due recognition and study—and I have tried to show you that they have not received due recognition and study—and may duly influence our policies and procedures, it is important that persons with a systematic background in genetics be placed in positions in which the decisions involving these matters are made. Truly this should be the case if there is any chance that genetic processes lie behind all the major damage done by radiation to man. Yet this is not the case at present.

For example—and let me not be misunderstood here—I have the highest regard for Dr. Shields Warren and for Dr. Brues and their associates, but I think it is important in this connection to point out that the official delegates of our country on the United Nations Scien-

tific Committee on the Effects of Atomic Radiation—and those are the gentlemen I have just mentioned—are neither of them geneticists. I am sure neither of them would wish to claim to be. I mean by that they would disclaim it.

Representative COLE. On that point, Doctor, do you know whether other members or official delegates from other countries on this committee are geneticists?

Dr. MULLER. There is one geneticist of bacteria, Dr. Appleyard of Canada. There is Dr. Caspersson from Sweden who studies chromosomes and other cell materials through the microscope. He is not exactly a geneticist. There is an alternate, Dr. Gopal Ayengar, from India who is a geneticist. Dr. Bacq of Belgium is in fields related to genetics.

So far as I know, those are the only ones that come near the subject of genetics who are on that committee. I stand to be corrected. They can, of course, and have, at least at the last meeting, I understand, had some geneticists present as consultants. Yet the chief discussions of that committee to date, so I have been told, have been on directly genetic matters, on these very questions here, especially on the effects on future generations.

You may remember that this country insisted that the delegates be chosen not by sciences but by countries and that the delegates by countries be chosen by the government and not by the scientific bodies. Some other countries put up strong resistance against that, but finally accepted it.

It may be noticed that most of the nongeneticists who deal with these matters, as I think is clear from the discussions of yesterday, are on the same side. They are on the other side from geneticists in regard to the major question of whether there is a threshold or whether the major effects on the body are linearly proportional to the dose all the way down to zero. That is an important issue in assessing the effects not only of the tests, but also of the peacetime uses of atomic energy.

I might say that when it comes to another body that is very important in this connection, the National Committee on Radiation Protection, we are in a better situation. Dr. Glass here, a very good geneticist, if I may say so, is a member of that committee, who was newly appointed just a few days ago. I was a member of one of the subcommittees for some years, although I am not sure whether I still am or not.

Representative COLE. Mr. Chairman, may I clarify the record? I understand Dr. Glass had been a member of this committee for 3 years.

Dr. MULLER. No, that is another committee. That is the Atomic Energy Advisory Committee on Biology and Medicine. I am now speaking of the National Committee on Radiation Protection which is under the auspices of the National Bureau of Standards and which is the one promulgating the permissible dose which we had explained to us the other day on the blackboard.

The latter committee do have, as I have said, a geneticist here and there. However, they have official representation from about 15 different organizations, mostly of a medical or governmental nature. Yet they do not have one official representative from any of the professional genetic organizations, such as the Genetics Society of America,

the American Society of Human Genetics, the American Genetic Association, or the Society for the Study of Evolution, all of which are in my opinion as closely concerned with this matter as for example the Radiological Society. Consequently, there is not sufficient representation among them of that genetic point of view of the mechanism which leads us to expect no threshold and to take the matter more seriously at small doses. This circumstance, I think, provides the reason why the record of this committee's decisions on the permissible dose, which as Dr. Taylor presented the matter yesterday appeared to show that they were so cautious, in actuality showed that the first dose they set was far too high, so that they had to set it lower. Then they found the second limit also was far too high and again they came down. And recently they found that the third limit in turn was too high and they came down once more. This does not indicate that they have been so cautious. It means they have not been cautious enough. The geneticists would not have set so high a permissible dose in the first place, on the basis of what we knew 30 years ago.

The grounds for the reduction in permissible dose that was made by the committee a few years ago, prior to the issuance of the National Academy's report, did not lie in considerations of genetic damage. For the permissible dose handbook specifically stated that this dose (of 0.3 roentgens per week) was set without regard to genetic effects. The geneticist members objected to that but it was carried anyway. In other words, it was known that the dose was considered too high on genetic grounds but it was adopted in spite of this, although it was acknowledged that it might be reduced again later.

Now that the Academy has made its report it has in fact been reduced a great deal more. I agree however with Dr. Glass, that it is probably due for even further reduction.

A similar attitude is reflected in the omission of any mention of the genetic effects of radiation in the courses on radiation in relation to health that are given both for our own people and for foreign selectees under AEC auspices at Oak Ridge. It is not enough to have biologists of some sort, or medical men, to reach decisions on these matters, unless they include a strong contingent of geneticists and of those who have a genetic point of view. Others are not likely to admit the danger from small doses.

In view of this situation and of the notorious resistance to the acceptance of genetic principles on the part of so many, not only of governmental appointees in the policymaking positions, but also of so many of the medical profession, a resistance that has prevented the medical profession for 30 years from duly protecting themselves, their technicians, and their patients when X-rays are used medically, and that has thereby subjected the reproductive cells of our population to very much more radiation than that from fallout—it is my opinion highly important that a National Radiation Health Institute be established as a part of the United States National Institutes of Health, but only if it contains a solid core of competent and versatile geneticists as one of its major features.

It is true that there is excellent research on the genetic and other effects of radiation being carried out in our country and that a considerable amount of it is made possible by the support or is done under the auspices of the AEC. This research, however, does not sufficiently insure the all-around consideration and study of these matters in rela-

tion to public health and well-being, and the promotion of adequate measures in application of the conclusions reached.

If anyone wishes to ask some specific questions on the Russian aspect of the situation, I would be glad to take them up.

Senator BRICKER. I just wanted him to discuss the situation and whether they are conscious of the conclusions you have come to and are presenting to us and what research is being done there with regard to the effects of fallout.

Dr. MULLER. I have to infer from my knowledge of Russia derived from various sources, including firsthand information gained 20 years ago, that they are and will be having to follow our lead. We can't look to them for useful information at present along these lines. For, as I think most people realize, there was a purge of geneticists and an expurgation of the subject of genetics from teaching in the school and universities, from the boards of publication of journals, and from research institutes. It has not been taught to students for about 20 years. Most of the leading geneticists were somehow done to death, and I say this advisedly.

Chairman DURHAM. You say they will have to follow us. Do you think they would follow us?

Dr. MULLER. Yes.

Chairman DURHAM. Do you think so?

Dr. MULLER. Yes. There are a few geneticists left, of course. I think that some of the politicians in leading positions, since Stalin died, realize the folly of their old ways in regard to the subject of genetics. We have solid evidence that it is now possible to advocate the principles of genetics and to do some research in it.

A few of the old research workers are left, and there are said to be plans to give them positions in which they can resume their genetic investigations. However, the quacks have not by any means been disestablished yet, although they do not hold as commanding positions as they had before. One of the older geneticists, Dubinin (he was not old 20 years ago), was even rumored to have been selected as one of their delegates on the U. N. Scientific Committee on the Effects of Atomic Radiation. If so, Russia did better than most of the other countries in regard to that committee because Dubinin was a real "honest-to-goodness" geneticist.

Chairman DURHAM. You do think that Government officials are getting advice on genetics?

Dr. MULLER. They are beginning to get advice on genetics again. But I also note that the quack group are still strong. We have proof of that in publications and in the fact that in the Conference on Genetics that was held in Japan last September the Russians sent, I believe, four delegates, and all of them belonged to this quack school. So there is a division on the matter in Russia now.

Chairman DURHAM. What do you think is the reason for the Russians not permitting the genetic scientists to take part in the United Nations?

Dr. MULLER. The Russian, Dubinin, is a geneticist. Most of the other countries didn't seem to realize that geneticists were needed, for this committee seemed to assume that physicians would know about the subject. However, you will find very few physicians in this country that have an education amounting to anything in genetics.

Chairman DURHAM. Did I misunderstand you? I thought you said they would not permit him to go to the United Nations panel.

Dr. MULLER. No. I said I had heard, but I cannot verify, that Dubinin was to go to that meeting as an official Russian delegate. Whether he went or not I do not know. Perhaps someone here knows. I would be interested to know if he did.

Representative HOLIFIELD. Dr. Muller, you have spoken in two instances in your presentation here of the prolonged official reluctance and the curious official silence. Is it not true that you were invited to give a paper at the Geneva Conference a couple of years ago?

Dr. MULLER. Yes.

Representative HOLIFIELD. The Geneva Conference on Atomic Energy?

Dr. MULLER. Yes.

Representative HOLIFIELD. Did you give it?

Dr. MULLER. It was printed in the proceedings.

Representative HOLIFIELD. That is not quite an answer to my question.

Dr. MULLER. No, I did not give it there. I was prevented from giving it.

Representative HOLIFIELD. Who prevented you from giving it?

Dr. MULLER. The story has some complications of detail, but the essential thing is that it was called off by higher echelons of the AEC. That was actually shortly after this article of mine had appeared in Science that I quoted from earlier, in which I said there had not been enough airing of the matter.

Representative HOLIFIELD. Were you given any reasons as to why you were not allowed to give your paper?

Dr. MULLER. Yes; there was not room for me. Also they were sorry they had to notify me so late because they had only just received word so late from the International Committee. It was afterward found that it was not the International Conmmittee that had asked to have my paper excluded. They had approved of having it given. That expression was used, however, in the official letter written to me by the AEC authority.

Representative HOLIFIELD. Are there any further questions?

Representative COLE. Mr. Chairman, it had been my understanding that the committee was going to allow our staff specialists in this field to interrogate the witness in any area in which they felt there was some need for further amplification. I would like to inquire if Mr. Hollister might not have some questions of Dr. Muller. I would suggest that hereafter Mr. Hollister would be invited to interrogate. He is reluctant to inject himself into the interrogation.

Representative HOLIFIELD. The Chair has informed Dr. Tompkins and Mr. Hollister that they have the privilege of touching me on the shoulder and asking questions of any witness. They certainly do have that privilege. It was announced at the beginning of the meeting, and we certainly intend to allow them that privilege.

Mr. Hollister, would you like to ask some questions of Dr. Muller before he leaves the stand?

Representative PRICE. Before he does, is Dr. Muller's paper before the National Academy of Sciences included as part of the record?

Representative HOLIFIELD. Dr. Muller, Mr. Price's question was,

have you presented your paper that you were to give at the Geneva Conference as part of the record of your presentation?

Dr. MULLER. I had not intended to do so. I would like to present something more recent—two things in fact—one short paper, Potential Hazards of Radiation, which is now in press in the journal Excerpta Medica published in Amsterdam. It is six typewritten pages with some references.

Then a little statement that I gave out last October 20, 1956, that I think I could read. It is very short.

> So devastating would be the damage done to both present and future generations by the nuclear explosions which any global war is likely to bring, that the great issue of today is not that of the relatively minor damage produced by mere tests of H-bombs, but that of taking all steps we safely can, such as the mutual discontinuance of these tests, which will tend to lessen international tensions and bring us nearer to all-round armament control. Unless this control is achieved in the short time open to us before thermonuclear weapons have become available to more countries still, and before intercontinental missiles have become a reality, we will find ourselves in a situation even more ungovernable and menacing than that of today.

I also wish to introduce a paper in the report by the World Health Organization study group on the effect of radiation on human genetics (see appendix, p. 1728). This report of the World Health Organization is being presented to the U. N. Committee on Radiation Damage and will be published next month sometime, it is expected. I have a copy of the paper of mine of that report here to include.

Representative HOLIFIELD. Thank you very much. Without objection they will be received.

(The document referred to, together with an article entitled "How Radiation Changes the Genetic Constitution" by Dr. Muller, follow:)

POTENTIAL HAZARDS OF RADIATION [1]

(By H. J. Muller, Indiana University)

Evidence has in recent years been accumulating for the broad conclusion that the great majority, if not all, of the damaging effects on life and health evoked by ionizing radiation are results of permanent changes produced in the genetic material: the chromosomes or their contained genes. These changes, when occurring in the genetic material of the germ cells, reach expression through reduction in the number of functional germ cells (infertility), increased mortality of zygotes of the first and subsequent generations (dominant lethals and detrimentals), and, in general, the alteration of one or more hereditary characteristics, that thereafter are transmitted in their new form (mutations).

Our knowledge of the nature of these effects, and of their manner of production, has been derived in the first place from studies of the descendants of exposed individuals, checked by cytological observations of cells derived from exposed germ cells. However, there is increasing ground for the inference that ionizing radiation produces changes in the genetic material of the somatic cells like those in the germ cells, and that it is these chromosomal and gene changes in the somatic cells that form the basis of most of the damage to the exposed individual himself, such as erythema and the various other aspects of radiation sickness, shortening of the life span, diverse malignancies, etc.

The primary changes in the genetic material, as disclosed by cytogenetic investigations on widely different forms of life ranging from viruses and bacteria to fungi, flowering plants, insects and mammals, may for convenience be divided into two groups. These are the *chromosome breaks*, that result in fragments the broken ends of which tend to unite with one another either in the old or in new arrangements, and the *point mutations*, that involve alterations at very localized positions on the chromosomes and are inherited according to Mendelian principles. It would take us too far afield here to discuss the proposi-

[1] Slightly modified version of article in press in June 1957 issue of Excerpta Medica (Amsterdam).

tions that there may be a fundamental similarity between the changes of these two groups, that they may integrade with and even overlap one another, and that, at any rate, the classification into one or the other group is in many cases uncertain. However that may be, the distinction remains of practical importance.

It is the point mutations which, although the more elusive of the two types of genetic changes, do the most harm in the long run. Even though evolution has come about by the natural multiplication of the very infrequent advantageous point mutations, the vast majority of them, whether arising naturally or induced by radiation, are of a detrimental nature, as is only to be expected of "blind" changes. Yet, contrary to popular opinion, mutations giving rise to conspicuous abnormalities, monstrosities, or freaks, are a great rarity, even after heavy doses of radiation. The point mutations induced in animal material by ionizing radiation have been found to be similar in their range of types and in the relative frequencies of those having different kinds of visible expression ("phenotype") to the naturally arising mutations, although the distribution of relative frequencies from gene to gene may be somewhat different for the two groups.

In considering the expression of a mutant gene we must distinguish between that which it has when homozygous, i. e. when inherited from both parents alike, and when heterozygous, i. e. from but one parent (the other having supplied a normal gene). Many mutant genes (possibly as many as one-fifth, as indicated by work on fruit flies) have such drastic effects when homozygous as to unconditionally kill the individual prior to maturity: these are the "lethals." The great majority of the remainder, when homozygous, cause some degree of impairment, even though relatively few of them give rise to readily visible abnormalities. In heterozygous condition their expression is usually much less pronounced than in homozygous condition and is very seldom recognizable; hence most mutant genes are termed "recessive." Yet even when heterozygous there is usually some slight, statistically important impairment of the capacity to live and reproduce. Hence, since the mutant gene is regularly transmitted to subsequent generations, it nearly always results, eventually, in the extinction (genetic death) of the line of descent carrying it, and this usually happens before the gene has had an opportunity to become homozygous.

These "genetic deaths" are seldom identifiable as such because the heterozygous individuals that suffer them show so little recognizable impairment. They represent the price paid by any population in preventing an unlimited accumulation of mutant genes within it. Studies on the frequencies of natural mutations in Drosophila, the mouse, and man, and of the effects of inbreeding in man, agree in indicating that each person carries on the average, mainly in heterozygous condition, at least four times as many mutant genes as would have been enough to kill him outright if they had been homozygous. It is further indicated that, in the mainly heterozygous condition in which the mutant genes actually occur, they tend, by their cumulative action, to cause the genetic death of at least 1 person in 5.

When the mutation rate is raised by exposure to radiation the frequency of genetic death is correspondingly raised, over a number of generations that is inversely proportional to the degree to which the mutational damage is expressed in the individual's of any given generation. Russell's studies on the mutations of 7 genes in mice show that some 30 r. delivered to the immature germ cells constitutes the "doubling dose," in that it induces in them as many mutations as arise naturally per generation. It is unlikely that in man the doubling dose is more than twice as high, and it may even be somewhat lower than in the mouse. At any rate, the correctness for man of this order of magnitude is indicated by studies of Turpin and Lejeune and of Macht and Lawrence, and is not contradicted by the lack of statistically significant findings in the studies made in Japan by Neel and Schull.

Since there is much evidence indicating a linear relation between the radiation dose and the frequency of the induced point mutations, even at extremely low doses, and the exactly cumulative nature of these radiation effects, it becomes possible to arrive at probable estimates of the minimum damage done to subsequent generations by any given chronic or acute exposure of parents. In view of the dearth of conspicuous abnormalities, one of the best overall measures of

this damage lies in a measurement of mortality, expressed for instance as length of life. The criterion has recently been used by Russell in his demonstration that neutrons applied to male mice cause a shortening of the average life-span of their progeny that is about as great as that caused in the directly exposed individuals: nearly 0.1 percent of the life-span per rep. Since, however, the effects on the progeny are likely to reappear throughout scores of generations before being terminated by means of genetic deaths, the total damage to the descendants is many times greater than to the exposed individuals.

Chromosome breaks, like point mutations, are induced with a frequency directly proportional to the total dose of radiation, regardless of how concentrated or dispersed the treatment has been. Some of the rearrangements of chromosome parts result from single breaks, and some from two or more breaks lying in the course of the same ionizing particle. The frequency of either of these types of rearrangements, like that of the individual breaks, varies directly as the total dose, regardless of its distribution in time. Other rearrangements, that result from a combination of fragments derived from independent breaks occurring within the same few minutes, have in consequence of this mode of origin a frequency that is insignificant when the radiation has been of low intensity but that rises rapidly with the intensity, approximately as its square, and that becomes of major importance at high dose rates. Thus chronic or repeated low exposures give rearrangements that are linearly related in frequency to the total accumulated dose, while higher dose-rates give disproportionately numerous effects.

A cell in which one or more chromosomes have been structurally changed by breakage will continue to function normally until cell division occurs. At that time the altered chromosomes often give rise to chromatin bridges that connect the daughter nuclei, interfere with their further multiplication, and ultimately result in death of the affected cell line. In the absence of a bridge, the daughter or descendant cells may come to lack parts of chromosomes and/or to have other parts in excess, and the resulting unbalance of gene proportions ("aneuploidy") tends to impair and even kill the descendant cells. Happening in the germinal line, these phenomena are expressed as infertility of the exposed individuals, and as lethality among embryos of later generations.

The chromosome damage, leading to postmitotic cell impairment and death, is also induced in somatic (body) cells. It provides an interpretation of such phenomena as the "law of Bergonie and Tribondeau" (relating the degree of tissue damage to multiplicational activity), the radiation death of individual somatic cells in tissue cultures (as in work of Puck and Marcus), and the delayed production of minute cataracts in irradiated lenses of the eyes. That it is the chromosomes rather than the protoplasm of cells which are ordinarily the seat of the more significant radiation changes, leading to cell death, has been shown in numerous studies, among them those of A. R. Whiting, Zirkle and Bloom, and Ulrich. It is only when a given number of cells has been destroyed within a given space and time that certain visible symptoms appear, such as reddening of the skin, reduction in number of white blood cells, intestinal hemorrhage, etc. However, there is no threshold for the individual cell effects, and analyses such as that of Quastler are increasingly implicating them as the basis of the clinical manifestations.

It is almost certainly through the individual cell deaths and impairments that minute doses of radiation, long continued or repeated, exert their action in shortening the life-span of the exposed individual. This effect, first analyzed by Boche and then by Sacher, has been calculated to cause a reduction in length of life of the order of several days for every roentgen unit received by the body as a whole during a person's lifetime.

On the other hand, leukemia and some other malignancies, the induction of which may also be linearly dependent upon radiation dose, are considered by geneticists as being more probably results of point mutations in somatic cells than of chromosome breaks. From the conclusions of Lewis it may be calculated that for a population of 160,000,000 with a lifespan like that in the United States each absorbed roentgen of whole-body radiation would result in some 10,000 cases of leukemia during their lifetime, while one-tenth the "maximum permissible dose" of strontium 90 would result in some 55,000 cases.

The present population of the United States has been reckoned to receive, on the average, some 5 r. of radiation to the gonads from medical exposures alone before the age of 30 (see Laughlin and Pullman) but the amount from all diagnoses and treatments may well be double this (see Schubert and Lapp). About 3 r. are received from the natural background radiation. The amount

from atomic test fallout is as yet much less, and is said to be of the order of .1 r., although atomic warfare, or insufficient precautions in the peacetime use of atomic energy, could raise it enormously. The present exposure of Western populations, caused largely by fluoroscopy and by roentgenograms of the lower trunk, is not enough to cause concern in regard to shortening of life of the exposed generation, but its effect on future generations must be a good deal greater. The same consideration applies to occupational exposures. It has led to the recent recommendations for intensification of radiation precautions promulgated by the National and International Committees on Radiation Protection, and the committees on the genetic effects of radiation of the National Academy of Sciences (United States), the Medical Research Council (Great Britain), the World Health Organization, and the United Nations. It has recently been reflected in increasing activity in this direction in medical and dental circles.

REFERENCES

BERGONIÉ, J. and TRIBONDEAU, L. Interpretation de quelques résultats de la radiotherapie et essai de fixation d'une technique rationelle. C. R. Soc. Biol. (Paris) 1906, 143 (983–985); BOCHE, R. D. Effects of chronic exposure to X radiation on growth and survival, Biological Effects of External Radiation ed. by H. A. Blair (McGraw-Hill, N. Y.) 1954 (222–252): DAMESHEK, W. and GUNZ, F. W. Diagnostic and therapeutic X-ray exposure and leukemia. J. Amer. Med. Association 1957, 163 (838–840); Editorial (anon.) Long-range effect of exposure to radiation, J. Amer. Med. Association 1956, 162 (475); HIMSWORTH, H. and 16 others, The hazards to man of nuclear and allied radiations, Med Res, Counc. (London) 1956 (128+vii pp.); LAUGHLIN, J. S. and PULLMAN, J. Gonadal dose from the medical use of X-rays. Nat. Acad. Sc.-Nat. Res. Counc. (Wash.) 1957 (105 pp.); LEJEUNE, J. Present evidence for genetic induced mutations from the offspring of those exposed to excess radiation. Rep. Com. on Effects of Radiation on Human Heredity, World Health Organization, 1957 (in press); LEWIS, E. B. Leukemia and ionizing radiation, Science, 1957, 125 (in press); MACHT, S. H. and LAWRENCE, P. S. National survey of congenital malformations resulting from exposure to roentgen radiation, Amer. J. Roentgenology and Radium Therapy, 1955, 73 (442–466); MORTON, N. E., CROW, J. F. and MULLER, H. J. An estimate of the mutational damage in man from data on consanguineous marriages, Proc. Nat. Acad. Sci. (U. S. A.) 1956, 42 (855–863); MULLER, H. J. Radiation damage to the genetic material, Amer. Scientist, 1950a, 38 (35–59, 126, 399–425); Our load of mutations, Amer. J. Hum. Genet. 1950b, 2 (111–176); Nature of genetic effects produced by radiation; Manner of production of mutations by radiation, Radiation Biology ed. by A. Hollaender (McGraw-Hill, N. Y.) 1954, 1/1 (351–626); After effects of nuclear radiation, J. Amer. Soc. Safety Engineers, 1956a, 1 (42–48); Ways to reduce genetic radiation hazards, Scope Weekly (Upjohn Co.) 1956b, 1 (1, 13); Further studies bearing on the load of mutations in man, Acta Genet. et Stat. Medica, 1956c, 6 (157–168); Damage from point mutations in relation to radiation dose and biological conditions, Rep. Com. on Effects of Radiation on Human Heredity, World Health Organization, 1957 (in press); NEEL, J. V. and SCHULL, W. J. Effect of exposure to atomic bombs on pregnancy termination in Hiroshima and Nagasaki, Nat. Acad. Sc.-Nat. Res. Counc. 1956, Publ. 461 (241+xvi pp.); NOLAN, W. E. and PATTERSON, H. W. Radiation hazards from the use of dental X-ray units, Radiology, 1953, 61 (625–629); PUCK, T. T. and FISHER, H. W. Demonstration of the existence of mutants with different growth requirements in a human cancer cell strain (HeLa), J. exp. Med. 1957, 104 (427–434); PUCK, T. T. and MARCUS, P. I. Action of X-rays on mammalian cells, J. exp. Med. 1956, 103 (653–666); QUASTLER, H. Modes of acute radiation death, Proc, int. Conf. Peaceful Uses Atomic Energy, Geneva, 1955, 1956, 11 (121–124); RUSSELL, W. L. Genetic effects of radiation in mammals, Radiation Biology ed. by A. Hollaender (McGraw-Hill, N. Y.) 1954, 1/2 (825–860); Shortening of life in the offspring of male mice exposed to neutron radiation from an atomic bomb, Proc. Nat. Acad. Sc. (U. S. A.) 1957, 43/4 (in press); SACHER, G. A. On the statistical nature of mortality, with especial reference to chronic radiation mortality, Radiology, 1956, 67 (250–257); SCHUBERT, J. and LAPP, R. E. Radiation: What it is and how it affects you. (Viking Press, N. Y.) 1957; ULRICH, H. Ein Vergleich der Rontgenstrahlenwirkung auf Kern und Plasma des Deosophila-Eies, Biol. Zent. 1955, 74 (498-515); WEAVER, W. and 15 others, Report of the Committee on Genetic Effects of Atomic Radiation, in Biological Effects of Atomic Radiation, Summary Re-

ports, Nat. Acad. Sc.-Nat. Res. Counc. (Wash.) 1956 (3–31); Whiting, A. R. Absence of mutagenic action of X-rayed cytoplasm in *Habrobracon*, Proc. Nat. Acad. Sc. (U. S. A.) 1950, 36 (368–372); Zavon, M. R. Radiation—helpful or harmful? J. Amer. Med. Association 1956, 162 (532–535); Zirkle, R. E. and Bloom, W. Irradiation of parts of individual cells, Science, 1953, 117 (487–493).

[Reprinted from the Bulletin of the Atomic Scientists, November 1955]

How Radiation Changes the Genetic Constitution

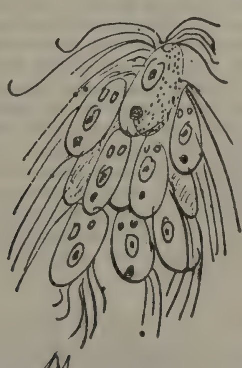
H. J. Muller

Dr. Muller, professor of genetics at Indiana University, prepared the following paper for presentation at the United Nations Conference on the Peacetime Uses of Atomic Energy, at Geneva. Although the paper was not given orally, it will appear in the published proceedings of the Conference.

The changes in the genetic constitution produced by ionizing radiation may for convenience be classified into two major groups: *chromosome aberrations* and *point mutations*.

The chromosome aberrations consist of losses and additions of whole chromosomes or chromosome parts and/or alterations, called structural changes, in the alinement of chromosome parts. Structural changes are caused by the breakage of one or more chromosomes at two or more points, followed by the junction of the fragments at their broken ends, so as to form a new arrangement; that is, a new linear sequence of their component hereditary particles or genes.[1]

Point mutations are changes confined to regions of the chromosomes so small that no loss or addition or change in arrangement of genes can be demonstrated by microscopic examinations or breeding tests. Since structural changes range from "gross" to those so minute as to be at the limit of being detectable as such, there are doubtless other cases of substantially the same kind, but below that limit of size, which become included among the point mutations. However, there is reason to infer that many of the point mutations produced in animals by radiation are not of this kind, but involve changes within the individual genes, and are therefore to be considered as "gene mutations." By this it is meant that these changes are restricted to genetic elements too small to be divided either by the process of normal hereditary recombination (crossing over) or by that of gross structural change. This seems to be true also of the great majority of genetic differences that exist naturally between individuals of the same species; that is, they appear to have arisen as gene mutations.



---

[1] H. J. Muller, J. Genet, 40: 1–66 (1940).

CHROMOSOME ABERRATIONS

The chromosome aberrations produced by radiation in the cells of somatic tissues that replenish themselves by proliferation cause necrosis in much of the tissue descended from these cells and abnormality in much of the surviving descendant tissue. This constitutes a major source of delayed radiation damage, some of it never repaired, in the exposed individual himself. The same series of events, occurring among the immature germ cells of the exposed individual, can result in his or her partial or complete sterility. Among mature and nearly mature germ cells, especially spermatozoa, there is a much higher incidence of induction of these chromosome changes, for any given dose of radiation, than among immature germ cells or somatic cells.[2] Recent evidence[3] confirms the inference[1] that this peculiarity depends upon the chromosomes being in a condensed (tightly spiralized) condition and that it therefore applies also to cells that are in mitosis at the time of irradiation.

Mature sperm or eggs in which chromosome aberrations (actual or potential) have been induced, function in fertilization, but many of the resulting embryos die in consequence of their abnormal chromosome content. Other embryos, in which there has been gross structural change without excess or deficiency of chromosome parts, develop into normal adult individuals. However, when these seeming normals reproduce, recombination occurs between the structurally changed chromosomes derived from one parent and their normal homologues derived from the other parent. In consequence of the nonmatching linear arrangement of the genes from the two parents, about 50 percent of the germ cells now produced have excesses and/or deficiences of chromosome content. These germ cells usually function, but give rise to embryos (of the second generation after exposure) which die in utero at an early stage.[4] This mortality of embryos tends to be repeated over an indefinitely long series of generations. For half of the surviving embryos of such a line of descent, although not themselves containing the lethal excesses or deficiencies, have the grossly changed linear arrangement of genes that, by recombination, again gives rise to these effects.

In modern human populations, there is a tendency to compensate or even overcompensate for reductions in the frequency of viable births, by purposely increasing the number of pregnancies.[5] Hence damage of this kind, once induced, does not tend to die out rapidly but may even spread.

Fortunately there are several factors which serve to limit the frequency with which these cases of inherited abortions are produced. One is the fact that the period spent by male germ cells in a mature or nearly mature state averages, at the very most, a few months, whereas they usually spend some 25 years or more—well over 100 times as long—as immature germ cells, relatively insusceptible to the induction of chromosome aberrations. Although the relative lengths of the corresponding periods for female germ cells are not well established, the germ cells are, even when nearly mature, much less susceptible than spermatozoa to the induction of the gross aberrations that cause inherited abortion.[6] It may be concluded that more than 99 percent of the germ cells which function after a given exposure of limited duration (comprising only a few days or weeks) were at the time of that exposure in an immature stage, relatively insusceptible to the induction of chromosome aberrations. In them, aberrations of all kinds were induced with far lower frequency than point mutations.

Even in the less than 1 percent of germ cells that are exposed to radiation of beta or gamma type during their susceptible stage, gross structural changes of chromosomes will be produced at a low frequency, relatively to point mutations, unless the total dose of radiation received in that period is fairly high, of the order of a hundred or more r. (roentgen) units. This is because the

---

[1] See footnote, p. 1069.

[2] However, some immature germ cell stages are much more susceptible to chromosome alteration than they appear to be when judged by the frequency with which such alterations are found later, on analysis of offspring derived from the cells that had been exposed while immature. This is because the descendant cells derived from those immature germ cells in which the chromosomes had been altered so often die out, and have their places taken by compensatory multiplication of descendant cells derived from those immature germ cells in which the chromosomes had not been altered. This consideration does not apply in the case of point mutations. (Note added October 5, 1955.)

[3] I. I. Oster, Excerpta Medica (8th Internat. Cong. for Cell Biol. No. 8: 406 (1954).

[4] P. Hertwig, Z. indukt. Abstammungs-u. Vererbungslehre 79: 1–27 (1940). P. C. Koller, Genetics 29: 247–63 (1944). G. D. Snell, Amer. Naturalist 68: 178 (1934).

[5] B. Glass, Amer. J. of Human Genetics 2: 269–78 (1950).

[6] G. D. Snell and F. B. Ames, Am. J. Roentgenol. Radium Therapy 41: 248–55 (1939).

production of these aberrations requires at least two chromosome breaks, and these are usually produced independently, by the tracks of different fast particles. On account of being in this sense double or multiple events, these aberrations vary in their frequency according to an exponent of the dose of radiation higher than one (commonly, about 1.5).[1] On the other hand, the point mutations vary as single events, according to the dose itself. Thus, as the dose is diminished, they do not drop off as fast as the structural changes do, and the latter become rare, relatively to the former.

It follows from the above considerations that inherited abortion caused by structural change is a relatively insignificant danger even in the case of a large dose of beta or gamma radiation that has been received in small fractions of not more than a few r. per month. If the amount received in any month is higher than this, however, measures should be taken to avoid this damage. These measures would consist in the prevention or avoidance of conception until the passage of several months after the high exposure. With a very high dose, however, all but the first month of this period would be sterile anyway.

When the exposure has been to alpha or neutron radiation, the production of gross structural changes tends to vary with the dose itself instead of with a higher exponent.[7] This is because both the breaks participating in such an aberration are usually produced by activations arising from the track of the same fast atomic nucleus. In consequence, both of this proportionality of the frequency of structural change to dose with this type of radiation, and of the fact that the more densely crowded activations from such radiation are actually more efficient in breaking the chromosomes, much lower doses, in reps, of neutrons or alpha rays than of gamma, beta, or X-rays give significant numbers of structural changes. Hence, the rule of not reproducing within some months after exposure should be applied in this case of much lower doses when the radiation has been of these types. In order to gage how low this limit should be placed, it should be taken into consideration that even 5 reps of neutrons, applied to spermatozoa, may be estimated to induce inherited abortion, based on gross structural change in some 1 to 6 among every thousand viable individuals derived from these spermatozoa.[8]

The frequency of natural occurrence of gross structural changes giving inherited abortion has not been studied extensively in mammalian populations, but it is known to be low. The highest recorded figure[9] is about 6 percent. Among offspring from spermatozoa treated with 500 r. X-rays, 25 percent have been reported by 2 observers.[10]

CHARACTERISTICS OF NATURAL POINT MUTATIONS

Among the genetic changes induced by exposure to radiation from artificial sources the point mutations are far more frequent and significant that the chromosome aberrations. Among the genetic changes that arise from natural causes (those somewhat misleadingly referred to as "spontaneous") the point mutations are still more frequent and important, as compared with the chromosome aberrations. Any ordinary population contains a large accumulation, or "load," of these natural point mutations, which have arisen in the course of many past generations. If any new point mutations are induced by radiation these are added to this already existing load of mutations. They thereupon become lost to view among the latter, in the sense that, with rare exceptions, the origin of any individual point mutation cannot be traced to the radiation. Thus, in order that radiation mutations may be viewed in due perspective, certain salient facts about the natural mutations should first be passed in review.

Natural point mutations occur sporadically. They are not individually controllable. Any such mutation may be thought of as resulting from an accidental ultramicroscopic encounter between a gene and some atom group, particle, or photon to which the gene happens, under the circumstances, to be vulnerable. It is probable that, on occasion, instead of the original or "mother-gene" becoming

---

[1] See footnote, p. 1069.

[7] A. Catsch, O. Peter, and P. Welt, Naturwissenschaften, 32: 230–31 (1944). N. H. Giles, Jr., Proc. Natl. Acad. Sci. U. S. 26: 567–75 (1940). N. H. Giles, Jr., Genetics 28: 398–418 (1943). H. J. Muller, Amer. Naturalist 88: 437–59 (1954). H. J. Muller and J. I. Valencia, Records Genet. Soc. Am. 20: 115–16 (1951); Genetics 36: 567–68 (1951).

[8] G. D. Snell, Proc. Natl. Acad. Sci. U. S. 25: 11–14 (1939).

[9] P. Hertwig, Biol. Zentr. 58: 273–301 (1938). P. Hertwig, Z. indukt. Abstammungs-u. Vererbungslehre 79: 1–27 (1940).

[10] W. L. Russell, "Genetic Effects of Radiation in Mammals," Radiation Biology (edit. by A. Hollaender), McGraw-Hill Co., New York, Vol. 1, Pt. II, Chapt. 12, pp. 825–59 (1954).

altered, the accident causes a misstep in the construction of the "daughter-gene," but the effect is much the same as if the old gene had itselm mutated. In either case, point-mutational changes are permanent. This implies that the changed gene tends to be very stable, as the original gene was, and that in reproducing it continues to give rise to daughter genes like itself, that is, in this case, of the new type. Thus it "copies itself" through an indefinite succession of generations.[11]

The frequency of mutations in general is influenced, however, by many conditions. Thus, cells in certain developmental stages have mutations occurring more frequently in them, in other stages less frequently.[12] There is some evidence that markedly detrimental disturbances in the cellular biochemistry, of whatever nature, tend to favor the occurrence of mutations, while the functioning of the cell within its normal range is associated with a low mutation frequency. Certain special substances, such as the mustard gas series, some organic peroxides and expoxides, and triazine are so conducive to mutation that they have been termed "mutagens."[13] Some of them can in fact be used to induce mutations at about as high a frequency as with radiation. When the distribution of relative frequencies of the different types of mutations induced by one mutagenic agent is compared with that induced by another, or with that of spontaneous mutations, considerable differences are often found, even though most types of mutations produced by one agent are also produced to some extent by any other, and also arise spontaneously but at a lower rate.[14]

The partial selectivity of action of mutagens does not give evidence of being of such a nature as to result in the mutations produced by a given agent, or under given conditions, being better adapted, as a group, for life in the presence of that agent, or under those conditions, than are the mutations which arise under other circumstances. That is, mutations arising independently of radiation like those produced by radiation are, so far as the organism is concerned, accidents, not adaptive responses. There is evidence indicating that the organism has, through a long period of evolution, been selected for the maintenance of biochemical operations which give it as low a frequency of "natural" mutations as can practicably be attained just as it has been also selected to react in such ways as to minimize the occurrence of other accidents.[15]

It is entirely in line with the accidental nature of natural mutations that extensive tests have agreed in showing the vast majority of them to be detrimental to the organism in its job of surviving and reproducing, just as changes accidentally introduced into any artificial mechanism are predominantly harmful to its useful operation.[11] According to the conception of evolution based on the studies of modern genetics, the whole organism has its basis in its genes. Of these there are thousands of different kinds, interacting with great nicety in the production and maintenance of the complicated organization of the given type of organism. Accordingly, by the mutation of one of these genes or another, in one way or another, any component structure or function, and in many cases combinations of these components, may become diversely altered. Yet in all except very rare cases the change will be disadvantageous, involving an impairment of function.

It is nevertheless to be inferred that all the superbly interadapted genes of any present-day organism arose through just this process of accidental natural mutation. This could take place only because of the Darwinian principle of natural selection, applying to the genes. That is, on the rare occasions when an accidental mutation did happen to effect an advantageous change, the resultant individual, just because it was aided by that mutation, tended to multiply

[11] H. J. Muller, Second Internat. Cong. Eugenics, N. Y., Abstracts, p. 7–8 (1921). H. J. Muller, Genetics 3 : 442–99 (1918). H. J. Muller, J. Exp. Zool. 31 : 443–73 (1920). H. J. Muller, Amer. Naturalist 56 : 32–50 (1922). H. J. Muller, "Mutation," Eugenics, Genetics, and the Family, Williams and Wilkins, Baltimore, Vol. I, pp. 106–12 (1923). H. J. Muller, Genetics 13 : 279–357 (1928).

[12] H. J. Muller, Yrbk. Amer. Philos. Soc. for 1945 : 150–53 (1946). H. J. Muller, Genetics 31 : 225 (1946). H. J. Muller, unpublished data.

[13] C. Auerbach, Cold Spring Harbor Symposia Quant. Biol. 16 : 199–213 (1952). C. Auerbach and J. M. Robson, Nature 157 : 302 (1946). M. J. Bird, J. Genetics 50 : 480–85 (1952). M. Demerec, G. Bertani, and J. Flint, Amer. Naturalist 85 : 119–36 (1951). K. A. Jensen, I. Kirk, G. Kolmark, and M. Westergaard, Cold Spring Harbor Symposia Quant. Biol. 16 : 245–62 (1952). J. A. Rapoport, Compt. Rend. Acad. Sci. U. R. S. S. 54 : 65–67 (1946). J. A. Rapoport, Bull. Biol. Med. Exp. U. R. S. S. 23 : 198–201 (1946). J. A. Rapoport, Compt. Rend. Acad. Sci. U. R. S. S. 61 : 713–15 (1948) O. Wyss, J. B. Clark, F. Haas, and W. S. Stone, J. Bact. 56 : 51–57 (1948)

[14] M. Demerec, Proc. Amer. Philos. Soc. 98 : 318–22 (1954).

[15] H. J. Muller, Genetics 3 : 422–99 (1918). A. H. Sturtevant, Quart. Rev. Biol. 12 : 464–67 (1937).

more than the others. By the continuance and repetition of this process, the type that had been normal became supplanted by other types, that were at least better adapted for life in certain particular environments, in in certain ways. Thus, the mutant gene of the previous era became the normal gene of today, and the whole system of genes of the species tended to become even more differentiated and highly organized. Yet at each stage the great majority of new mutations, if examined before being put through the sieve of selection, must have been detrimental to life or to reproduction, as they are today in all species studied, no matter what the degree of advancement of the species.

As important for the survival of a species as the differential multiplication of the few better adapted mutants is the reduction in number and eventual dying out, in competition with the "normal" type, of the much more numerous mutants that are less fit than the normals. Since each generation supplies a fresh crop of these mutations, to be added to those inherited from earlier generations, it is obvious that without this negative selection the system of genes would undergo continued decay. Thus after a time it would become completely heterogeneous, disorganized, and degenerate.[10] In the past, only natural selection has saved it. This selection makes it practically inevitable that any detrimental mutation, no matter how small its harmful effect, will in the long run become limited by tipping the scales against some descendant who carries it, causing his premature death or failure to reproduce.

However, this dying out of the unfit mutants is in most cases rather long delayed. One reason for this delay is the fact that mutant genes are in the great majority of cases heterozygous, that is, present in individuals who have received the corresponding normal gene from their other parent, and that in such a situation the normal gene usually produces most of the effect. The normal gene is for this reason said to be "dominant," and the mutant gene "recessive," even though the mutant is seldom completely without expression when heterozygous.

Another reason for the delay in the dying out of mutant genes lies in the fact that even in those relatively rare individuals who are "homozygous" for a given mutant gene, by reason of having inherited that same gene from both parents, the amount of abnormality is often not very great. Hence, even in this situation the gene usually confers a much less than 100 percent risk of premature death, or of failure to reproduce. It may be noted in this connection that the idea that most mutations are monstrosities or freaks is a popular misconception. In fact, only a tiny minority of mutations cause very conspicuous visible abnormalities.

CALCULATION OF NATURAL MUTATIONS PRESENT IN A POPULATION

The total number of point mutations (or, more correctly, of point-mutant genetic conditions) present in any population at a given time is a product of two interacting numerical factors. The first factor, a, is the total number of new point mutations that arise in one generation. The second factor, b, to be multiplied by the first, is termed the persistence. It represents the total number of individuals of successive generations by whom, on the average, any given mutation, present at first in one individual, comes to be inherited.[16] This same relation holds for mutations of any particular type as well as for the totality of mutations.

Obviously b, the persistence, depends upon the ability of the individuals carrying the mutation to live and breed, as compared with normal individuals. If, for simplicity, we assume the whole population to be of stable size, then b, for the average mutation, or for any given type of mutation, is the reciprocal of c, the average chance that an individual who has inherited it will be killed prematurely, or will fail to reproduce, as a result of the one or more functional impairments occasioned in him by that mutation. In getting this average chance of elimination, c, we must estimate the relative frequencies of individuals heterozygous and homozygous for the mutation, and multiply the chance of elimination of each of these types, taken separately, by its relative frequency. When this is done it is found that usually, despite the much smaller

---

[10] See footnote, p. 1071.

[16] H. J. Muller, Amer. J. Human Genet. 2: 111–76 (1950). H. J. Muller and S. L. Campbell, unpublished data. (This reference was omitted from original. Note added October 5, 1955).

detrimental effect in the heterozygous individuals, their relatively large numbers cause most of the eliminations, and most of the total genetic damage to the population, to occur in this group. Thus, in most cases, the homozygous group can for practical purposes be ignored.[16]

In order to apply this method of calculation to human populations we must first have estimates of *a* and *b*. At present such estimates are very indirect, and serve only to indicate a broad range, within which, somewhere, the actual value is probably located. The fruitfly *Drosophila* has thus far been the only organism in which anything like a direct approach has been made to an observed value for either *a* or *b*, and even here the results are subject to very large errors. In this material it can be estimated that, in a population of 100 million, *a*, the number of new mutations arising naturally per generation that becomes transmitted to the next generation, is on the average at least 8 million, and that *b*, the persistence or average number of individuals of successive generations which finally come to inherit any given mutation, is considerably more than 20 and probably more than 40. This makes *ab*, the number of mutations carried by the population of a hundred million in any given generation, probably more than 320 million, that is, probably more than three per individual.

The estimate of *a* for *Drosophila* was obtained by first taking the observed frequency, 0.18 percent, with which "recessive" fully lethal mutations (those that invariably kill homozygous individuals) usually arise in the X chromosome per germ cell per generation when no mutagenic treatment is used.[12] This figure was then multiplied by 6, the ratio of recessive lethals in all the chromosomes to those in the X chromosome. This figure had to be obtained from experiments in which radiation was applied to spermatozoa.[17] The product, 1.08 percent, representing all lethals, was in turn multiplied by 4, the ratio which all mutations detrimental enough to have been detected by a given technique were found to bear to fully lethal mutations. This figure 4 also was based on radiation mutations.[18] Finally, the second product, 4.3 percent, was multiplied by 2, because each individual results from 2 germ cells, and the resultant percent, 8.6, was multiplied by 100,000,000, the number assumed to exist in the population.

That the application of the ratio 6, derived from radiation work, to natural mutations is legitimate has been shown by special tests. However, among natural mutations as a group, the ratio of all mutations to lethals is probably a good deal higher than 4, the ratio found among mutations produced by irradiating spermatozoa. For the radiation mutations include a greater proportion of structural changes and these are more often lethal. This is one reason why the final figure for *a* is very conservative. The other reason is that the methods of detection used failed to find mutations that produced less than about 10 percent risk of premature death, even if they caused considerable infertility, and such mutations may have been relatively numerous.

The figure for *b* is based on tests, carried out independently by two groups of investigators,[16, 19] to determine how much risk of premature death is conferred by a "recessive" lethal mutation when it is heterozygous. In both cases an average figure of about 3 percent to 5 percent risk of death was obtained. This would result in only 1 heterozygous individual among some 25 being killed and would hence allow the average lethal a persistence of 25. That is, it would tend to be passed on to some 25 individuals, on the average, before it died out. Since, however, a considerable majority of mutations are not so detrimental as to be fully lethal when homozygous, and most of them are probably not even 50 percent lethal when in that condition, the figure of 1 in 25 (4 percent) for the risk of death when heterozygous must be considerably higher than that holding for the average mutation, and the persistence, being the reciprocal of this, would be considerably higher than 25. That is why 40 was used as a better guess for *b*, but the observed distribution of mortalities indicates that even it is likely to be too low.

Before we can convert our figure *a* for newly arising mutations in *Drosophila* into a corresponding figure *a* for man we must obtain some indication of the

---

[12] See footnote, p. 1072.
[16] See footnote, p. 1073.
[17] R. L. Berg, Genetics 22 : 225–40 ; 241–48 (1937).
[18] J. J. Kerkis, Summ. Commun. XV. Int. Physiol. Congr. 198–200 (1935). J. J. Kerkis, Izv. Akad. Nauk SSSR 75–96 (1938). H. J. Muller, Verh. 4, Int. Kongr. Radiol. 2 : 100–02 (1934). N. W. Timofeeff-Ressovsky, Strahlentherapie 51 : 658–63 (1934). N. W. Timofeeff-Ressovsky, Nachr. Ges. Wiss. Gottingen N. F. 1 : 163–80 (1935).
[19] C. Stern, G. Carson, M. Kinst, E. Novitski, and D. Uphoff, Genetics 37 : 413–49 (1952). C. Stern and E. Novitski, Science 108 : 538–39 (1948).

ratio of mutation frequency in *Drosophila* to that in man. As yet the only line of approach to this problem lies in a comparison of the frequencies, in the two species, of natural mutations that produce certain specific effects, and that may be inferred to occur at given highly limited positions in the chromosomes. Although the evidence of this kind is meager and imperfect, there is enough of it to show that in *Drosophila* a mutation of any one specific type, located in a specific chromosomal position (so as to give rise to what is technically known as an "allele" or "pseudo-allele" of some preexisting mutation) arises, on the average, with a natural frequency of between 1 in 100,000 and 1 in 300,000 germ cells, the most likely figure being about 1 in 200,000.[20] In mice there is little published data of this kind as yet but it would indicate a figure in the range between 1 in 40,000 and 1 in 400,000 or, most likely, about 1 in 140,000.[21] In man, an estimate of between 1 in 50,000 and 1 in 100,000 has been arrived at, on the basis of a much larger amount of data than in either mice or *Drosophila*, but the uncertainties of the methods used in man are much greater.[22] These apparent differences in mutation frequency between the three species may well correspond to the different numbers of cell divisions which take place in their respective reproductive cycles, since these numbers for flies, mice, and men are related about as 1: 1.5: 2. At any rate, it is likely that the average frequency of mutations of any specific type in man is higher than in *Drosophila*, probably from 2 to 4 times as high. To be conservative, we will adopt the lower figure, 2.

It is, however, likely that the ratio of frequencies of specific mutations in man to those in the fly would not be nearly as high as the ratio of total mutation frequencies in man to those in the fly. For man, and mammals in general, give evidence of having a more complicated organization, all told, than the fly, especially when the complications of the nervous system are taken into account. Mammals may therefore be expected to have a more complex germ plasm than flies, one in which a larger number of different kinds of mutations of specific types can occur. This agrees with existence of a larger amount of the genetic substance, polymerized deoxyribonucleic acid, in mammalian than in fly chromosome sets. Therefore we are in all probability obtaining a low minimal figure for a in man if we multiply the *Drosophila* a by only two.

For the value of b in man or other mammals there is as yet little basis for a decision. The existing indications point strongly to the conclusion that natural mutations in mammals in general, including man, are, as in *Drosophila*, prevailingly recessive, yet not completely so. Moreover, they certainly include a fairly abundant group of "recessive" lethals, but it is probable that mutations having a lesser degree of detriment are more frequent than lethals. At this preliminary stage of our knowledge of the subject, then, we have little ground for using a markedly different value of b for mammals than for *Drosophila*.

The figure of about 6.5 is thereby arrived at as a minimal one for the content of recessive, definitely detrimental mutations (including lethals) per individual human being. In a preliminary calculation using related methods, the figure 8 was arrived at.[16] These estimates, as recently shown by Slatis,[23] can be checked in a more direct way. The method consists in observations of the frequency with which homozygous individuals, showing the more definite abnormality often associated with a homozygous mutation, appear among the offspring of marriages between close relatives. Application of this technique has led Slatis, very tentatively as yet, to the figure 8 as the most probable present approximation to the number of natural mutations of the kind in question for which a person is, on the average, heterozygous. This method now needs to be applied on a much larger scale but the present result is enough to be reassuring, in indicating that our mode of calculation is giving figures of the right order of magnitude.

It should be emphasized that in these calculations we are dealing only with these mutations which are detrimental enough to give a "sizable" risk of genetic extinction by way of premature death; that is, one as great as about 0.5 percent in the case of the heterozygous individual, or 10 percent in the case of the homozygous one. We do not know how many mutations arise which are less harmful than this, or which cause extinction mainly by their interference

[16] See footnote, p. 1073.
[20] H. J. Muller, J. I. Valencia, and R. M. Valencia, Rec. Genet. Soc. Amer. 18: 105–06; and Genetics 35: 125–26 (1950).
[21] W. L. Russell, Cold Spring Harbor Symposia Quant. Biol. 16: 327–35 (1952).
[22] J. B. S. Haldane, Proc. 8th Internat. Cong. Genet. (1948); Hereditas 35 (Suppl.): 266–78 (1949). J. V. Neel, and H. F. Falls, Science 114: 419–22 ((1951).
[23] H. M. Slatis, Amer. J. of Human Genet. 6: 412–18 (1954).

with reproduction, not with life itself. However, even if there are relatively few, those few which have an average grade of detriment within the same order of magnitude as the frequency of their origination by mutation, will accumulate so as to be inordinately numerous in the population. They will provide a very considerable proportion of the superficially observable genetic variability. Moreover, the frequency of the different types of mutations of this group will differ greatly from region to region, in response to differences in the conditions of selection, as well as to random influences.

Since the frequency with which mutant genes of any given degree of detrimental effect exist in the population at any one time is the product ab, where b is inversely proportional to c, the degree of detrimental effect, it is evident that the existing mutant genes have a distribution, with respect to their harmfulness, very different from the distribution to be found on examining mutations as they arise. For, among the mutant genes as they exist in the population as compared with them at their origination, the less harmful ones are (in inverse proportion to their harmful effect) more numerous than the more harmful ones. For that very reason each slightly harmful mutation that arises tends to cause as much detriment to the population as a whole in the end as each drastically harmful or lethal mutation does, since it compensates for its relatively small degree of harm by afflicting correspondingly more individuals. In consequence, the total amount of genetic damage done to a population by mutations is much more closely proportionate to the total frequency of mutations arising per generation (a/N, where N is the number of individuals in the population) than to the frequency of mutations existing in the population (ab/N).[24] If a is raised or lowered, however, it may take scores of generations before its changed value becomes proportionately reflected in the altered average fitness or mutational load of the population. A similar lag occurs if b is altered, as happens when the rigor of selection is increased or decreased.

CHARACTERISTICS OF POINT MUTATIONS PRODUCED BY RADIATION

In the plant material studied by Stadler,[25] evidence was obtained, based on the intensive study of a few types of mutations, that the great majority of apparent point mutations induced by radiation probably consisted of losses of a small section of a chromosome including more than one gene, unlike what was usually true of the natural mutations. In the animal material best studied with reference to this question, that of *Drosophila*,[26] there is evidence that such "sectional deficiencies" do comprise a good deal larger proportion of the point mutations obtained by irradiation of the mature germ cells than of the point mutations arising naturally. However, the apparent point mutations produced by irradiation of immature germ cells of *Drosophila* do not include substantially more that on further analysis prove be be demonstrable "sectional deficiencies" than are found among the natural mutations. Moreover, the characteristics of the effects produced on the individual, both in *Drosophila* and mice,[10, 27] also indicate that a large proportion of these radiation-mutations are as truly changes within the genes as are the mutations of natural origin.

In general, then, in the animal material, the radiation-mutations strongly resemble the natural ones. Practically all types of natural point mutations that have been looked for in extensive irradiation experiments have been found to be produced by radiation also. Like natural mutations, of course, the great majority, although not quite all, of those produced by radiation, are detrimental. Moreover, the great majority have far less dominance (i. e., less expression in the heterozygous individual) than the normal genes from which they arose. Once arisen, the radiation mutations, like the natural ones, are permanent, reproducing themselves as such.[28]

---

[10] See footnote, p. 1071.

[24] J. B. S. Haldane, Amer. Naturalist 71: 337–49 (1937). (Volume and year given through typographical errors as 11 and 1949 in original. Note added Oct. 5, 1955.)

[25] L. J. Stadler, Cold Spring Harbor Symposia Quant. Biol. 9: 168–77 (1941). L. J. Stadler, Science 120: 811–19 (1954). L. J. Stadler and H. Roman, Genetics 33: 273–303 (1948).

[26] H. J. Muller, J. I. Valencia, and R. M. Valencia, Genetics 35: 126 (1950).

[27] H. J. Muller, "Gene Mutations Caused by Radiation," Symposium on Radiobiology, John Wiley & Sons, New York, Chap. 17, pp. 296–332 (1952).

[28] H. J. Muller, Cold Spring Harbor Symposia Quant. Biol. 9: 151–65 (1941).

Just which mutation is produced by radiation on a given occasion is of course a matter of "accident," as is true of natural mutations. However, the total frequency of the mutations produced by a given dose of radiation varies to some extent with the accompanying conditions, as in the case of natural mutations, although the conditions in question are to some extent different ones in the two cases. The conditions which influence the production of point mutations by radiation include genetic differences, differences in cell type or stage, differences in metabolic reactions, and (a category overlapping the previous one) differences caused by the application of special chemical or physical treatments.

For the most part, the same influences have been found to promote or hinder the action of radiation in causing point mutations as in causing structural changes of chromosomes. For example, chromosomes in condensed stages are more susceptible to the induction of changes of both types. Some findings of interesting differences in this respect have been reported, however. Among these are the observations that sperm cells of *Drosophila* several days prior to their release, and therefore perhaps in the spermatid stage, are much more susceptible than mature spermatozoa to the production of radiation of structural changes, but not of point mutations.[29]

In accordance with the view, first proposed by Rapoport[30] on the basis of chemical work by Fricke,[31] that the mutagenic action of radiation is exerted via the production of actively oxidizing radicals or molecules, it is found that radiation mutagenesis of both major types is positively correlated with the amount of free oxygen present at irradiation. Physical or chemical influences which appear directly or indirectly to increase or decrease the abundance of oxygen available for conversion into mutagenic radicals influence correspondingly the frequency of mutations produced. There is, to be sure, evidence indicating that not all the mutagenic action of radiation takes the same pathway, and that some of it may be quite unconnected with oxidation. But, however that may be, the above and other findings, by demonstrating the conditional nature of radiation mutagenesis, constitute a disproof of the target hypothesis of such mutagenesis, at least in the simplified form in which it had sometimes been applied.[32] Moreover, these findings are of considerable practical value in having led to the working out of treatments, described in other sections of this conference,[33] which give hope of affording significant protection against the mutagenic action of radiation. The fact that certain treatments, even when given after irradiation aid in such protection, is especially noteworthy, both from a theoretical and from a practical standpoint.

[29] K. G. Lüning, Acta Zool. 33 : 193–207 (1952).
[30] J. A. Rapoport, Zhur, Obshchei Biol. 4 : 65–72 (1943).
[31] H. Fricke, J. Chem. Phys. 2 : 556–57 (1934). H. Fricke, Cold Spring Harbor Symposia Quant. Biol. 3 : 55–63 (1935).
[32] H. J. Muller, J. Cellular Comp. Physiol. 35 (Suppl. 1) : 9–70 (1950).
[33] A. Hollaender, Science 121 : 624 (1955). A. Hollaender, W. K. Baker, and E. H. Anderson, Cold Spring Harbor Symposia Quant. Biol. 16 : 315–26 (1952).

In material of varied kinds, but more especially in *Drosophila*, there is good evidence that over a considerable range of dose (in *Drosophila*, from some 50 r. to more than 1,000 r., a more than twentyfold range) the frequency of point mutations (like that of chromosome breaks) is directly proportional to dose.[34] Moreover, they are independent of the timing of the dose, over an enormous range, provided cellular conditions are held constant.[35] Below 25 to 50 r. the mutation frequency is so low that it has hitherto been impossible to obtain sufficient data, and above 1,000 or 2,000 r. the determination of frequency may be interfered with by a selective elimination (through chromosome aberations) of the cells that happened at irradiation to be in a more susceptible state.[36] Since, however, in the work with low doses and low time-rates of delivery of gamma radiation the germ cells of some series were traversed by only one electron track in a period of a half hour or more, on the average, and still showed a frequency of mutations proportional to the total dose, there is reason to infer that no dose or intensity of such radiation is without its proportionate production of point mutations. Moreover, if this is true of gamma radiation it must be at least as true of radiation producing tracks more densely crowded with ionizations.

Despite the equal mutagenic efficiency of different doses and dose rates of ionizing radiation, it is not necessary to infer that a point mutation or a break is ordinarily the consequence, direct or indirect, of a single activation or even of a single ionization. For all the ionizing radiation studied, has some of its ionizations produced in clusters of minute diameter. If two or more ions commonly cooperate mutagenically, however, it might be thought that this would become evident by causing the frequency of mutations to vary as the square or some higher power of the dose. Yet this would not be true if those ions had to be as near together as the ones in a natural cluster, for such close juxtaposition as this would not be brought about with appreciable frequency by raising the dose and the dose rate within toleration limits.

That such cooperation in mutagenesis does occur is indicated by recent observations to the effect that fast neutrons appear to be approximately twice as efficient as X or gamma rays in inducing point mutations in the chromosomes of *Drosophila* spermatozoa,[37] and are probably a good deal more efficient still, relative to X or gamma rays, in inducing chromosome breaks.[38] Presumably alpha rays likewise would be more efficient than X or gamma rays in these respects. One possible interpretation of this higher effectiveness of fast neutrons would be provided, on the Watson-Crick hypothesis of the structure of the genetic material, by the doubleness of the fibers in which the rearrangements are produced, if we suppose that the occurrence of the mutation or break is much facilitated when both fibers are simultaneously affected.

The effectiveness of fast neutrons in inducing point mutations is actually higher than it appears to be, because intensive studies of given cases of these seeming point mutations have shown that in fact a considerable proportion of them involve a double or multiple effect within a very localized chromosome region.[39, 40] This greater clustering of effects with neutrons than with X or gamma rays is to be expected, in view of the greater concentration of the ionizations in the tracks of the ionizing particles released by neutrons, provided that the mutational effects arise in close proximity with the activations that induce them. Since this clustering of effects causes many of them to be lost to

---

[34] C. P. Oliver, Z. indukt. Abstamm. u. Vererbungslehre 61; 447–88 (1932). W. P. Spencer and C. Stern. Genetics 33: 43–74 (1948). N. W. Timofeeff-Ressovsky, Experimentelle Mutationsforschung in der Vererbungslehre, T. Steinkopf, Leipzig (1937).

[35] K. G. Lüning, B. Lindell, and R. Falk, Acta Radiol. 43: 89–92 (1955). J. T. Patterson, Biol. Bull. 61; 133–38 (1931). S. P. Ray-Chaudhuri, Proc. Roy Soc. Edinburgh B62; 66–72 (1944). N. W. Timofeeff-Ressovsky and K. G. Zimmer, Strahlentherapie 53: 134–38 (1935). D. E. Uphoff and C. Stern, Science 109: 609–10 (1949).

[36] H. J. Muller, I. H. Herskowitz, S. Abrahamson, and I. I. Oster, Genetics 39: 741–49 (1954).

[37] P. T. Ives, R. P. Levine, and H. T. Yost, Jr., Proc. Nat. Acad. Sci. USA 40: 165-71 (1954). G. H. Mickey, Amer. Naturalist 88: 241–55 (1954). H. J. Muller, Rec. Genet. Soc. Amer. 23: 58 and Genetics 39: 985 (1954).

[38] W. K. Baker and E. Von Halle, Science 119: 46–49 (1954). A. D. Conger, Science 119: 36–42 (1954). J. S. Kirby-Smith and C. P. Swanson, Science 119: 42–44 (1954). W. L. Russell, Liane B. Russell and A. W. Kimball, Amer. Naturalist 88: 268–86 (1954).

[39] H. J. Muller and J. I. Valencia, Rec. Genet. Soc. Amer. 20: 115–16; and Genetics 36: 567–68 (1951).

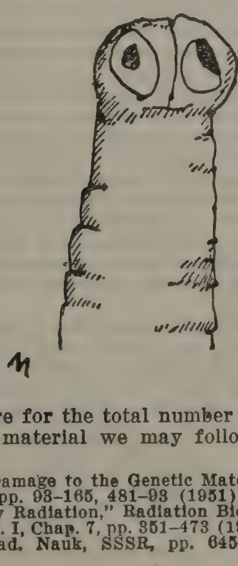
[40] H. J. Muller, "The Manner of Production of Mutations by Radiation," Radiation Biology (edited by A. Hollaender), McGraw-Hill Co., New York, Vol. I, Chap. 8, pp. 475–626 (1954).

view by reason of their proximity to each other (except when special techniques of analysis are used), the mutagenic potentiality of the fast neutrons is correspondingly underrated in most experiments. So far as genetic damage to the population is concerned, however, a double or multiple effect of the given kind adds no more to the mutational load than does a single effect. Hence for present purposes fast neutrons may be regarded as no more than twice as effective as X or gamma rays in producing point mutations.

ESTIMATION OF THE TOTAL POINT MUTATIONAL DAMAGE FROM A GIVEN AMOUNT OF RADIATION

It has been noted that the important quantity in the determination of the total amount of genetic damage is not the amount of harm done to the individuals who have inherited the mutations in question but only the total number of these mutations. For a mutation doing less harm to an individual will, as if in compensation, be passed down to a correspondingly larger number of descendant individuals. It has also been noted that an approach to a direct estimation of the total number of mutations arising has thus far been made only in *Drosophila*, and that this calculation has involved the use of data from radiation experiments. This work can therefore be applied to the estimation of the total damage arising from a given dose.

The principles have already been explained whereby a minimum value for the total number of mutations is obtained by getting the number of lethals in the X chromosome and then multiplying this by 6, to get the number of lethals in all the chromosomes, and again by 4, to get the total number of mutations causing at least 10 percent detriment to life, when homozygous. (A correction is made in this calculation, based on certain tests, in order to estimate the number of point mutations without including the structural changes.) When this calculation is carried out, using the results obtained at any given dose, the resulting number can then be expressed in terms of the total number of point mutations produced by a single r. unit, by using the principle of proportionality of point-mutation frequency to dose. It is then found that this number turns out to be about 1 mutation among 2,000 germ cells per r. (that is 5×10^{-4}/r.) for X or gamma rays applied in the usual way to mature spermatozoa.[41] The more important figure, representing the result of irradiation of the more prevalent stages (gonia) of immature germs cells of adult *Drosophila*, is only a fourth to a half of this, according to the conditions. It is probable that there are even lower values for certain other immature stages of *Drosophila* germ cells, as for instance those in the embryonic polar cap.[42]



In order to obtain a figure for the total number of mutations produced by a given dose in mammalian material we may follow the procedure which we

[41] H. J. Muller, "Radiation Damage to the Genetic Material," Science in Progress, Yale University Press, New Haven, pp. 93–165, 481–93 (1951). H. J. Muller, "The Nature of the Genetic Effects Produced by Radiation," Radiation Biology (edited by A. Hollaender), McGraw-Hill Co., New York, Vol. I, Chap. 7, pp. 351–473 (1954).

[42] Z. I. Berman, Izvest. Akad. Nauk, SSSR, pp. 645–78 (1939). Helen U. Meyer, unpublished data.

adopted in the calculation of natural mutations. This involved a comparison of the mutation frequencies involving particular types of mutations, located in given positions on the chromosomes, in *Drosophila* and in mammals, and then applying the ratio thus found to the figure for total mutation frequency in *Drosophila* so as to convert it into the presumed corresponding value (a minimal one) for mammals.

Fortunately, there is available for this comparison a much more reliable body of data, for both groups of organisms, than that which we had recourse to in the case of natural mutations. The average frequency of point mutations of the kind in question in *Drosophila*, based on a study of 10 types (loci), was found to be about 1.4×10^{-8}/r. for any given type, when the radiation was applied to inactive immature germ cells (oögonia).[43] The different types seldom varied from one another in frequency by a factor of more than 2 in material abundant enough for judging this matter (that in which spermatozoa had been irradiated.

In the mammalian material, comprising irradiated spermatogonia of mice, Russell[20] has reported an average mutation frequency of about 25×10^{-8}/r., based upon 7 specific types of mutations (loci). Here the range of variation between the different types was greater than in the above *Drosophila* material, but their mean agreed well with their mode, and 4 of the 7 types conformed fairly closely with this mean. It is clear on comparison of the two sets of results that the susceptibility of the mammalian material is at least an order of magnitude higher than that of the flies, the observed factorial difference in results being 18. To obtain a minimum estimate of the total frequency of mutation in the mice we must therefore multiply by 18 the figure arrived at for gonial cells of flies. (It is of no consequence that in the flies oögonia were studied and in the mice spermatogonia, since special comparisons[44] have shown these two cell types to be alike in mutagenic susceptibility as they are expected to be.) Since the figure for the gonia of flies had a lower limiting value of 1.25×10^{-4}/r. the minimum value for mice becomes 2.25×10^{-3}/r. This is the frequency for a germ cell, not for an offspring derived from two such germ cells; for the offspring it would be 4.5×10^{-3}/r.

In performing this calculation we are, as in the case of the natural mutations, assuming that the hereditary material of mammals is no more compound than that of flies; i. e., that there is not a greater number of different specific types of mutations in mammals than in flies, despite their seemingly more complicated organisms and their larger amount of deoyribonucleic acid. The total frequency of mutations per r. may be a good deal higher than here calculated not only because of the inadequacy of this assumption but also because weakly detrimental mutations and those mainly affecting fertility rather than individual survival have not been included. Moreover, only the lower limiting value for the somewhat variable mutation frequency of fly oögonia was used. All this emphasizes the fact that our estimate is decidedly on the "conservative" side.

At the same time, it is true that the value is one for mice, not human beings. All that can be said to this is that, so long as we lack data on an organism still closer to man, it is necessary, provisionally, to base our judgments on this result, and that, since mice are so much closer to men than flies are in almost every other important respect, it would be strange if they were not closer in their mutagenic properties as well. Moreover, the factors which might be expected to cause a significant difference in the natural mutation frequencies of mice and men—their great discrepancies in length of life, size, and number of cell generations in the reproductive cycle—would not be expected to exert significant influences on the frequency with which mutations are produced in them by radiation.

The minimum figure of 4.5×10^{-3}/r. point mutations for the offspring of parents both of whom were exposed can be expressed in the form: "at least 1 induced point mutation per offspring, on the average, for each 220 r. of exposure to both parents. From this it is evident that many of the children who were conceived by Hiroshima survivors at any time after their exposure must have contained one or more mutations induced by the radiation. Similarly, children conceived

---

[20] See footnote, p. 1075.
[43] H. J. Muller, Seventh Annual Report to Amer. Cancer Soc., Inc., pp. 120–21 (1952).
[44] K. V. Kossikov, Genetics 22: 213–24 (1937). R. I. Serebrovskaya and N. I. Shapiro, Compt. Rend. Acad. Sci. U. R. S. S. 2: 421–28 (1935).
NOTE.—Only when these two papers are taken in conjunction with one another does the equal mutagenic susceptibility of gonial cells of the two sexes become evident.—**H. J. M.**

by parents both of whom have been exposed to the so-called "permissible dose" of 0.3 r. per week (15 r. per year) for as long as 15 years would on the average contain at least 1 induced mutation. It is probable that the same is also true of the children of many radiologists, dermatologists, and dentists.[45]

The recent study of Macht and Lawrence,[46] gives direct evidence of genetic damage in such cases and is in this respect superior to the studies made in Japan. Moreover, studies of Moeller et al.[47] show that the population in general is already receiving significant amounts of radiation from medical diagnoses. Sonnenblick [48] finds that exposures of this kind are seldom adequately controlled.

When it is considered that practically every mutation must eventually become eliminated from the population, after having—even if imperceptibly—hampered enough descendants so as finally to be a deciding cause, in the last of the line, of his premature death or failure to reproduce, then it becomes evident that practically every mutation represents a postponed disaster. Thus the genetic damage, that to later generations, caused by a given total dose is seen to be far greater than the damage to the exposed individual himself. In view of this, measures and regulations concerned with radiation protection should be based, at least in the case of persons who may later reproduce, primarily on the risk of genetic damage or, more specifically, of point mutations in their germ cells, rather than on the risk of damage to their own bodies. This would cause such measures and regulations to be far more stringent than they are at present.[49]

THE INDUCED MUTATIONS IN RELATION TO THE NATURAL LOAD

On our conservative estimate of 16 million natural mutations arising per generation in a population of 100 million, a frequency of 0.16, it would take only about 37 roentgens of gamma radiation delivered to the population to produce a quantity of new mutations equal to the new natural ones, and thus to double the mutation frequency. Our conservative estimate, however, was based on the assumption of only 1×10^{-5} as the average freqeuncy of a mutation of some specific type, involving a given chromosomal position. According to this assumption the actual data, which indicate about 2×10^{-5} as the frequency of mutations of a specific type, are misleadingly high, because of certain sources of technical error. Since, however, this is a matter by no means proved as yet it remains quite possible that the amount of radiation necessary to double the mutation frequency is 75 roentgens or higher. This is approximately the value that we used in our earlier treatments of the subject,[49, 50] in which it was assumed that the observed 2×10^{-5} frequency for mutations of a specific type was approximately correct. These considerations illustrate the considerable margins of error in any present quantitative treatments, and the need for greater exactitude of knowledge.[51]

The present uncertainty regarding the natural mutation frequency carries with it a corresponding uncertainty regarding what proportion of the natural mutations in man are contributed by natural radiation. There is also uncertainty regarding this question based on variation in the amount of natural radiation. If we suppose that in some typical regions as much as 6 roentgens are accumulated, on the average, in the span of one human reproductive generation (25 to 30 years), then, on the more conservative estimate that the natural mutation frequency is equal to what would be induced by 37 roentgens, it turns out that some 16 percent of the natural mutations in man are produced by natural radiation. On the higher estimate for natural mutations, some 8 percent of them would be radiation-induced. In either case, the figure must be far higher than for short-lived organisms, such as mice or flies. On the other hand, in some

[45] H. J. Muller, Bulletin of the Atomic Scientists 11 : 210–12, 230 (1955) and Science 121 : 837–40 (1955).

[46] S. H. Macht and P. S. Lawrence, Amer. J. Roent. and Rad. Ther. 73 : 442–66 (1955).

[47] D. W. Moeller, J. G. Terrill, Jr., and S. C. Ingraham, II, Public Health Rep., U. S. Publ. Health Serv. 68 : 57–65 (1953).

[48] B. P. Sonnenblick, J. Newark Beth Israel Hosp. 6 : 81–42 (1955).

[49] H. J. Muller, Acta Radiol. 41 : 5–19 (1954).

[50] H. J. Muller, Amer. J. Obstet, and Gynec. 67 : 467–83 (1954).

[51] Much of the information concerning mutation frequencies in Drosophila cited in the present report was derived from work by the present writer and his associates that had been supported by grants from the Atomic Energy Commission (contract AT (11–1)–195), the American Cancer Society (given on recommendation of the Committee on Growth of the National Research Council, United States of America), and the Rockefeller Foundation.

human populations living at a high altitude, with its greater cosmic ray intensity, the contribution of radiation to the natural mutation rate must be twice as high as here estimated. Still higher values must obtain for some populations living in regions where radioactive minerals are abundant.

Many persons unfamiliar with genetics have regarded the seeming normality of the children born to survivors of the Hiroshima and Nagasaki bombings as evidence against the conclusion that the amount of radiation there received produced a significant amount of genetic damage. This misunderstanding arises from their lack of realization of the following points.

1. Few mutations are sufficiently dominant to give readily perceptible effects when inherited from only one parent, as they are in the vast majority of cases.

2. Even though these effects are not perceptible they are nearly always sufficient to hamper the individual somewhat, and finally, usually in a very distant descendant, to cause the extinction of that line of descent.

3. In any heterogenously breeding population, such as is found anywhere outside of the geneticists' fields and laboratories, there is already so much natural genetic variation, representing an accumulation of many generations of natural mutations, that the additional mutations caused by the radiation would become lost to view among them even if they were as abundant as those that would arise naturally in the course of a number of generations. Thus, the genetically damaged population will eventually have to pay the costs, but these will be spread out over so many small installments, and so intermingled with the greater weight of other payments, as hardly to be recognizable. All this was of course well known to geneticists before the observations on the children at Hiroshima and Nagasaki were conducted, and led them to express serious doubts that any genetic effects would be demonstrable there, even though they had no doubts that they had actually been produced.[41]

These points may be better appreciated if it is realized that in *Drosophila* also it had not been possible to demonstrate the mutagenic action of radiation by mere inspection of the individuals of the first, second, or third generations after exposure. Exact genetic methods had first to be worked out [11] and these are of course unavailable in man. Even following an exposure of fly spermatozoa to some 5,000 roentgens, which we today know causes each offspring, on the average, to receive at least three induced mutations, hardly one abnormal offspring is usually to be found among 100 examined, yet the damage is there, and it will be exerted if the population is allowed to continue.

At the same time, it is true, unlike what many nongeneticists suppose, that the effects of the genetic damage are more strongly exerted in the first generation of offspring than in any subsequent generation. They very gradually subside, in the course of many generations, as the population is purged by the dying out of the unfit. Even the recessive effects, those present in individuals homozygous for the given mutations, are found most frequently in the first generation, and then less and less frequently if the population breeds naturally rather than being subjected to a geneticist's controlled inbreeding manipulations. Moreover, there is a much higher chance that a given induced mutation will become homozygous by meeting, at fertilization, a gene of the same type derived from the great accumulated store of natural mutations, than one of that type which, like itself, had been induced by the radiation.

Since the worst effects are already exerted in F_1, what the Hiroshima observations do demonstrate clearly is that the genetic damage to posterity caused by exposure to between one hundred and several hundred roentgens is not *conspicuously* detrimental, and is well within limits consistent with the survival and self-perpetuation of the population. This might have been reckoned as probable without the direct evidence. For, according to the conclusion that the average individual is already heterozygous for some 6, 8, or even more mutations which when homozygous would be fairly conspicuous and/or detrimental, it does not seem likely that the addition in heterozygous condition of just one more, induced by some 200 roentgens, would result in any very evident change in the picture. This remains true even when we take into consideration the fact that the already existing mutations have already passed, to varying extents, through the sieve of selection and are therefore not, on the average, as detrimental as the newly induced ones.

The apparent contradiction between the fact that a really serious amount of genetic damage was produced and the fact that none is evident even in the most

---

[41] See footnote, p. 1079.
[11] See footnote, p. 1072.

afflicted generation (the first), is reconciled by the manner in which the damage is spread out, thinner and thinner, over a great number of generations. There is a kind of buffering or dilution of the damaging effects, by the normal genes that dominate over them and thus delay their elimination. Thus the effects are spread out in time, in inverse proportion to their dilution in any one generation, but the total damage remains just as great as if concentrated. Moreover, even though the induced mutations may be many times the number that would arise naturally in one one generation, they are nevertheless few in relation to the accumulated natural "load." Hence they can raise by only a rather small percent the number of genetic shortcomings already present in the population.

Despite these buffering influences, it would be impossible for a population to tolerate, generation after generation, an exposure which, given to only one generation, would cause no perceptible deterioration. Gradually, as elimination rose enough to balance the new mutations, an equilibrium level of accumulation would be approached, and at this new level the then existing accumulated load would be as many times greater than the original accumulated load as the then existing mutation rate was greater than the original mutation rate. Thus, if 37 roentgens doubles the mutation rate, a population which had received this dose for many generations would at last have twice as many ills of genetic origin as we have. Yet we already have more than enough for comfort.

Not to be neglected in the picture is the other end of the balance mechanism; the rate at which elimination of mutations goes on. Under modern civilization we interfere so much with this that we are probably raising the load of accumulated mutations as fast as by applying some tens of roentgens to everyone's reproductive organs.[45] Under these circumstances the raising of mutation frequency at the same time, by exposure to radiation, might tend to bring us to a genetic situation that it would be difficult to cope with.

All these questions need to be not only discussed but actually investigated far more realistically than they have been in the past. Otherwise we may at last find ourselves, genetically, facing a parallel to already accomplished deforestation and erosion, on an even grander scale. This problem is not only one that is concerned with the possible aftermaths of atomic war. It must be faced equally by the proponents of peace if we are to have an atomic age, with its risks of prolonged "permissible" exposures arising from industrial uses and radioactive waste products.

For peace will, we hope, go on and on through a great series of generations. Under these circumstances, it will be the more necessary to control and limit the radiation received by the population at large in every generation. For, given enough generations, the equilibrium level of damage will be reached, at which that damage will no longer be buffered, but will accurately correspond with the existing mutation frequency. Then, a relatively small number of roentgens per generation will exert an inordinately larger effect than it seems to now. At our present juncture, before that process has more than begun, far-seeing policies should be established. These must guard us against the dangerous fallacy that what cannot be seen or felt need not be bothered with.

This subject of protection of human beings against the genetic damage produced by radiation must, until suitable policies are established, far overshadow in its importance that of the utilization of radiation in the genetic improvement, for human purposes, of organisms potentially useful to man, or in the elimination or reduction of noxious organisms. However, these constructive uses of radiation in biological engineering will come increasingly to the fore as the more menacing aspects of radiation are brought under control. There is already abundant evidence of the possibility of such beneficial applications on a considerable scale.[41, 52]

At the same time, the dangerous mistake should not be made of considering man as a species who would himself undergo a long-term benefit from the application of radiation to his germ plasm. His own reproductive material is his most invaluable, irretrievable possession. It is already subject to an amount of variation which, in relation to his present reproductive practices, borders on the excessive. Under these circumstances, man's first concern in dealing with radiation must be his own protection.

---

[41] See footnote, p. 1079.
[52] A. Gustafsson, Cold Spring Harbor Symposia Quant. Biol. 16: 263–281 (1952). A. Hollaender, Ann. Missouri Botan. Garden 32: 165–178 (1945).

Representative HOLIFIELD. Are there any questions by members?

Representative VAN ZANDT. Dr. Muller, in regard to Russia, can you estimate the number of years it will take Russia to catch up with our progress in this field of radiation?

Dr. MULLER. I believe it will probably take them a long time. One would have to assess political factors there which are essentially unpredictable. Personalities play such a large role there, much larger than here, despite their theory of economic determination. Anyway, my estimate would be 10 or 15 years.

Representative HOLIFIELD. Thank you very much, Dr. Muller. Mr. Hollister wishes to ask you some questions.

Mr. HOLLISTER. Dr. Muller, I understand that you are familiar with the work of the Atomic Bomb Casualty Commission, and I wonder if you would comment on it and on the quality of and the conclusions from the data.

Dr. MULLER. I think it was evident to geneticists from the beginning that it was very doubtful whether any positive or essentially negative evidence could be obtained from a study of that kind, because human populations are so variable and it is so next to impossible to obtain two groups to compare, one irradiated and the other not, which are essentially similar in other respects. In view of that, I think that the present lack of results is not surprising even to most of those who took part in the investigation. I do not think they are to be blamed for it. There was some slight chance—because of the uncertainty of the estimates of the human mutation rate—that if the induced mutation rate was exceptionally high then some evidence of it could be obtained.

I think that it is very unfortunate to cite the lack of results from the study as indicating that there is no effect. I think that those reporting on the matter for the AEC in a December 1956 publication that I recently saw were quite right in saying that the data obtained there are not at all out of line with the expectation based on results with mice, that is, the frequency of induced mutations could well have been just as high among the human beings for the dose received as it would have been for mice, which is a matter we know a lot more about through Dr. Russell's experiments. We can go a little further than that and say that it could not have been a great many times higher than it is in mice, otherwise there would have been demonstrable results.

But we need not feel at all secure or relieved that no effects were found.

I remember that in a meeting of a committee called to decide whether these studies should continue one of the persons on the committee said, after I had remarked that I thought no effects would be found and that that would not prove anything, "That will be a very good thing because it will tend to allay the public fear on the matter."

Chairman DURHAM. Did you participate in writing the report?

Dr. MULLER. No; I did not. I may say this: I do disagree with a lot of the discussion in the last chapter where exception is taken to drawing conclusions from the results obtained in lower organisms. Certain criticisms are leveled against the work on flies and mice, for example, that I think are demonstrably unjustified and have a mistaken position. But that is not the major part of the work. When it

comes to the work on humans, I think that was done as well as could be expected under the very difficult circumstances.

Representative HOLIFIELD. Are there any further questions?

Representative VAN ZANDT. Dr. Muller, in the December 3, 1956, issue of the Federation of American Scientists you are quoted as saying this:

It is reckless to increase the risk of war by continuing H-bomb tests. It is not the fallout from these tests that is at issue at this time but the war feeling. The first step for peace open to us is a discontinuance of tests by both sides. If breached by either side it can be detected by the other.

Will you comment on that statement?

Dr. MULLER. I was not speaking there primarily as a geneticist, but I think physicists are pretty generally agreed that the setting off of a test in the megaton range can be detected with certainty by the other side. So that a breach would be known and in that sense we already have 100-percent-effective inspection for tests of that kind.

Therefore, it is in that sense safe if both sides agree to discontinue the tests for them to act upon that agreement, since as soon as one side breaks it, the other side will know it.

Senator ANDERSON. As a matter of fact, Doctor, the first times that we detected tests we detected them with instruments that are primitive compared to what we now have; is that not true?

Dr. MULLER. And we detected ordinary atomic bomb tests of the other side.

Senator ANDERSON. So that a megaton test would be easily detectable, and there is no possibility of deception.

Dr. MULLER. Yes.

Representative HOLIFIELD. Thank you very much.

The next witness is Dr. W. L. Russell.

Dr. Russell comes from Oak Ridge, and has done important work at the Laboratory. We are glad to have you here and to have your statement.

STATEMENT OF DR. W. L. RUSSELL, OAK RIDGE NATIONAL LABORATORY [5]

Dr. RUSSELL. Mr. Chairman, members, the testimony to be given here is presented in response to one of the requests by this committee for scientific results on the biological effects of radiation caused by events other than fallout. Assuming the present estimates of radiation from fallout to be approximately correct, it would in fact be virtually impossible at the present time to measure the genetic effects of fallout in mammals. In estimating the genetic hazards of fallout, we are, therefore, forced into using the information obtained from experiments, such as those to be described, in which much higher levels of radiation were used.

---

[5] B. A., Oxford University, 1932; Sherman Pratt fellow, Amherst College, 1932–33; fellow, University of Chicago, 1933–34; asistant, department of zoology, University of Chicago, 1934–36; Ph. D., University of Chicago, 1937; research associate, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine, 1937–47; principal geneticist, Oak Ridge National Laboratory, 1947 to present; research and publications on the genetic effects of radiation in mice. In charge of the Mammalian Genetics and Development Section of the Biology Division of Oak Ridge National Laboratory. Member of the United States delegation to the 1955 Geneva Conference on the Peaceful Uses of Atomic Energy. Member of the Committee on Genetics Effects of Atomic Radiation, National Academy of Sciences. (Submitted by witness.)

The results described here were obtained from a series of experiments conducted in the Mammalian Genetics and Development Section of the Biology Division of the Oak Ridge National Laboratory. This program was started in 1947, the year the Atomic Energy Commission was founded. The preliminary work involved the construction of animal rooms and laboratories, the development and building up of special stocks of mice necessary for the experiments, and certain pilot experiments, including studies on the effects of radiation on fertility. The major experiments were started in 1949.

I was very happy that Dr. Sturtevant said what he did this morning about the lack of suppression of information and the desire to do good work in the national laboratories. I heartily concur, but such a statement comes more forcefully from a person outside the AEC, and one of Dr. Sturtevant's standing. I will say no more on this, other than that neither I nor any of my colleagues would attempt to do scientific work under conditions that were not free.

I think I might also add, and it would be ungrateful of me if I did not, that in addition to working under free conditions and not having our information suppressed, we have received personal encouragement, especially from the Division of Biology and Medicine of the AEC, including its Directors, from Dr. Warren, the first one, to the present one, Dr. Dunham.

Before the results of these experiments with mice were obtained, estimates of genetic hazards of radiation in man were based primarily on data from experiments with the fruitfly, *Drosophila.* Some information on the genetic effects of radiation in mammals was available, but most of this dealt with major chromosomal aberrations which are probably not an important hazard in human exposures. There was virtually no information on radiation-induced gene mutation rates in any mammal. The information now available from our experiments has therefore played a basic role in the new estimates of genetic radiation hazards made and published last year by the National Academy of Sciences committee in the United States and by the Medical Research Council committee in Great Britain. The United Nations Scientific Committee has also used this information.

In response to the request by this committee, I should like to present here a simplified up-to-date summary of our results, emphasizing the aspects which are useful in estimating human hazards. More detailed technical accounts have been submitted for the record to supplement this oral presentation. Again it must be kept in mind that these data were not obtained from fallout radiation. However, they can be used to estimate the genetic hazard from fallout, provided the radiation dose to the gonads is known. I shall describe two types of experiment. One of these measures the rates at which genes mutate by studying the effect of radiation on a particular selected sample of genes. The other measures damage in a population as a result of the total amount of mutation induced.

Most of our work up until recent times has been concerned with the former method, and so I shall describe the results obtained by it in more detail. The measurement of gene mutation rates is important, both for absolute and for comparative purposes. The particular method that we have used was chosen in the hope that it would be a fine-edged tool for making comparisons. One of these that was ob-

viously badly needed was a comparison of the mutation rates in any mammal with those in Drosophila. The mutation rate method chosen by us seemed likely to be the one which would give the most meaningful information on such a species comparison. Our present information on this point indicates that mouse genes are on the average approximately 15 times as sensitive to radiation as Drosophila genes. The earlier estimates of genetic hazards in man based on results in Drosophila have, therefore, been revised.

Another question on which information was badly needed was whether or not there is any recovery from genetic damage with time after irradiation. Some geneticists believed that there would be no such recovery. Others thought that some recovery might occur. Critical evidence on this point had not been obtained. Most of the information along these lines came from experiments with Drosophila sperm, whereas the evidence needed was for immature sex cells. Let me elaborate for a moment on the importance of the cell stage, i. e., immature versus mature germ cells, before we continue with the question of recovery.

In the testis, the immature germ cells—called spermatogonia—persist throughout life. Cells are constantly budded off from them and, after further multiplication, develop into the mature sperm cells. The time required for the development of a mature sperm from an immature spermatogonium is only a few weeks. When we are exposed to continuous or intermittent radiation, the dose received by a cell up to and including the spermatogonium stage is the total dose received over the whole period from conception of the individual up to the time when the spermatogonium starts its final development. This is obviously going to be much greater than the dose received during the few weeks of its final development into a sperm cell. Even with acute radiation received as a single dose, the chance of a fertile mating occurring within a few weeks after exposure is small compared with fertile matings that will occur at later intervals. Thus again it is usually the dose received by the immature cells which will count. Therefore, the problem of genetic hazard of radiation in man relates primarily to the immature germ cells. It is on these cells that our studies of induced mutations in mice have been conducted. This has many important implications, one of which is on the question of recovery, to which we can now return.

How might the question of cell stage affect recovery following irradiation? It is possible to imagine that an immature germ cell in which a mutation has been induced might multiply at a slower rate than a normal cell. This is one example of a possible mechanism by which some recovery from genetic damage might occur. It is obvious that experimental data from mature germ cells in Drosophila could not answer such a problem. Considering the possibility that I have just mentioned, along with others, it was clear to us that the question of recovery with time had to be investigated for immature germ cells. It also seemed important that the problem be examined in an organism with a generation time much longer than that of the fruitfly, which is only 10 days, and in mammalian gonads, which are quite different in their makeup and function from those of insects. Having now obtained this information, we find that in spite of the possible mechanisms by which recovery might have occurred, there

is in fact no evidence of any significant recovery with time after irradiation. The offspring of a mating made a long time after irradiation is just as likely to contain a gene mutation as is the offspring of a mating made shortly after irradiation. I might interject here that I am talking only about irradiated spermatogonia. If we included the sperm, there would be some recovery within a short time after radiation. In human hazards, however, we are concerned, as I have already explained, primarily with the immature germ cells. Thus it would appear that it is the cumulative dose which is important, and this principle is now established for the immature germ cells of a mammal. I have dwelt on this point at some length because I have found it to be one that is often raised in discussion. We are so used to the body being able to recover from various types of damage that it is quite reasonable to require rigorous proof that this does not occur with genetic damage.

Another problem that was investigated by our method for measuring gene mutation rates is the relation between mutation rate and dose. On the basis of experiments with other organisms, a linear relation was expected. In other words, it was expected that mutation rate would be directly proportional to dose, that, for example, doubling the dose would double the mutation rate.

Dr. Crow this morning assumed this principle and stated that he was basing his assumption on Drosophila. Therefore, I think it appropriate to state what information we have in this respect in the mouse.

Our first data came from males exposed to 600 roentgens. As the data started to come in from males exposed to 1,000 roentgens it became clear that the mutation rate was significantly lower than expected on the basis of the 600 roentgen results. It seemed likely that this result, which was unexpected on the basis of Drosophila results obtained up to that time, might be due to the fact, already emphasized, that we were dealing with immature germ cells (spermatogonia) in the mice, whereas Drosophila results had been obtained from mature germ cells. If, for example, there were differences in sensitivity to mutation induction correlated with sensitivity to killing of the cells, then at the higher doses the mutation rate observed might represent only the mutation rate for the surviving and more resistant cells. Careful studies in our laboratory by Dr. Oakberg on the amount of killing of spermatogonia with various doses of radiation support this possibility.

Whatever the explanation might be, it was clear that it was important to obtain data on mutation rates at doses lower than 600 roentgens. Since the relation between mutation rate and dose has been found not to be directly proportional above 600 roentgens, it was possible that it would also not be proportional below 600 roentgens. We have therefore begun experiements at lower doses. The data so far obtained from a 300-roentgen experiment show no significant departures from proportionality with the 600-roentgen results. More data are, however, needed.

Representative Holifield. At that point, Dr. Russell, could you tell this committee how near you think the experiments on mice would correspond to the radiation of human beings? You have made the statement that the mouse is 15 times more sensitive than the fruitfly. Can you give us a comparison between the mice and the human being?

Dr. RUSSELL. I have prepared a short statement involving the whole problem of extrapolating.

Representative HOLIFIELD. Is that contained in the statement?

Dr. RUSSELL. It is not in the typed copy you have, but I shall be glad to read it.

Representative HOLIFIELD. Or could you give me a summary right now?

Dr. RUSSELL. I believe a summary is the best we can do at the present time. I might explain a little more about this dose relation, if I may, on the board.

If we put the 600-roentgen mutation rate at this point here, the 1,000-roentgen result instead of being up here, as expected from the linear relation found in Drosophila, turned out to be down here [pointing on blackboard].

Representative COLE. Doctor, would you go back to the microphone and repeat what you said, because I do not understand it.

Dr. RUSSELL. The first result we got was with the 600-roentgen experiment and is shown here. The points immediately above and below it that I have connected with the line represent the 95-percent confidence interval for this result, that it, with 95 percent probability the true value should lie within that range. The 1,000-roentgen result was expected, from the linearity found in Drosophila experiments, to be correspondingly higher than the 600-roentgen, but actually came out lower. In other words, the relation is not linear. This raises the question of why there is linearity in Drosophila and not in the mouse.

As I have said, we felt that perhaps this lack of linearity in the mouse was due to the killing of the spermatogonial cells themselves, and that at the higher dose the result represents the mutation rate of more resistant cells which were not killed. At the lower dose you get a mutation rate for more sensitive cells, some of which have not been killed.

The question arises what is the curve going to do below 600 roentgens? You can no longer predict that it will be linear between 600 roentgens and zero. There is now some likelihood that it will be higher than expected on a linear basis. Up to this time in the hearings we have had a good deal of discussion of whether the mutation rate is linear and if whether some somatic effects are linear or whether they go up this way, that is, concave upward. These mouse mutation results represent something going this way, that is, convex upward. In other words, the effect at lower doses might be higher than expected on a linear basis. It was obviously important to obtain data at the lower doses. The 300-roentgen result looks like this. At the present time it falls close to the straight line drawn between the results for the 600 roentgens and the zero doses, but as I say, I think we need more data on this point.

Representative HOLIFIELD. This would indicate, then, if this theory becomes established, that the lower dose rates would cause more mutations in the germ cells than the higher rate.

Dr. RUSSELL. This is a possibility raised by this departure from linearity at high doses.

Representative HOLIFIELD. That is because the higher dose kills the cell.

Dr. RUSSELL. That is right. The amount of killing of cells is quite high so it is still possible to get some departure from linearity at lower doses. So we feel that this point needs further experimentation.

Representative HOLIFIELD. But you are not yet ready to set the rate?

Dr. RUSSELL. Three hundred roentgens was the first attempt to provide some information between 600 roentgens and zero. As we go down in the dose, it is much harder to obtain the data because fewer mutations are obtained.

Chairman DURHAM. Are you using only flies?

Dr. RUSSELL. This is all on mice.

I might also interject at this point another comment. A good deal has been said about there being no Drosophila data on mutation rates below 25 roentgens and about there being no mouse data below 300 roentgens. Yet, again referring to Dr. Oakberg's work in our laboratory, he can see and actually measure the killing of spermatogonial cells with doses as low as 2 or 3 roentgens, and even with 1 rep of neutrons. A dose of 22 roentgens kills half of the sensitive spermatogonia. So it seems to me that if we can actually see cells that have been killed by doses as low as 2 or 3 roentgens, and can put this on a quantitative basis, if cells are actually killed at these dose levels, there is no question in my mind that there will be genetic effects from doses as low as this.

Representative HOLIFIELD. This has a tremendous impact on the theory of threshold, does it not?

Dr. RUSSELL. It certainly helps to answer the question of threshold for genetic effects. Of course, the question of threshold for some somatic effects is not answered by this, because killed cells might be replaced by normal cells. So it does not conclusively prove anything about the question of a threshold for somatic effects. I think it supports the already quite well established point of there being no threshold for genetic effects, because here is direct experimental evidence on mammals that cells can be killed measurably by doses as low as 2 or 3 roentgens.

Representative HOLIFIELD. You have been able to observe those?

Dr. RUSSELL. The killed ones can be observed, yes.

Another comparative study that can be made with the gene mutation rate method used by us is on the variation in mutation rates of different genes. Extensive information on seven different genes in the mouse shows a wide range in their mutation rates. The difference between the lowest and the highest rates is more than thirtyfold. This finding is of interest in many respects. For example, it raises the possibility that the so-called rate-doubling dose, the dose required to double the spontaneous rate, might be quite different for different genes.

The results from our gene mutation rate method that I have so far described have been useful primarily for comparative purposes. Thus we have been able to compare mutation rates in mice and fruitflies; we have been able to compare mutation rates at short and long intervals after irradiation; we have compared mutation rates at different doses; and so on. Additional work now going on with this method will, in addition to measuring mutation rates at lower doses, also give information on other comparisons—for example, a comparison between the mutation rates in females and males, a comparison of the

results from long-continued low-level irradiation with those from single dose acute radiation, and so on.

Although designed primarily to provide information for the comparisons that I have already described, the gene mutation study has given some information on the nature and amount of total damage to be expected. First with regard to the nature of the damage, considerably more than one-half of all the radiation-induced mutations obtained from the seven genes studied in detail have proved to be recessive lethals—that is, when an offspring inherits the mutation from both parents, it will die. It seems unlikely that this result is biased in the unfavorable direction, because before the experiments were started it was not known whether or not any of the seven genes chosen for the study could mutate to lethals.

Representative HOLIFIELD. Will you explain that term "recessive lethal"?

Dr. RUSSELL. That is when an offspring inherits the mutation from both parents, it will die. When the organism inherits it from one parent, it won't die. It seems unlikely that this result is biased in the unfavorable direction, because before the experiments were started it was not known whether or not any of the seven genes chosen for the study could mutate to lethals. In other words, the sample of genes chosen was not chosen for production of this type of mutation at all. It was not known whether they could even mutate to lethals.

Some information has been obtained on the time at which these lethals kill the individuals who inherit them from both parents. All the lethals that have occurred at one gene locus kill the offspring at about weaning age. The lethals obtained from another gene locus apparently vary considerably in their time of killing. More information is needed on this point, but it is already clear that a large proportion of the lethals already studied kill at times that would be considered tragedies in human experience. Most of the lethals found do not belong to the category of lethals in which death occurs so early in development of the embryo as to pass unnoticed.

It was anticipated that some, perhaps most, of these lethals would have some deleterious effect when inherited from only one parent. Such deleterious effects have already been observed for some of the lethals. It was expected that these deleterious effects might be observable only in a statistical sense in populations. However, in some cases the deleterious effect of a lethal when inherited from only one parent is large enough to be detectable in the individual. It should be mentioned that a lethal which has a deleterious effect when inherited from only one parent will express its damage in the population far more frequently in this way than in the more drastic effect that occurs when it is inherited from both parents.

As has been mentioned, the results of the gene mutation study can be used to estimate total mutation rate. One way of doing this is to take the information from Drosophila on the ratio of total mutation rate to mutation rate at specific genes and use this to make the calculation of total rate on the basis of mutation rates at specific genes in the mouse. This method of estimation was used in the report of the Genetics Committee of the National Academy of Sciences. Dr. Muller used it before, and I think Dr. Crow incorporated this idea in his calculation this morning.

More direct methods of measuring the overall genetic damage are desirable and one of these is the second of the two methods I should like to describe in this testimony. This method was suggested by the evidence that had begun to accumulate that there are slight dominant deleterious effects of mutations formerly regarded as recessive. Two lines of such evidence came from our work on mice. One has already been mentioned, namely, that at least some of the recessive lethals obtained have some dominant effects. The second line of evidence came from the large populations of animals examined for gene mutations. In that study, the offspring were kept to 3 weeks of age, and it was found that survival to this age was slightly lower in the offspring of irradiated males than in the controls. As a result of these findings, and of evidence from other organisms, it began to seem probable that other effects might be detectable in the first generation offspring of irradiated mice. Shortening of life was chosen as an effect that might reveal, as a statistic of a population, the presence of a variety of minor weaknesses which individually could not be easily detected. Pilot studies on longevity in the descendants of irradiated mice were accordingly started. The results from the first of these were presented before the National Academy of Sciences a few weeks ago, and have just been published.

Dr. Crow did not discuss these this morning. I asked him whether be did not believe them. He said "No," he had left them for me to discuss, and I think he felt it was more appropriate as they are quite recent data.

Representative Cole. When you say that he responded to the question of whether he believed them or not, by saying "No," did he mean he did not believe them, or that was not the reason?

Dr. Russell. That was not the reason.

Senator Anderson. Are you going to discuss them?

Dr. Russell. I have submitted for the record the detailed account of this which has been published. I shall be glad to read from this or cite any parts.

Senator Anderson. You were here the other day when I read briefly from the news story.

Dr. Russell. Yes.

Senator Anderson. Is that what it is based on?

Dr. Russell. Yes.

Senator Anderson (reading):

> Neutron radiation from atomic bomb can shorten the life of a man's children.

This is quoting from you.

> Offspring from a man exposed to such radiation will have their lives shortened on the average of 20 days for each unit of radiation their father has received.

Dr. Russell. Yes.

Senator Anderson. We have been talking about receiving up to 400 roentgens. We multiplied that out and came to 8,000 days, and that was 22 years.

Dr. Russell. There are two major qualifications which I believe were included in some of the news reports. I remember that they were in the full Science Service report. These qualifications are first, that we were dealing with neutrons which are probably more effective than X-ray or gamma rays. The other is that we were not dealing with the spermatogonia. In this experiment we deliberately tried to

maximize everything to get the effect on scale so that if there were any such effect, we would detect it. In this we used the maturing germ cells which for mutation rates are at least, shall we say, 2 to 4 times as sensitive as the spermatogonia for straight gene mutation rates. It is possible they are even more sensitive for this effect, but this we don't know yet. A more conservative conclusion from the results is the one I put in the discussion. I think at the present time we would be justified in estimating the effect on the first generation of offspring to be between one-tenth and equal to the effect on the shortening of life of the exposed individuals.

I think it is perhaps fairer to talk about this than the 20 days which, in several respects, is maximized.

Senator ANDERSON. I know. Apparently this article in the Science Service was quoting you. It said, "Dr. Russell found, however, that 'There was a significant effect of radiation on the length of life of the offspring.' In the male mice"—I am sorry, the quotation marks were dropped there. It says the life of the offspring was shortened 0.61 days, I assume, for each unit of radiation.

Dr. RUSSELL. Yes.

Senator ANDERSON. Does that refer to mice?

Dr. RUSSELL. Yes; that refers to mice. A unit of radiation in this case was the rep of neutrons rather than the r. for X-rays or gamma rays. There should be some reduction factor. This is not known. But perhaps it is a factor of about 2.

Senator ANDERSON. Quoting further from this article—

> Using this information for human beings, Dr. Russell has figured out that a man's life will be shortened from 5 to 35 days for each unit of radiation received by the father.

Is that a correct statement?

Dr. RUSSELL. That is correct. This again refers to the conditions of this experiment, which are not the conditions of human exposure.

Representative PRICE. What was the unit of radiation?

Dr. RUSSELL. The unit of radiation was a rep.

Representative COLE. A rep is what fraction of a roentgen?

Dr. RUSSELL. A rep is equivalent to the roentgen in its physical effect on tissue, but is more effective in its biological effect for most types of biological effect.

Representative HOLIFIELD. Are you drawing the line between the neutrons which would be received close to a bomb explosion, and the gamma and other types of rays that would be received far away?

Dr. RUSSELL. That is partly correct. There would be a mixture of neutrons and gamma rays. Our experiment was set up only to measure the neutron effects. The animals were shielded behind lead to filter out the gamma radiation. The experiment, as I emphasized in the paper, was not conducted primarily for this particular study. We got the data which showed this rather striking effect, so reported it. We are now conducting experiments designed deliberately to measure the shortening of life. But we felt we should not wait for the completion of these before reporting the effect already observed. However, the qualifications are important, and I must insist that they be understood. Otherwise if you just take the 20-day figure and do not put the qualifications on it, it is probably a much larger effect than would be obtained under normal conditions of human exposure.

Senator ANDERSON. It says—

This shortening of life in the immediate offspring, Dr. Russell warns, in the Proceedings of the National Academy of Sciences, will turn out to be of a magnitude that will warrant serious consideration as a genetic hazard in man.

Is that a correct quotation?

Dr. RUSSELL. That is a correct quotation. I have the same statement in this testimony I was about to read. I do believe that, in spite of the qualifications, this is still a serious effect. I think the 20-day figure is too high. For conditions of human exposure, at least by our present estimates, it should be somewhat lower, but I still think it would be serious.

Senator ANDERSON. From 5 to 35 is a safe figure.

Dr. RUSSELL. No; that would still be under these conditions. If you want a definite figure, I would say at the present time something more like 1 to 10. These are guesses. It is much safer to publish the data you have and put the qualifications on them, than to make guesses about the quantitative values of the qualifications.

Senator ANDERSON. It was pointed out that it could take up to 10 and with 400 r, that would produce 4,000 days which would be 11 years, which would be quite a shortening of human life.

Dr. RUSSELL. Yes.

Senator ANDERSON. Which would be serious.

Dr. RUSSELL. Yes, 400 r. would be serious. To conclude the statement on this, some of which we have already discussed: Our data on this effect are not yet as extensive as we should like. However, though the first sample studied was small, it was sufficient to yield a statistically significant effect which appears to be large and, therefore, of general importance. It should be kept in mind that some of the conditions of this experiment were set up deliberately to increase the chance of obtaining a detectable effect. However, since the effect observed appears to be so large, it seems likely that, even when allowance is made for the conditions of human radiation exposure, shortening of life in the immediate descendants will turn out to be of a magnitude that will warrant serious consideration as a genetic hazard in man.

In conclusion, I should like to emphasize one point in addition to those already expressed in this presentation, a point that Dr. Crow mentioned this morning. The layman tends to think of mutations in dramatic terms as gross monstrosities occurring in the first generation following radiation. Our studies on mice confirm the results obtained from other organisms showing that these are exceedingly rare types of genetic damage. Mutations which cause slight deleterious effects are far commoner.

Before summarizing these points, I should like to say a word or two about extrapolation from mouse to man, the question which the chairman raised earlier. There are, of course, risks in this. I think some of the earlier testimony, from medical workers and others, overemphasized the difficulty. Yet, applying animal results to man is exactly what is done all the time in medicine in the testing of drugs and so on. Others who objected to the extrapolation of mouse mutation data to man had no qualms about extrapolating the rate-doubling dose. I personally would feel safer about extrapolating the induced mutation

rate in mammals from mouse to man than about extrapolating the rate-doubling dose itself.

I will certainly agree that there are some risks in extrapolating on the quantitative points, that is, the actual mutation rates. I think the risks are very much less in extrapolating with regard to the principles or relations between various points. Such principles as the lack of recovery with time, the shape of the dose curve, the relative frequencies of different types of mutations and so on, it seems to me, can be extrapolated with fair confidence.

Some of these are principles that cannot be extrapolated from fruitflies, because of the biological difference between flies and mammals. Other principles, of course, which were very well established in flies can be extrapolated to mammals. I have been stressing in this report those which I think needed work directly on mammals.

I have listed nine points of this testimony in summary.

1. Present data indicate that mouse genes are approximately 15 times as sensitive to the induction of mutation by radiation as fruitfly (*Drosophila*) genes.

2. There is no recovery from genetic damage with time after irradiation. This principle has now been established by direct experimental evidence on the material in which investigation was badly needed, namely, the immature germ cells of a mammal.

3. Mutation rates following a dose of 1,000 r. show a significant departure from proportionality with the rates obtained at lower doses. Mutation rates at 300 r. and 600 r. do not as yet show any significant difference from proportionality. However, more data are needed, especially in view of the observed departure from proportionality at the higher dose.

4. Different genes show widely different radiation induced mutation rates.

5. More than one half of the radiation-induced mutations obtained have proved to be recessive lethals—that is, when an individual inherits the mutation from both parents it will die.

6. Most of these lethals do not belong to the category in which death occurs so early in development of the embryo as to pass unnoticed. On the contrary, they kill at times that would be considered tragedies in human experience.

7. The deleterious effect of some lethals when inherited from only one parent is large enough to be detectable in the individual.

8. Rough estimates of the total mutation rate expected from a given dose of radiation have been made from the sample of mouse genes studied.

9. More direct methods of measuring overall genetic damage are being used. The first results from one of these show a significant shortening of life in the first generation offspring of irradiated male mice.

In presenting this statement today, I hope I have given the committee a useful picture of at least a part of the basis on which conclusions on the genetic hazards in man have been reached. Thank you.

Representative HOLIFIELD. Thank you very much, Dr. Russell. I think you have given a very important statement here. It certainly seems to prove that in the case of genetics, at least, any radiation is harmful to the genes.

Are there any questions?

Representative COLE. Mr. Chairman, I do not recall that the doctor responded to your inquiry with respect to the relative sensitivity of human cells in comparison with mice.

Dr. RUSSELL. I think there is some question about extrapolating the exact mutation rate from mice to man. I think there is less risk in extrapolating the principles that we have established in our experiments. However, I think the best we can do at the present time is to use the organism closest to man on which we have data, and that is the mouse. I personally would feel less worried about extrapolating the induced mutation rate from mouse to man than I would about some other extrapolations that have been made.

Representative COLE. Then you are unable to give us any estimate of the ratio between fruitfly, mice, and human being?

Dr. RUSSELL. We have the ratio between mouse and fruitfly. This is 15 to 1. With regard to man and mouse, we have no information other than such as has been discussed. It appears from the Hiroshima and Nagasaki data that man is probably not greatly more sensitive than the mouse. Otherwise more damage would have been observed. However, the results on the Japanese study are fully consistent with a rate as high as has been found in the mouse. I would, like Dr. Muller, quarrel with some of the final conclusions of the report on the study in Japan. I would quarrel with one statement there about this particular point. Even so, in the report on this study, the authors do not raise any question about man being less sensitive than the mouse to radiation-induced mutation. I think the data are not extensive enough to rule out the possibility that he could be more sensitive than the mouse.

Chairman DURHAM. Your second summary point you base on how many years of research, that is, there is no recovery from genetic damage?

Dr. Russell. The first indication came after, I would say, about 2 years' work after the basic experiments were started. This has been further confirmed by more extensive data as time has gone on.

Representative HOLIFIELD. This refers to your experiments on mice.

Dr. RUSSELL. Yes.

Representative HOLIFIELD. Thank you very much, Dr. Russell. We will place your article, Shortening of Life in the Offspring of Male Mice Exposed to Neutron Radiation From an Atomic Bomb, in the record at this point.

(The material referred to follows:)

[Reprinted from the Proceedings of the National Academy of Sciences, vol. 43, No. 4, pp. 324–329, April 1957]

SHORTENING OF LIFE IN THE OFFSPRING OF MALE MICE EXPOSED TO NEUTRON RADIATION FROM AN ATOMIC BOMB[1]

By W. L. Russell, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn.

Communicated by Sewall Wright, January 31, 1957

Introduction.—Only in recent years has evidence begun to accumulate that there are slight dominant deleterious effects of mutations formerly regarded as

[1] Work performed under Contract No. W-7405-Eng-26 for the United States Atomic Energy Commission.

recessive.[3-5] The results to be reported here, and our earlier work on mice,[4, 6, 7] indicate that such effects may add up to an important part, perhaps the most important part, of the genetic hazards of radiation in man. The evidence from the earlier work on mice is that appreciable deleterious effects of radiation become manifest in the first-generation offspring. This evidence is of two kinds. First, work on radiation-induced mutations at specific loci in spermatogonia has shown that among the recessive lethals, which comprise more than one-half of all the mutations recovered, many have dominant deleterious effects which, even for individual mutations, are sometimes large enough to be detected easily. Second, overall population damage was found in the large numbers of animals that are raised as far as 3 weeks of age in the specific loci studies. In all such experiments carried out, the survival to 3 weeks of age is significantly lower in the offspring of irradiated males than it is in the controls. (It should perhaps be pointed out that neither of the above effects, nor the effect reported in this paper, is the result of what the geneticist usually refers to as "dominant lethals," which are major chomosomal aberrations that cause early death of embryos and which, as has been pointed out elsewhere,[6] are probably not an important hazard.)

Our earlier work that showed a significant effect on survival to 3 weeks of age in the offspring of irradiated males led us to expect that there would be measurable deleterious effects later in life. The data reported here show that such is indeed the case. These data furnish a third kind of evidence of first-generation damage and perhaps the most striking one. They were obtained as a byproduct of another investigation, and they are not as extensive as we should like. However, they are the only data we have on this subject that were collected under the expensive and difficult conditions of a field test of a nuclear detonation. Furthermore, although the sample was small, it was sufficient to yield a statistically significant effect which appears to be large and, therefore, of general importance.

Materials and methods.—The material used in the present longevity study was the byproduct of an investigation of the relative effectiveness of neutrons from a nuclear detonation and from a cyclotron in inducing dominant lethals in the mouse.[8] In order to reduce the gamma component of the radiation to a proportion that would not appreciably interfere with the estimation of neutron effects, the animals were shielded with lead. The exposure chambers available were lead hemispheres of 7-inch wall thickness and 14-inch inside diameter. Young adult hybrid males, obtained by crossing inbred 101 strain females with inbred C3H strain males, were exposed inside the hemispheres placed at various distances from the detonation. Control males were placed in hemispheres 2 days before the detonation and for a length of time approximately the same as that required for the exposed animals. Further experimental details are described in the report of the earlier work.[8] One day and a half after the detonation, each male was placed with four adult untreated females of the same hybrid strain. At 18½ days after irradiation each surviving male was placed with a new group of 4 females. Most of the females that became pregnant were killed at a late stage of gestation for the dominant-lethal study. However, since the number of pregnancies turned out to be more than adequate for the dominant-lethal experiment, several of the females were allowed to come to term. It was the offspring of some of these females that were saved for the longevity study described here. All these animals came from matings made from 19 to 23 days after irradiation. A few animals died before weaning, and these were not included in the data reported here. At weaning age the sexes were separated and the animals grouped, so far as possible, six to a cage. They were kept in the same grouping throughout their life span. They were checked at least twice weekly for deaths. Only one animal died at less than 1 year of age, indicating that the conditions under which the animals were kept were good.

The total (neutron plus gamma radiation) dose inside each lead hemisphere was measured, as described in the earlier publication,[8] by means of tissue-

[2] C. Stern and E. Novitski, Science, 108, 538–539, 1948.
[3] H. J. Muller, J. Cellular Comp. Physiol., 35, suppl. 1, 205–210, 1950.
[4] W. L. Russell, Cold Spring Harbor Symposia Quant. Biol., 16, 327–336, 1951.
[5] C. Stern, G. Carson, M. Kinst, E. Novitski, and D. Uphoff, Genetics, 37, 413–449, 1952.
[6] W. L. Russell, in Radiation Biology, Vol. I, ed. A. Hollaender (New York: McGraw-Hill Book Co., 1954), chap. xii.
[7] W. L. Russell, Proceedings of the International Conference on the Peaceful Uses of Atomic Energy, 11 (New York: United Nations, 1956), 382–383, 401–402.
[8] W. L. Russell, L. B. Russell, and A. W. Kimball, Am. Naturalist, 88, 269–286, 1954.

equivalent ion chambers designed for this purpose at short notice.[9] Subsequently, extensive testing and recalibration of these chambers[10] has led to a revision of the original dose estimates. The doses reported in the present publication are the revised estimates. As was reported earlier,[8] attempts to measure the gamma component of the radiation by means of film dosimeters left a large uncertainty as to the size of this factor. Later tests have been made in which both ionization chambers and chemical dosimeters were used to measure the gamma component inside the lead hemispheres when these were exposed to fission neutrons. According to the latest information,[11] these tests indicate that the gamma-radiation exposure in our experiment was almost certainly less than 10 percent of the total dose.

Results.—The median and mean lengths of life, together with the number of animals, for each dose group are given in table 1. An analysis of variance showed that neither grouping in cages nor sex had a significant effect on length of life. It seems likely that larger samples would show some effect of both of these factors, but as there was no significant effect of them in the present experiment, the data were pooled.

TABLE 1.—*Length of life in the offspring of male mice exposed to neutron radiation 19–23 days before mating (deaths before weaning age excluded)*

Total dose to parent (rep) [1]	Number of offspring	Median length of life of offspring (days)	Mean length of life of offspring (days)
6	103	823	792
31	50	741	754
71	5	717	699
118	22	739	723
136	8	666	688
186	2	756	756

[1] Includes some gamma radiation, estimated to be less than 10 percent of the total dose.

To test whether there was a significant effect of radiation on the length of life of the offspring, the means were fitted to a straight line by the method of weighted least squares. This gives an intercept of 786 days and a slope of -0.609 ± 0.238. Since the residual variance is less than the within-subclass mean square, there is no evidence of nonlinearity over the dose range tested. Even if the true shape of the curve is nonlinear, it will be conservative, in making the test of significance, to assume linearity. The larger mean square was used to compute the variance of the slope, and a two-sided t-test shows that the slope differs significantly from zero at the 1-percent level. If one is willing to accept a one-sided t-test as more appropriate, the significance level is 0.5 percent. Thus there is strong evidence of shortening of life in the offspring of the exposed males.

Discussion.—It is noteworthy that a significant shortening of life was detected in spite of the small sample and the considerable genetic variability that must have been present in a population that was the F_2 of a cross between inbred strains. Furthermore, the weighted mean dose received by the exposed fathers was only moderate, being less than one-sixth of the 30-day median lethal dose as measured from other animals of the same strain exposed under the same conditions at distances closer to the same detonation. While it is true that certain features of the experiment, which will be discussed later, tended to maximize the shortening of life, nevertheless the result observed appears to be so large that it seems quite possible that shortening of life is an effect that might be detectable in studies of the offspring of exposed parents in human populations.

---

[8] See footnotes, p. 1097.
[9] C. W. Sheppard and E. B. Darden, appendix to J. S. Kirby-Smith and C. P. Swanson, Science, 119, 42–45, 1954.
[10] C. W. Sheppard, M. Slater, E. B. Darden, Jr., A. W. Kimball, G. J. Atta, C. W. Edington, and W. K. Baker, Radiation Research (in press).
[11] G. S. Hurst, personal communication.

TABLE 2.—*Shortening of life in the offspring of fathers exposed to neutron radiation 19 to 23 days before mating—Observed result in the mouse and extrapolation to man (deaths before weaning age excluded)*

	Mouse	Man
Point estimate	0.61 day/r. e. p. to father	20 days/r. e. p. to father.
Lower 95-percent confidence limit	0.14 day/r. e. p. to father	5 days/r. e. p. to father.
Upper 95-percent confidence limit	1.07 days/r. e. p. to father	35 days/r. e. p. to father.

In view of the lack of information on this subject, and specifically the fact that no data of this nature were ready for consideration prior to the writing of the 1956 report of the National Academy of Sciences Committee on Genetic Effects,[12] it is desirable to consider what the present data might indicate when they are extrapolated to man. Taking the estimate obtained from the curve fitted to the mouse data, and assuming that the shortening of life in man would be proportional to this, gives, on the basis of a 70-year length of life in man, the figures shown in table 2. It should be kept in mind that the results were obtained from neutron irradiation. The relative biological effectiveness of neutrons for this effect is not known, but it seems likely, from other data on mutations, that gamma and X-radiation would be less effective than neutrons. It should also be emphasized that the effect observed here is probably a maximum one, since the offspring were obtained from matings made between 19 and 23 days after irradiation. Our data from experiments on mutations at specific loci [13] indicate that the sperm utilized in matings made within this time interval would have been derived from cells in a sensitive stage of gametogenesis at the time of irradiation. From approximately 2 to 4 times as many mutations are recovered from this stage as from the spermatogonial stage, which is the important one so far as radiation hazards in man are concerned.[6] It is also possible that the spectrum of mutations from irradiated spermatogonia would be qualitatively different and, conceivably, less effective in shortening life. However, there is no direct evidence of this, whereas there is evidence from our specific loci studies that some mutations induced in spermatogonia have, even individually, a dominant effect on length of life that is detectable. To summarize this paragraph, it should be remembered that the estimates given in table 2 are based on neutron irradiation of a postspermatogonial and sensitive stage in gametogenesis and that X- or gamma irradiation of spermatogonia would almost certainly produce a smaller effect.

Another way of considering the magnitude of the observed results, so far as its human implications are concerned, is to compare the shortening of life in the offspring of irradiated fathers with that in the irradiated individuals themselves. The data on shortening of life of the males exposed to this same detonation will be presented in detail elsewhere. Briefly, the percentage shortening of life of these animals, based on 24 controls and 128 exposed animals, is 0.078 percent per r. e. p.

The present data, expressed in the same form, give 0.077 percent shortening of life in the offspring for each r. e. p. received by the father; that is, approximately as much effect as on the exposed individuals. Thus the best estimate from our present data is that, for neutron irradiation of the sensitive stages in spermatogenesis, the shortening of life in the offspring of irradiated males will be similar in magnitude to that in the exposed individuals. Again, the effect from irradiation of spermatogonial stages would probably be less.

Whether the *ratio* of effect in offspring to effect in exposed individuals will be different for X- and gamma rays from that observed for neutrons will, of course, depend on whether the relative biological effectiveness of neutrons is different for the effect on the offspring and the effect on the exposed individuals. Present, incomplete data on these points give no grounds for expecting that the ratio of effect in offspring to effect in exposed individuals will be less for X-rays than for neutrons. Weighing the evidence reported here, and making

[12] The Biological Effects of Atomic Radiation: Summary Reports (Washington: National Academy of Sciences, National Research Council, 1956).

[13] W. L. Russell, USAEC Unclassified Report ORNL-2155 (Washington; Office of Technical Services, Department of Commerce, 1956).

some allowance for the many uncertainties, it seems reasonable to predict that, even under the conditions of radiation exposure in man, shortening of life in the offspring of irradiated fathers will be between 10 and 100 percent of the shortening of life in the exposed individuals themselves. It should be remembered that this excludes an additional effect on the offspring; namely as measured in the mouse, death before weaning age. Also, and more important, since the shortening of life is probably the result of mutations with slight dominant effects, the damage would not end with the first-generation offspring but would, to a certain, and probably large, degree, be transmitted to later generations.

Summary.—Length of life in the offspring of male mice exposed to moderate doses of neutron radiation from a nuclear detonation is shortened by 0.61 day for each r. e. p. received by the father over the dose range tested. This figure excludes death before weaning age. The 95-percent confidence limits are 0.14 and 1.07 days per r. e. p. Extrapolating to a proportional shortening of life in man gives 20 days per r. e. p.received by the father as the point estimate and 5 and 35 days as the 95-percent confidence limits. The offspring were obtained from matings made from 19 to 23 days after irradiation and, therefore, represent the effect of irradiation on germ cells in a postpermatogonial and sensitive stage of gametogenesis. It is probable that irradiation of spermatogonia (the stage that is important from the point of view of human hazards) would give a somewhat smaller effect. However, since the present data show an effect on the offspring which is as large as the shortening of life in the exposed individuals themselves, it seems likely that, even when allowance is made for the conditions of human radiation exposure, shortening of life in the immediate descendants will turn out to be of a magnitude that will warrant serious consideration as a genetic hazard in man.

The author gratefully acknowledges the cooperation of Mr. R. L. Corsbie, Dr. E. P. Cronkite, Dr. H. H. Plough, Dr. R. E. Carter, Dr. E. F. Oakberg, Dr. C. W. Sheppard, and Dr. V. P. Bond, all of whom gave valuable assistance in various phases of the work at the test site. The author is also indebted to Dr. A. W. Kimball for statistical advice and computations and to Mrs. Josephine S. Gower and the other members of the Mammalian Genetics and Development Section who assisted with the laboratory work.

Representative HOLIFIELD. Before we have our discussion, we are going to ask Dr. Hardin Jones, to give his presentation.

STATEMENT OF DR. HARDIN JONES, UNIVERSITY OF CALIFORNIA RADIATION LABORATORY [6]

Dr. JONES. Thank you.

Representative HOLIFIELD. Dr. Jones, this is your prepared statement?

Dr. JONES. Yes.

Representative HOLIFIELD. It will be accepted for the record. Are you going to summarize it?

Dr. JONES. Yes.

(The statement referred to follows:)

STATEMENT OF HARDIN B. JONES, PROFESSOR OF MEDICAL PHYSICS, PHYSIOLOGY; ASSISTANT DIRECTOR, DONNER LABORATORY, UNIVERSITY OF CALIFORNIA

My field is the physiological basis of human health problems. In research I have contributed appreciably to: (1) an understanding of some metabolic disturbances associated with heart and vascular disease; (2) evaluations of the

[6] Donner laboratory, division of medical physics, University of California, Berkeley 4, Calif. Physiology, Los Angeles, Calif., June 11, 1914. Bachelor of arts degree, California, at Los Angeles, 1937; master of arts degree, California, 1939; fellow, 1940–45; doctor of philosophy degree (physiology), 1944. Instructor, medical physics and physiology, California, 1946–47; assistant professor, 1947–49; associate professor, 1949–54; professor, 1954; assistant director, division of medical physics, 1948; research associate, radiation laboratory, 1947–. With Atomic Energy Commission, 1944. Cancer Society; Historical Science Society; Physiology Society. Radiobiology; metabolism, physiology of gas exchange; regional blood perfusion; biological effects of radiation; eipopistein metabolism and physiological change with age (from American Men of Science).

cancer problem and especially human cancer therapy; (3) the study of aging in that I have been able to construct a general explanation of aging that can be subjected to experimental study and which has been useful in evaluating factors contributing to improvement or deterioration of health and lifespan. I have been especially interested in estimation of effects of radiation in man.

SUMMARY

In natural radiation exposure and the extent to which it is increased by fallout, we are dealing with effects that are minute compared to other factors of importance to the health of man. Estimation of the magnitude of these small effects depends on determination of the dosages to be expected and the biological responses associated with these doses. This paper deals with the latter factor.

There is no direct evidence that doses as small as these produce any harmful effects; some have therefore jumped to the conclusion that they produce none. There is much evidence that a variety of undesirable effects occurs in cells, tissues, and whole organisms when exposed to doses large enough to establish either a positive or a negative result with reasonable confidence. Some of that evidence is presented here. Since it indicates an effect proportional to dose in the known range, the gap in the unknown range with which we are concerned is filled by arbitrarily assuming that the same proportionality holds. The principal justification for that assumption as a working hypothesis is that to ignore a small factor of risk, if it is real, may be costly if that small risk is applied to a very large population.

The similarity of the effects of all harmful processes, including aging, disease, and irradiation, has been expressed in terms of a concept involving an equivalence between units of the damaging agency and effective increase in physiological age over the chronological age. Factors obtained in this way may be used to estimate the cost in health and lifespan of any of the circumstances tabulated. As a present step, while recognizing the uncertainties of the assumptions on which it is based, the factor for irradiation may be used to compare costs and gains with respect to the atomic energy program. Much more effort should be devoted to the accurate determination of the quantities involved.

COMMENTS TO CONGRESSIONAL COMMITTEE, JUNE 1957

I am trying to evaluate every circumstance which can add up to or subtract from average human health and useful life span. Some of my colleagues jokingly refer to me as a prophet-of-doom because many of my estimates are best explained in measures of morbidity or mortality risks. This is how we must quantify human experience in order to evaluate factors that may truly be worth accepting or avoiding. It is not enough to know something is good or bad for us; we must express this knowledge in quantitative terms in order to be able to compare costs in health or life span with gains in other directions. Very fbrequently we accept circumstances having a known and measurable risk, such as using the bathtub, crossing the street, overeating, or riding a device for transportation, simply because doing so gives us some definite pleasure or gain that is reasonably more than the estimated cost in risk of harm. However, we are conscious of risk, in that bathtubs now are made with nonslip bottoms, streets have crosswalks and traffic signals, and our mechanical devices are made as safe as is thought to be warranted economically.

Since we are concerned here with the evaluation of radiation effects upon humans, I would like to tell you in some detail about interpretations I believe are reasonable estimates of this problem. I would like to separate information that is generally accepted from that based upon reasonable but debatable arguments; and I shall also use the evident uncertainty regarding answers to critical problems both as a caution concerning current interpretation of results and to indicate needed critical information. My concern for estimation of even minute effects places me at times in awkward situations—when quoted out of context, I may appear to be constructing "scare" stories. This I am not doing. Effects such as I have estimated are too small to be measured directly in individual humans affect estimated numbers of people only when large populations are subjected to the risks in question. Better estimates should eventually be made for each problem we can now discuss, but any estimate will have to depend on statistical studies of large populations.

I state definitely my summary belief that estimated effects of radiation from recommended occupational exposures and from fallout are minute costs compared to the gain to man of abundant useful energy and the extraordinary advantage we have gained for free nations by the awesome presence of effective atomic weapons. I would not, however, assume that, this formula for peace may continue to work indefinitely. Just as defense strategy, economic costs, possibilities for international agreement and other political, economic, and military factors are constantly reevaluated as circumstances change, the biological costs of the atomic age must be reappraised continually.

We all recognize that the world must find more energy to be used for human betterment. Many facts attest to this. Ten percent of the world's population is now using 90 percent of available energy and everyone—whether he already has much or little—wishes to have more of the material comforts made possible through the use of mechanical power. Populations everywhere are increasing, knowledge is increasing, need is increasing. In a world of dwindling available energy, humans very shortly would face a crisis of relative poverty. The availability of atomic energy promises instead a new age for man, marked by extraordinary technical progress that can be the result of both growth of knowledge and increase of efficiency. These asset values are well known; the liabilities are less definitely established. At the moment, we need to find out with much greater precision many basic facts of effects of radiation in man, the biologic costs of radiation exposure, how its effect can be minimized or avoided. It is quite evident that the problems that the atomic age has brought us are more complex than had originally been visualized. At the same time, it is true that the gain through atomic energy is considerably greater than was foreseen even a few years ago. While the physics, chemistry, and engineering of development of atomic energy needed and deserved the great attention they received, it is now evident that the factor of human tolerance of radiation needs further attention because of lack of precise knowledge regarding critical radiation effects. In my opinion, the next stage should emphasize a greater relative effort in this part of the problem, because it is imperative that we reduce speculation and establish estimates of biologic effects with more certainty. At the moment, in spite of the shortcomings of current information concerning these important problems, there seems to be no difficulty in continuing the development of atomic energy and at the same time accepting very cautious limits of radiation exposure.

Much of the information concerning radiation exposure as a problem to man developed almost wholly from the great sponsorship of this field of scientific inquiry by the Atomic Energy Commission. Many of the points currently under critical discussion, such as mutation rate associated with very low radiation doses or the quantitative estimation of fallout, might have been neglected had not those in authority shown a responsible comprehension of these problems and undertaken to support unusual costs as a part of the overhead of development of useful energy.

Now, I would like to present to you an outline of some of the facts and arguments that seem to be of special importance in current estimation of the effects of radiation in man. I wish once again to caution that, in these discussions, we are considering radiation risks that are very much like other commonplace risks to which we have long been exposed, such as driving automobiles—indeed, these newer risks are usually very much smaller.

Everyone wishes that risks to health and life might be reduced to zero. If that could be done, we would all live forever without growing old! Every moment of life, we face certain average risks of mishap—rarely of improvement. These average risks are the sum of many contributing factors, some large, some small. In individual affairs, very small risks are frequently regarded as no risk at all. On the other hand, in a popular presentation of hazards with a view toward emphasizing their importance, any risk that is greater than zero can be presented in a way that attracts concern, even though it may affect only one person in the entire world. A widespread exaggeration of hazards would, of course, have prevented any technical progress. I believe it would be an equally great mistake to dismiss, as being equivalent to zero, those small radiation effects we are now considering. It is true that most of these estimated effects are exceedingly small, and I can give many examples of ordinarily accepted circumstances that modify health to a much greater extent. The problem of differences of opinion derived from the same body of facts, is really the problem of deciding "how small is small," in the light of whatever is to be gained.

Similarity of radiation effect in mammals

In estimating radiation effects in humans, there is a striking similarity to observations established in experimental studies with animals. Cancer induction, radiation sickness, and genetic change occur at equivalent exposures. The average lethal doses are within the same range. Exposures causing burns or tissue damage are comparable. In general, this similarity would be expected from our understanding of the chemistry and physics of radiation effects and from the very similar structure of body cells of mammals of widely varying species. As an approximate truth, damage that is incurred by radiation exposure becomes more apparent as time elapses, so that for many effects, such as the induction of cancer, there is a latent period during which the effect of radiation is rarely observed. Roughly, the latent period for the induction of cancer is relatively long or short, depending upon the relative life span of the species. Thus, greatly simplified, the problem of making comparisons of radiation effect between, say, the mouse and man is a question of the relative time scale. Very similar relations in development of other diseases with time are observed between man and other mammals when time is expressed as a fraction of the life span. In such a comparison, the biology of aging in the mouse is remarkably similar to that of man, if one estimates 1 day's life of the mouse to be equivalent to 1 month's life of man.

Life-span-shortening effects of radiation

The conclusion from all studies of animal populations exposed to radiation in the range of 100–1,200 roentgens of whole-body exposure is that these dosages shorten life span. The shortening of life span of laboratory animals by these exposures is an established fact for either acute of chronic exposure. It is, however, distinctly unproven whether these effects apply on a relative basis to man, though the consistency for the many species of mammals tested lends great plausibility to this hypothesis. If we do make such an assumption, as I have done on several occasions since 1953, one obtains a number that suggests that the average human life span loss at a dose of 100 roentgens may be about 500 days, or about 1⅓ years. We can be certain that some number exists defining radiation induced life-shortening in man, but we cannot state the number with confidence. It can at best only be approximated. It is very important that we do learn what its value is, even though great effort and cost are attached to finding it. In the absence of definite knowledge, we must do the best we can to estimate this quantity.

In discussing this question, several additional considerations arise. Is life span loss proportional to radiation exposure? Most evidence can be interpreted in this sense, but not exclusively. It appears possible that life span loss may be somewhat greater than the above estimate at very large single doses, even though proportionality may hold in the small-to-moderate dose range. For very small chronic exposures, observation of the effect becomes very difficult and it is possible that no effect on life span may exist; however, current data are truly inadequate to test either hypothesis. Nevertheless, in mice, for example, a significantly enhanced and early appearance of tumors has been caused by exposure to 0.1 roentgen per day, the lowest dose yet studied for life span effects; and although no comparison with man can be made on this basis, it may be inferred that some effects occur at low doses. Even though the studies that enable us to speculate upon effects in this dose range have been extraordinary undertakings of technique and labor, we still need to have much more information of this kind to establish the bearing of radiation exposure upon life span loss. One of the most important points concerns whether exposure rate is a factor in the lower dose ranges, since it determines whether one may express this radiation effect in terms of loss per roentgen of radiation exposure. I feel that, with respect to large doses of radiation, the majority of evidence supports a concept that life-span loss is proportional to exposure; and, in the absence of human data on low doses and low rates of accumulation of dosage, it seems safest to assume that the same relation holds at these levels. On this basis, we might say that 1 r. of whole body radiation is perhaps equal to becoming 5 days older. In the absence of definitive information on radiation-induced aging in man, I believe it is reasonable to use this tentative number, even though it may be subject to revision as more certain information is acquired. Study of radiologists and atom bomb survivors may provide a better answer. In this regard there are conflicting opinions and reports. It has been evident for several years that radiologists have 6 to 10 times more leukemia than their expected rates.

Some additional studies of life expectation suggested shortened life span for radiologists. Dr. Shields Warren recently estimated that average age at death was 6 years less for radiologists than for other physicians. Thus, one might infer that radiologists have about 6 years shorter life span. Lewis has recently reviewed this conclusion and reports that, when the distribution of ages among radiologists is considered, the death rate for that group as a whole is no worse—and possibly better—than for physicians in general. However, by using both the individual ages of deaths provided to me by Dr. Warren and the number of registered radiologists, my colleague, Grendon, and I have constructed the age-specific death rates for radiologists. They are found to have the same death rate risk as the general population at ages under 60; but over 60, the death rate is about twice as high as expected. I had come to similar tentative conclusions several years ago by estimating approximate death rates of radiologists from obituary notices in the professional journals. I think it is reasonable to conclude tentatively that radiologists have a higher than expected death risk at older adult ages. This kind of evidence of radiation effect upon man is so important that a relatively great effort should be employed to make a precise study of individuals with known occupational exposures. It is true that the effects are probably very small but they are effects we need to know with relative accuracy, and such study is directly to the joint of estimating the effect of accumulating radiation exposures in humans.

What about dosage measurements in human studies of radiation exposures

Another crucial element in estimation of radiation effect in man is determination of exposure dose. Even though I believe that radiologists may have slightly shortened life span at older ages, there is considerable difficulty in correlating this estimate with a measure of exposure radiologists may have experienced to produce this aging. We simply do not know the average or individual exposures, and direct estimates of exposure can be stated only within rather absurd limits such as 100 r. to 5,000 r. of accumulated whole-body irradiation. There is the possibility, also that only a few greatly exposed individuals may be responsible for the extra deaths, giving a falsely high death rate to the entire group. Such difficulties also plague current attempts to evaluate findings among the Japanese survivors of atomic bombing. It is absolutely necessary to refine estimates of exposure of individuals in order to arrive at a proper evaluation of human life-span effects and to be able to test such data for proportionality or lack of proportionality of induced effects of exposure. A truly valid assessment of radiation effect in man has not been made and would require for its accomplishment the upmost in competent personnel and financial support, on a scale commensurate with its complexity. A great part of the potential and useful evaluation of effects of radiation in Hiroshima and Nagasaki is in jeopardy because too little is known concerning true exposure. The several evaluations of leukemia risk in these Japanese are in doubt because exposure is unknown and evidence for exposure is quite inconsistent with distance from the blast and shielding. Thus, the true magnitude of the leukemia-inducing effect of radiation may be very much smaller or larger than it is now estimated to be. With some effort, the dose might be estimated for accurately for those who developed leukemia and those who were in certain exposure categories. Many critical decisions concerning effects of radiation will need to be made, and it is important to have a proper number to apply in hte construction of these estimates. If such a number can be obtained by making an additional effort, I believe we should do so.

Proportional effects versus threshold effects in man

Attention to effects of radiation first established that there are acute effects, especially marked by tissue destruction, from which recovery subsequently takes place. Recovery in this instance is of the same quality as recovery from any usual kind of injury. As the amount of radiation given in a single dose declines, acute effects decrease more rapidly than the dose, because acute effects are due to tissue destruction and too little damage is done at any one site at doses of about 100 r. and below to evoke measurable systemic responses. Thus, there are some kinds of radiation effect that are not seen at doses of about 100 r. or less. These include immediate radiation sickness, death, burns, ulceration, hair loss, severe anemia, or sterility. All of these symptoms are largely the result of destructive effects of radiation upon rapidly dividing cells. Such cells for the most part are being replaced at a rapid rate, so that the loss of a small number may be quickly compensated. When radiation damage is considered in

terms of acute effects developing soon after radiation exposure, there is general and convincing evidence of at least partial recovery from the effects of exposure. Attention exclusively to this phase of the problem leads some to the opinion that human can tolerate single intense exposures to several hundred roentgens and subsequently recover all of the gross features of normal health.

As we direct our attention to injury of tissues or cells instead of confining it to whether an individual lives or dies, we find in general that effects of radiation are much more nearly proportional to radiation exposure. For whole categories of effects, such as genetic effects, destruction of cells, artifical aging of individuals, and induction of cancer, there is evidence—some completely convincing, some only a reasonable argument—that radiation effects at the cellular level are proportional to exposure to radiation. It is quite possible that some of the effects, such as the induction of cancers or of artificial aging, may not be as likely to occur per roentgen of radiation exposure at low exposures as at higher exposures. These points will be settled only by additional study of the problem experimentally in various animal species and by utilizing every opportunity to study chance human exposures. As a working estimate, it seems reasonable to postulate that effects at low dosage and low rates of dosage accumulation are proportional to know effects at higher doses. First of all, such a working hypothesis seems plausible in the light of the experimental evidence previously mentioned, and secondly, it is a cautious position to take to protect human health.

I believe that the general evidence relating biological effects to exposure shows a remarkable similarity between genetic effects and changes in cell numbers and cell quality in the other body tissues (somatic tissues). Muller, for example, argued as long ago as 1939 that the somatic tissue effects of induced aging and carcinogenic change were quite comparable to genetic effects induced by radiation, and that both effects upon the somatic cells were probably of the same kind as the genetic changes in cells measured directly by the geneticist. Radiation effects on various cells of mammals, such as genetic effects and the survival of cells, including germinal cells, blood-cell-forming cells, and embryo cells, roughly fall into a category of proportional effects of radiation in which the chance of effect is 1 to 3 cells affected out of a thousand, per roentgen exposure.

Radiation effects such as upon growth are quite in keeping with other effects at the cellular level, and human growth change comparable with experimental values of radiation growth suppression in mice. The sum of such evidence suggests that life-span changes and induction of cancers are accelerations of normal aging produced by decreases in cell numbers and cell qualities in amounts proportional to radiation exposure. The effect is frequently made more complicated, it is true, by the ability of the body to repair tissue damage through replacement of injured cells with functional ones; but, fortunately, this modification helps us to escape acute radiation effects.

Were it not for the simultaneous development of atomic energy and broadly supported studies applying to health safety, we would not be in today's position of understanding radiation induction of cancer and other effects to the point of realizing that we should estimate these effects at very low exposures. Only a few years ago, we would have assumed that such effects could not exist. Attention to the effects of radiation in the small-dose range means that these efforts will be extended and that the problem becomes much more difficult technically. It is important to emphasize, in addition to life-span-shortening and cancer-inducing effects, the effect of radiation from generation to generation and upon embryologic development, both of which may be effects that are proportional to radiation exposure into small-dose ranges; if they are, they need to be estimated in terms of human costs.

I would like to turn attention to some of the other environmental effects with which radiation exposure may be compared. For several years, I have been evaluating a large number of environmental circumstances collected from various sources in terms of effects upon the life span and death rate. A summary tabulation is given in tables I and II. The exact numbers attached to some of these effects are in dispute, because there are different choices concerning the estimated magnitude of the effect. Some of the effects are related to reversible circumstances as, for example, marital status, occupational hazards, metabolic disease; other factors, such as radiation exposure, are permanent. The latter class includes traumatic injury, childhood disease, and country of origin. Some of these estimated effects are additive; others are not additive because they are partial measures of the same problem. For example, if one estimates the effect of fat

metabolism on life span from consideration of fat-carrying molecules in the blood, this estimate will already contain the evaluation to life risk that can be obtained from obesity status; and, similarly, the cigarette-smoking effect is also evaluated by blood fats, so that these are not additive. Differences between males and females, urban versus rural dwelling, and national differences appear to be entirely additive.

Differences in life span in this list are given in terms of change of physiologic age. An individual in a category listed as +3 years—as, for example, when a person's mother lived to be 90 while his father lived the average life expectancy—is considered to have the death risk at any adult age of a person 3 years younger chronologically.

Such effects on life span can also be established for, say, automobile driving or employment in industry. The effect corresponding to recommended limits for human radiation exposure (as, for example, 50 roentgens of accumulated exposure in occupations) is very much less than any of these other estimates, perhaps being —0.7 year. The effect of fallout estimated by these methods to be about one one-hundredth of this value. It is quite obvious that each of these effects can be worth our attention, although the average person is not aware at any time that change is taking place. Even such effects as a difference of 10 years in physiologic age are quite unlikely to be noticed by the casual observer watching the tide of life from day to day. Detection of these effects is possible only by employing statistical tools, and the conclusions apply only to averages—not to any one specified individual, except in terms of the statistical concept of risk.

The additive nature of disease categories and factors underlying development of disease suggests a very important point with regard to radiation effects, namely that, even though we cannot reverse the effect of radiation damage itself, it should be possible to counter this effect by enhancing health in other ways. The general recession of disease and improvement of health over this century are clear evidence that great gains can be made in the direction of better health and longer useful life. Some of these gains may more than offset the adverse effects of radiation, and some of these gains may be expected to arise as the direct result of the AEC program in research.

TABLE I.—*Relative displacements of physiologic age by factors that accentuate aging or loss of life span (minus time) or retard aging (plus time)*

REVERSIBLE	Years
Country versus city dwelling [1]	+5.0
Married status versus single, widowed, divorced [1]	+5.0
Overweight: [2]	
25 percent overweight group	−3.6
35 percent overweight group	−4.3
45 percent overweight group	−6.6
55 percent overweight group	−11.4
67 percent overweight group	−15.1
Or an average effect of 1 percent overweight	−.17
Occupational exercise versus sedentary occupation [3]	+5.0
Smoking: [4]	
1 pack cigarettes per day	−7.0
2 packs cigarettes per day	−10.0
Atherosclerosis: [5]	
Fat metabolism from consideration of cholesterol or lipoprotein concentrations in human serum:	
In 25 percentile of population having "ideal" lipoprotein concentrations	+10.0
Having average lipoprotein concentrations	0.0
In 25 percentil of population having elevated lipoproteins	−7.0
In 5 percentile of population having highest elevation of lipoproteins [9]	−15.0
Diabetes: [6]	
Uncontrolled before insulin—1900	−35.0
Controlled with insulin:	
1920 Joslin Clinic record	−20.0
1940 Joslin Clinic record	−15.0
1950 Joslin Clinic record	−10.0
Antibiotics	+

PERMANENT	Years
Female versus male sex [1]	+3.0
Familial constitutions: [7 8 9]	
2 grandparents lived to age 80	+2.0
4 grandparents lived to age 80	+4.0
Mother lived to age 90	+3.0
Father lived to age 90	+4.4
Both mother and father lived to age 90	+7.4
Mother lived to age 80	+1.5
Father lived to age 80	+2.2
Both mother and father lived to age 80	+3.7
Mother died at 60	−.7
Father died at 60	−1.1
Both mother and father died at 60	−1.8
Recession of childhood and infectious disease over past century in western countries	+15.0
Life insurance impairment study: [7]	
Rheumatic heart disease, evidenced by—	
Heart murmur	−11.0
Heart murmur plus tonsillitis	−18.0
Heart murmur plus strep infection	−13.0
Rapid pulse	−3.5
Phlebitis	−3.5
Varicose veins	−.2
Epilepsy	−20.0
Skull fracture	−2.9
Tuberculosis	−1.8
Nephrectomy	−2.0
Trace of albumin in urine	−5.0
Moderate albumin in urine	−13.5

[1] Vital Statistics of Denmark, Netherlands, Sweden.
[2] L. I. Dublin and H. H. Marks, Mortality Among Insured Overweights in Recent Years, presented at 6th annual meeting, Association of Life Insurance Medical Directors (Metropolitan Life Insurance Co., October 1951).
[3] The Registrar General's Decennial Supplement, England and Wales Occupational Mortality, pt. I, 1951 (Her Majesty's Stationery Office, London, 1954).
[4] E. C. Hammond and D. Horn, The Relationship Between Human Smoking Habits and Death Rates, Journal American Medical Association, in press, presented at annual meeting, American Medical Association, New York, June 4, 1957.
[5] J. W. Gofman and H. B. Jones, Obesity, Fat Metabolism, and Cardiovascular Disease, Circulation 5, 514 (1952).
[6] E. P. Joslin, H. F. Root, P. White, and A. Marble, The Treatment of Diabetes Mellitus, ninth edition (Lea and Febiger, Philadelphia, 1952).
[7] Society of Actuaries, Impairment Study (Peter F. Mallone, Inc., New York, 1951).
[8] As measured in 1900 (Beeton and Pearson). These effects may be measurably less now, as environment is changing to produce greater differences between parents and progeny. Also, in 1900, it was a greater feat than now to live to be 80 or 90.
[9] This 70 percent difference in distribution of lipoproteins, between 25 percent lowest and 5 percent highest, is equivalent to a total of 25 years in relative displacement of physiologic age.

TABLE II.—*Statistical distribution of lifetime shortening by travel and industrial accidents* [1]

[Calculation based on Vital Statistics of 1949, values for adult white males 20 years and older

All accidental deaths	−2.3 years per individual in United States of America.
Travel accidents:	
Accidents involving railways	−0.06 year per individual in United States of America.
Accidents involving ships	−0.04 year per individual in United States of America.
Motor-vehicle accidents involving driver and passengers.	−0.67 year per individual in United States of America.
Assuming only half of population spends appreciable time in automobiles.	−1.3 years per individual at risk.
Pedestrian motor-vehicle accidents	−0.2 year per individual in United States of America.
Assuming this effect largely involves the urban portion of the population.	−0.4 year per individual at risk.
Aircraft accidents	−0.05 year per individual in United States of America.
Assuming that ¼ of the population (actually, probably much less) uses airplanes.	−0.2 year per individual at risk.
Accidents involving industrial machinery	−0.04 year per individual in United States of America.
Assuming only 30 percent of males are employed using industrial machines.	−0.27 year per individual at risk.

[1] These values are based upon numbers of deaths attributed to accidents; the estimates of life span lost are actually perhaps slightly low because survivors who are maimed, and hence have reduced life expectancy, are not included in these estimates.

Dr. JONES. I will deviate from the prepared statement to save time. I can use the blackboard and anyone reading my ad lib remarks can fall back back on the prepared statement if he has difficulty following me.

Representative HOLIFIELD. That will be fine.

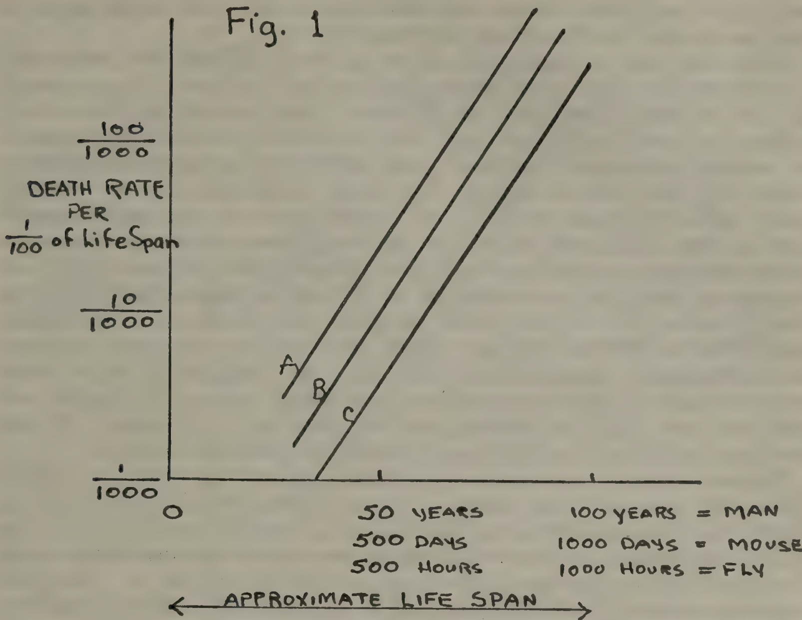
Dr. JONES. My general problem in health research is to try to evaluate as many things as we can that have some bearing on human biology, human biology from the standpoint of the degree of good health we might achieve and how long we can maintain our life in relatively good health. These things can be expressed in terms of length of life, they also have to do with measures of death rate, because this is how we measure the risks of failing to remain alive.

I would like to present a diagram of this (fig. 1) because I think it helps to understand the problems of human biology. Ordinarily vital statistics for man are presented in units of death rates, that is, deaths per thousand of population per year. This level would be 1 death per thousand per year, this would be 10 deaths per thousand per year, 100 deaths per thousand per year, and so on.

I regret giving information in these terms because they are exceedingly morbid values to deal with. Sometime in the future I would like to turn them around and express them in units of vitality.

We are interested in the problem of vitality and not the problem of morbidity. If we draw this function (line B, fig. 1), this could essentially represent the aging of men or women of the United States. These are approximately the age-specific death rates. If we consider individuals at the age of 30, they have a risk of approximately 2

Fig. 1



deaths per thousand individuals per year. At age 50, we find the death risk per year is 10 per thousand and so on.

We find this is a very regular sequence of events. It was described over 130 years ago by Benjamin Gompertz.

The very interesting thing about this is that if the horizontal distance represents life span of man in years, we can convert this equation to the biology of the mouse by changing the time scale to make the life span equal 1,000 days and then our line represents the aging of the mouse. The longer he lives, the larger the risk of dying.

One can also interpret the increasing death risk on the basis that physiologic degeneration is an accumulative affair. The more degeneration accumulates, the more likelihood there is of further decay; and the more decay in toto, the more the death risk. These concepts are relatively important to us because they may explain the very great differences in death rate between populations, either of humans or mice. If we plot the death rates for several populations of mice, we might get marked differences in death rate at each age (lines A and C, fig. 1). The interesting thing is that the biological equation always gives us the same slope. That is, the rate of progression of the death rate tendency is always the same for that species.

In this case, the death rate doubles for each age increase of approximately 80 days for the mouse, or approximately 8½ years for man. Otherwise, the equations are very much the same. The range of variations shown (a factor of 4) in death rate at any age occurs under average circumstances.

I can illustrate some of the differences among human populations. If we say this line represents death rates in the United States today, the Scandinavian countries are in a much better position, with lower death rate risk on the average. We can separate out some population

groups in the United States and find intrinsically low death rates, as good as those of the Scandinavian countries.

What has been happening? If we go back at least 100 years, say, to 1850, we find high death rates; and we find that over the course of the last century, throughout the Western World, death rates have been shifting in the direction toward lower and lower values, even though the rate of change of the death rate with age has remained characteristically the rate of change for man. This helps us to identify many factors that are probably of importance to us in a public health sort of way. I do not think that some of these things can be established with great certainty, but at least the effects of certain conditions have been identified as existing or not, which is quite important to us. One of the main things that has caused a shift from very high death rates in adult life to very low death rates at the same age, has been the recession of the childhood diseases. As childhood diseases have been eliminated, due to progress of nutrition and public health and medicine over the past century, we find that health in adult life has also been better and better, as evidenced by lower death rates.

We can identify a number of factors that have an effect on health. I will put some of them down. The recession of childhood diseases during the last century has added about 15 years of useful adult life. We can tentatively estimate differences associated with exercise—a deplorable thing to discuss when we do not get enough of it. The gain due to exercise might be of the order of 5 years. The effect of obesity, based on life-insurance statistics, is apparently proportional to extent of obesity, where 1 pound of overweightness is equal to 1 month of lifespan.

If you think of it the other way, if you are 1 pound leaner, it works in reverse or you have a 1 month gain.

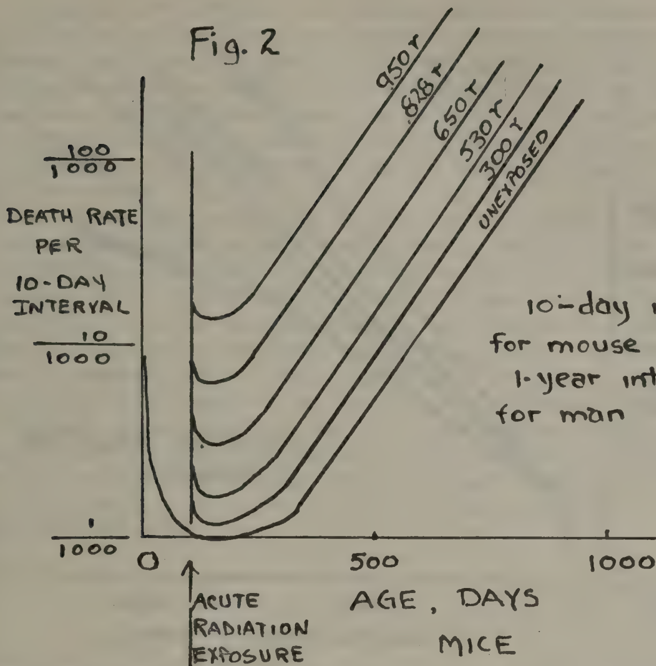
There are differences between living in the country and the city. There are very great differences between various population groups by nation of origin, not related to politics but to human environmental factors.

This type of analysis is important to us because it gives us a background to use with respect to the irradiation problem; particularly, if we look at the effects of irradiation on the mouse, we can use these same equations.. We will be talking in terms of a lifespan of a thousand days instead of the human lifespan of 100 years. The biology of aging of the mouse is like this [drawing on blackboard] (fig. 2). If we give single doses of radiation early in life, what we find is that the death rate goes up momentarily, due to the acute effects, and then, among the mice that recover, there is a displacement of the death rate just as though these mice were already older than they were before.

This apparent aging is the primary thing that characterizes the radiation effect on mammals; the general things that cause death are just about like the things that would have caused death anyway if the animals had been a little older. There are certain things that might really be specific for radiation toxicity, but in general radiation injury represents an increase in natural tendencies toward death. If we give twice as much radiation, this line would move up twice as far.

This brings us, then, to the concept of proportionality. As far as this kind of increase in the death rate is concerned, we seem to have a linear displacement that will move the line toward higher death

Fig. 2



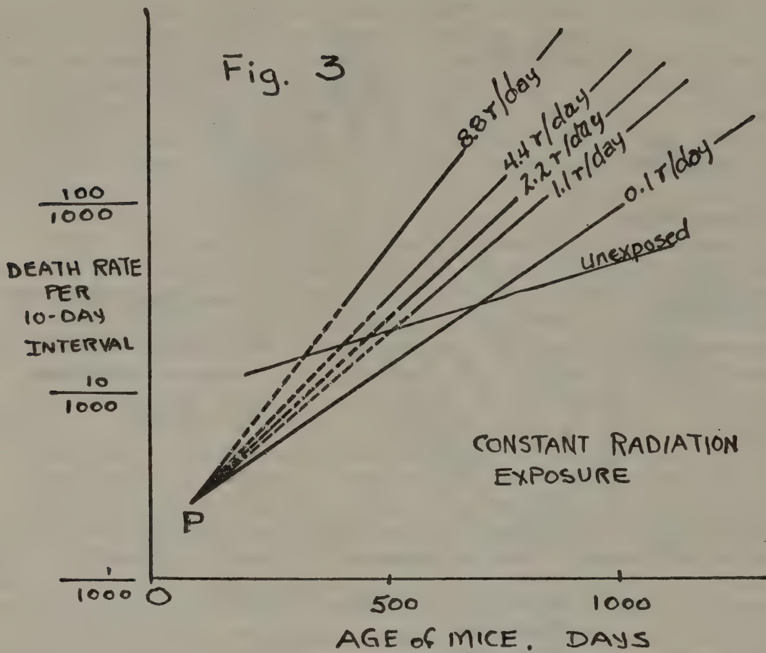
rates in amount proportional to exposure. The amount of injury, which is measured by the increase in death rates, is proportional to the amount of single-dose exposure between 100 and 1,200 roentgens. We can't go to higher exposures because the best techniques we have available will not enable these animals to live beyond the acute phase.

So this gives us one of the best illustrations of proportionality of radiation effect. Even so, there is a great deal of debate among my colleagues as to whether we should really conclude that these effects on life span are still proportional at very low doses. The reason for that is that the basic information we have to go on is relatively scanty. In spite of the fact that we would like to have better data, it has taken a great deal of effort on the part of most of our great laboratories to accumulate the information we now have.

Some of the experiments have been rather great undertakings in terms of numbers of animals, housing of animals, and the numbers of scientific investigators that have been a part of these studies.

A very large study was done in Operation Greenhouse, one of the atomic blasts in the Pacific, where large numbers of mice were used, and where we have good tests of the proportionality. Even in this case there was a difficulty in estimating the physical doses of these animals, and in the higher dose ranges it looked as though the effects on life span were relatively greater than at the lower dose rates. However, at the lower dose rates, the concept of proportionality still seems to hold in that the curve becomes a straight line which seems to go through zero effect at zero dose.

Some of the other difficulties in establishing proportionality of radiation damage are evidenced in terms of another effect that was demonstrated in mice, and that is the chronic radiation exposure ex-



periment of the late Dr. Lorenz. Fairly large numbers of mice were used in this study. In this case, when radiation is given constantly, one does not find that these lines are displaced in a parallel fashion; but, because the little increments of radiation damage are constantly being given, the line has a steeper slope, and the slope of this line will increase in proportion to the amount of radiation given.

So then, if radiation began at this point (point P, fig. 3) and we have varying dosages—maybe this is a tenth of a roentgen per day, this might be 1 roentgen per day, 2, 4, and 8—one can see that the change of death rate with age becomes steeper and steeper as we go to the higher chronic exposures. Unfortunately, with respect to interpretation of the results at the lowest dosages, the control mice were housed separately.

As far as the direct comparison of slope of the death rate curves is concerned, the unirradiated controls seemed to have a lesser slope than even the 0.1-roentgen-per-day group. But when the curves are put together, the control mice intersect in this fashion, (fig. 3), so that early in life the control mice had a higher death rate than the mice irradiated at 0.1 r. per day, and this variation essentially happened twice. So that the direct observation is that the animals that had a tenth of an r. per day lived slightly longer than those that had no irradiation; but the former group did have twice as many tumors as the latter.

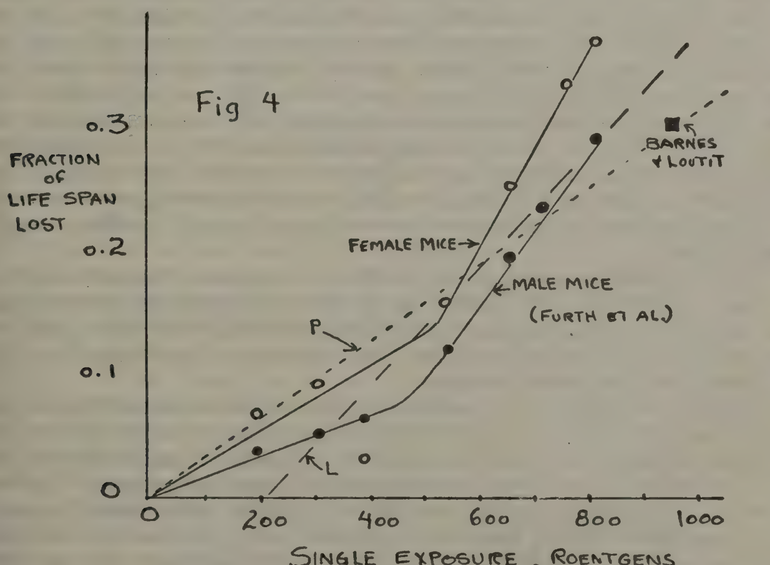
Such differences in death rate are seen quite commonly among animals that are housed separately in slightly different environments, in spite of every effort to make conditions identical. In such cases, however, the slope of the line has remained the same, but the position of the line has been displaced.

I don't know any way out of this particular dilemma except to get further information from repetition of these experiments. The anomaly has given some people a bulwark to fall back upon in arguing that perhaps there is a threshold in terms of radiation effect on the life span. On the other hand, I would place little confidence in this argument, because these animals were housed separately.

Representative HOLIFIELD. What do you mean housed separately and controlled?

Dr. JONES. The experimental mice were chronically irradiated, so they could not be kept in the same room or region of the building as the control mice, which are kept in a normal environment for comparison. They had to be housed in a different room. These particular control animals were even raised at a different time period because the animals that were contemporary with the mice that were irradiated happened to have an infection and the colony was lost. This limits the interpretations one can place upon these experiments.

Even so, expected effects of 0.1 r. per day are so slight, and the numbers of animals are so small, that the results do not let the investigator test significantly either way whether the effects of irradiation on life span are subject to a threshold or are continuous in the small-dose range (fig. 4).



Solid lines are best fit, aging proportional to dose
LINE-P, possible fit, aging proportional to dose
LINE-L, possible fit, aging beginning at a threshold of 200 roentgens

So we have to conclude in general that there is no direct evidence of life span effects in the small-dose range. We can only draw inferences from effects at higher doses. But I believe if we add everything we have available in the higher dose range, of the order of 100 r. up to 1,000 r., that the effects seem to be reasonably proportional to

radiation exposure. Just as the geneticists have trouble in going below 25 roentgens in testing proportionality, I think we have trouble going below 100 r. in establishing that life span effects can occur at very low doses. It is certainly possible to test for radiation effects in this region, but it will be difficult and the testing will not be completed for some time to come.

I also think that everywhere possible we should make an effort to obtain such information directly from man. Of course, there is the study of the Japanese. There is also the study of the radiologists that we discussed briefly yesterday. These studies have some limitations. If one makes the best estimation possible of the death rate for radiologists and compares it with the white male population, it does appear that up to the age 50 the population of radiologists and the white male population have similar death rates. Above this age, the death rate of radiologists is increased above the general population, so that the displacement in death rate, which corresponds to age, makes radiologists effectively at least 10 years older than nonradiologists of the same chronological age. This really only tells us that a life span effect of radiation can occur in man. It is very difficult to say what the ratio of life-shortening to radiation dose is.

It is very difficult to say what the exact number is because the numbers of radiologists have been increasing in each year's time. Over the last 20 years, the number of radiologists has increased by a factor of 3 or 4. With this increase, there has been an increase not only in the younger ages but in all ages. So we have no idea as to what the average dose of radiologists may have been. We can only guess that this 10-year difference in aging is associated with an average dose of somewhere from 200 r. to perhaps as much as 1,000 r. I personally doubt whether this 1,000-r. value taken as an upper limit could really be expected, because I think the effects of 1,000 roentgens of whole body irradiation would be more severe than we see it in the average radiologist.

There is, however, very little doubt that many of the radiologists who are responsible for this higher death rate are really groups that had much greater than average irradiation, and that the main population of radiologists having much less exposure than these few are really subject to a lower death rate.

It is also important at this time to point out that we really need to have a value that we can say represents the effect of radiation on the life span of man. The best thing we can do directly in the absence of such a number, I believe, is to estimate it from the mouse. Unfortunately, we know so much about the biological equations of aging, how diseases develop in both the mouse and man, that we can use this system of simply converting 100 years to 1,000 days to give us this information directly, and use it with whatever confidence limit needs to be applied, remembering that the information is from the mouse.

When we do that, we will find that, taking the mouse data directly, we might conclude that 1 roentgen perhaps—and the "perhaps" is big—is equal to minus 5 days in terms of individual life span. On the average, the individual exposed to 1 roentgen would be like an individual without this amount of radiation who was 5 days older. You see, for all practical purposes within the model as constructed, radiation exposure simulates the ravages of time. So we are justified from the standpoint of the general logic of these events in making this conversion between roentgens of exposure and units of life span loss.

But this is not all. This value, within the confidence limits of the data, might truly range from 1 day to greater than 15 days. We simply don't know. I would advise us all to look at the problem as carefully as possible and use whatever means we can to get such information.

While I cannot be certain where this value may truly lie, the sum of all information I have available to work with would lead me to trust tentatively as a direct average without weighting it for any factor at all this value of 5 days. If we look at the Japanese data in terms of death rate, it is possible that these will fall somewhere within the liimts of 1 day to perhaps greater than minus 15 days per roentgen. We simply do not know yet.

I had occasion to make some very rough estimates some time back and all I could say was that it looked as though there was a life span loss on the Japanese. As we know, there is quite a leukemia rate associated with it, similar to the increase in leukemia rate in radiologists.

Very briefly, I might examine a few factors that are perhaps related to the shortening of life span. They may help to explain radiation effects. I think this particular result of radiation is possibly due to some combination of two effects: the tendencies for the cells to undergo mutations, such as Dr. Muller and others described as somatic mutations, and also the reduction in numbers of functional cells in the animal. If we look at the numbers of functional cells in the animal, or the effects of radiation on mutation rate, we would be looking in effect at a corresponding increment of the death rate. If we look at the cells directly in terms of the response to radiation. for many cells in mammals we will find that there is proportionality in numbers of cells left alive, and number killed per roentgen.

From a lower value of 10 roentgens up to values of 2,000 roentgens in the rat or in the mouse or human blood cell estimates, it appears that, per roentgen in this range, you would have between 0.3 percent and 1 percent of the cells killed. It looks as though the effect of radiation in mammals under a variety of circumstances, for cells like the marrow cells or lymphatic cells, is within this range.

Also, the cells in the developing embryo, which also have a high rate of cell division, have approximately this same range of radiation sensitivity. We can show that the small effect of radiation on the growth of children irradiated before birth in Japan is about the same rate as this, 0.3 percent per roentgen, and this turns out also to be equivalent to the data that Dr. Russell described a moment ago on his irradiated mice. So we have some general idea that in the various mammals we can study, the effect upon cell numbers seems to be relatively the same among the various mammalian species, and also the life span effects seem to be roughly the same as far as we can test; but it does leave us with the great desire to know much more about these effects, and also to know what kind of number we should estimate for the life-subtracting effect of radiation in man.

Senator HICKENLOOPER. Do I understand that range you put on the board a moment ago from 10 roentgens to 2,000 roentgens, that an exposure of 10 roentgens would kill 0.3 percent of the cells and exposure of 2,000 roentgens would kill 1 percent?

Dr. JONES. The range of 0.3 percent to 1 percent of cells killed per roentgen applies throughout the range from 10 roentgens to 2,000 roentgens. One has to interpret the proportionality in what we call

the hit equation. You cannot kill the organism twice. So that in upper doses very few cells are left untouched.

Senator HICKENLOOPER. That is the thing that confuses on that statement. I would interpret what I understand you to mean that as compared to an exposure of 10 roentgens and 2,000 roentgens that the 2,000 roentgens would kill only about 3 times as many cells percentagewise as the 10 roentgens. That is my understanding of your statement.

Dr. JONES. No. If we test for this range where we have available data in the mouse, regardless of whether we give 10 roentgens or give 2,000 roentgens, we will find that we have 0.3 percent of the cells affected per roentgen. So that, per roentgen, as far as the effect on rapidly dividing cells is concerned, it will be three cells of each thousand cells affected.

Senator HICKENLOOPER. I understand. I missed the equation to the single roentgen.

Dr. JONES. Yes. The remarkable thing is that this is such a good proportionality effect.

Representative HOLIFIELD. Dr. Jones, we want to adjourn at 5 because the committee has to go into executive session on another matter.

Dr. JONES. I can summarize this section. I think life-span effects do exist. I have no reason to doubt this at all. I have some reason to believe that we should look with caution on the argument that a threshold effect exists, although we cannot be absolutely certain that a threshold effect might not exist. But as far as my opinion is concerned, on the basis of having examined all the facts at my disposal, I do not believe a threshold effect is very likely to exist, I think we should try to get better information, not only for this point, but also to find out directly in man how much each unit of radiation subtracts from life span, because in an atomic age everyone is going to need to know this number with a great deal of certainty. At least, it should be determined to 1 or 2 significant figures and not merely within the range of perhaps a factor of 10.

Representative HOLIFIELD. Thank you. Your summary is very interesting, and the statement that you have submitted will be studied carefully.

I have two papers by Dr. Jones that I would like to place in the record at this point.

(The material referred to follows:)

A SUMMARY AND EVALUATION OF THE PROBLEM WITH REFERENCE TO HUMANS OF RADIOACTIVE FALLOUT FROM NUCLEAR DETONATIONS

Hardin B. Jones,[1] Donner Labortory of Biophysics and Medical Physics, University of California, Berkeley, Calif., January 14, 1957

ABSTRACT

The tolerable amount of radiation exposure to humans is probably less than formerly estimated. It is shown, however, that accumulated effects of the low-level worldwide exposure to radiation from fallout to date is relatively small. The genetic effects are not large enough to be statistically detectable. The health effects, as expressed in life expectancy, are much smaller than those of such factors as infectious or chronic disease, metabolic disturbances, smoking, obesity, lack of exercise, and environment and marital status. Predictions of strontium 90 levels to be expected in the next 2 decades indicates, however, that bone irradiation may become detectably harmful.

[1] With suggestions and critical review gratefully acknowledged to R. Lowry Dobson, John W. Gofman, John H. Lawrence, Burton J. Moyer, William Siri, Curt Stern, and Edward Teller.

INTRODUCTION

Nuclear detonations form radioactive isotopes in quantities so enormous that they must be reckoned in terms of many thousands of curies, the equivalent of many pounds of the element radium. A portion of this radioactivity is dispersed into the atmosphere and subsequently falls upon the land and sea. However, the vastness of the land, air, and water of the earth provides a means of dilution so great that even these large quantities of radioactive materials are soon reduced by distance and time to exceedingly small concentrations of radioactivity. The problem of radioactive fallout in relation to human beings involves the need to know the quantity that becomes a part of human environment, and to know the effect upon man of ionizing radiation from fallout.

This summary concerns low-level worldwide fallout. It must be recognized, however, that very intense fallout may be experienced in the vicinity of an atomic detonation. For example:[a]

"On March 1, 1954, an experimental theromonuclear device was exploded at the United States Atomic Energy Commission's Eniwetok Proving Grounds in the Marshall Islands. Following the detonation, unexpected changes in the wind structure deposited radioactive materials on inhabited atolls and on ships of Joint Task Force 7, which was conducting the tests. Radiation surveys of the areas revealed injurious radiation levels; therefore, evacuation was ordered, and was carried out as quickly as possible with the facilities available to the task force.

"Although the calculated accumulated doses to the exposed human beings were believed to be well below levels that would produce serious injury or any mortality (267 Marshallese received 14 r. to 175 r.) * * * All of the exposed individuals have recovered from the immediate effects (burns, loss of hair, anemia) without serious sequelæ. Nevertheless it is planned to evaluate the medical and genetic status of the group at appropriate intervals with a view to learning what if any of the known late effects of radiation exposure may be observed. Obviously and indeed fortunately the number of persons (92 Marshallese) receiving 75 roentgens exposure and greater is too small to make it possible to determine with any degree of accuracy the effect on life span."

EFFECTS OF RADIATION EXPOSURE IN HUMANS

The reasonable and superficial evaluation of radiation hazard is that humans can obviously tolerate exposure to several hundred roentgens, recover from immediate effects, and remain in "normal" health and functional capacity. Recently, however, we have become aware of deleterious long-term effects of radiation which, however subtle, appear to be proportional to the total quantity of radiation exposure and may be assumed to act even at very low levels of irradiation. Except for these long-term changes, our understanding of radiation effect usually has been concerned with two important facts that dominated our thinking about these problems:

(*a*) For certain kinds of radiation damage and injury, there is recovery. Individuals recover from acute symptoms produced by sublethal radiation exposures even though they may show general sickness, burns, loss of hair, anemia, etc. Recovery from acute radiation effects is analogous to recovery from any other acute injury or infectious process in which damaged tissue is healed and repaired.

(*b*) These obvious signs of radiation effect are associated with relatively large single doses of radiation (greater than 100 r.). As dose size is decreased, detectable acute effects decline, becoming disproportionately small, so that there is a true threshold of dose of irradiation, at about 100 r., below which these particular acute manifestations of radiation do not occur.

THE PROPORTIONAL-EFFECT CONCEPT OF IRRADIATION

Recent evidence that long-range effects of radiation simulate aging effects comes from a variety of sources and is consistent with information relating radiation effects with genetic change and changes in cell-population numbers and quality. Evidence and logic support an argument that small increments of radiation-induced morbidity persist as small permanent changes in body functional struc-

[a] Quoted from Charles Dunham, A Report on the Marshallese and Americans Accidentally Exposed to Radiation From Fallout and a Discussion of Radiation Injury in the Human Being (United States Atomic Energy Commission, July 1956).

tures, which become detectable as aging, neoplastic disease, and genetic change (see appendix A). However, it remains to be proven experimentally that these effects do occur as the result of small irradiation exposures. The testing of this question is not likely because it would involve great technical difficulties. Attempts to procure some evaluation of the problem relating small-doze (5 r. to 10 r.) effect to life span would involve study under uniform conditions of perhaps several million mice. It is pointed out that in order to establish the effect of the smallest doses as yet measured for genetic effect, namely 25 and 50 r., the geneticist Curt Stern and his associates worked for 6 years and examined approximately 50 million individual flies. They came to the acceptable conclusion that these small exposures have the same effect per roentgen upon gene mutation as at higher exposures to radiation. Ionizing-radiation effect, in the depression of blood-forming function and blood-forming cells, is proportional to radiation dose even down to 5 r. (Hennessy and Huff). Other effects upon blood cells, leading to abnormal doubling of the cell nucleus, are now reasonably established by Dobson in the range of 0.1 to 0.3 r. of single exposure, but are not yet tested for proportionality.

There is no reason to doubt the general evidence of a proportional effect of radiation; but it is also possible that linear extrapolations of higher doses to the small-dose range may not give a true representation of the problem. It is known for some kinds of cellular response to radiation that there can be no effective change in function until two or more similar critical entities within the cell are affected. Some kinds of observed injury, however, appear to depend upon the effect on one critical entity per cell; and other observed injuries may be the result of damage to any one of a number of critical functional parts. The response that depends upon a chain of two or more detrimental changes shows a lesser apparent effect in proportion to radiation exposure at the low-dose ranges. Although this kind of irradiation response does not argue against extrapolation of radiation effect, it may explain a factor of —2 or —4 buffering against detectable radiation effect in the lowest exposure ranges; or it may even have the opposite effect, because radiation effect can add onto partially initiated dysfunctional changes in structures that otherwise would have remained functional.

Radiation effect is most frequently estimated in animals that are rather uniformly irradiated over the whole body. Thus, we are usually generalizing from the observed result of whole-body exposure. In some studies (Kaplan), shielding a relatively small portion of the bone marrow from radiation may protect the animal from generation of thymic tumors. In others, local irradiation is associated with induction of cancer in that region, quite independently of exposure or shielding of the remainder of the body.

The problem of estimating radiation effect and making recommendations concerning it is not the simple problem of avoiding exposure at levels at which there is a detectable or predictable response. This is especially true when considering radiation effect through systems that allow proportional extrapolation to very small radiation exposures. It is always important to keep radiation exposure to a minimum; but it is also important in the understanding and evaluation of the relative importance of radiation effect to establish its place in the entire climate of factors that can modify health. Similar—and, at times, greater—effects upon health can be shown to result from a large number of common environmental factors.

Also, the problem is not simply that of effect of body irradiation upon health. It is necessary to evaluate the effect upon human beings of all known phenomena resulting from the onset of the atomic age, including general socio-economic factors related to our well-being, which are dependent upon progress and the development of useful energy.

ADVANTAGES MINUS COST EQUALS NET GAIN

The sum of evidence would lead to the conclusion that radiation probably does affect man's health subtly, and—like money and time—it should be exchanged for equivalent advantages.

Since the usefulness of atomic energy—including material and energy gain and defense measures of prime importance—is a positive result, and the radiation effect upon humans generally is a negative result of the atomic age, atomic-energy usefulness minus harmful radiation effect must be equated to the net gain. Therefore it is critically important to estimate hazard quantitatively, and to be mindful of other factors while doing so. However, there is no unanimity of opinion at this time as to the precise balance that should be achieved between advantages and disadvantages of use of atomic energy, because certain

qualifying factors are still too poorly known. Uncertainties exist which can mean either underestimation or overestimation of the effect of radiation. This brief synopsis roughly appraises the biological costs of exposure to radiation and presents information which must largely guide decisions in the interim until more precise information on radiation effect is available.

A summary of current knowledge of radiation tolerance or hazard and fallout is provided in several major public documents that have appeared in 1956 in broad survey of the problems to man of atomic radiation and fallout.[3 4 5 6 7]

THE CONCEPT OF MAXIMUM PERMISSIBLE DOSE

Early estimates of that amount of exposure to ionizing radiation which constitutes a permissible occupational hazard placed the upper limit at 0.1 r. per day. Such a value was exceedingly conservative in view of information available at the time it was established. It is lower by a factor of 50 than chronic exposures leading to physiological disturbances and radiation sickness, and by a factor of 1,000 to 5,000 than the dose which, in a single exposure, might threaten life. Also, at the time it was proposed, 0.1 r. was the lower limit of radiation exposure dose known to elicit any biological response. Evidence on the magnitude of physiologic response of the individual to radiation in the range of a few hundred roentgens has not changed; but extensive information on effects of lower levels of radiation has recently appeared. This knowledge requires a reevaluation of the cost to humans of radiation exposure in terms of (*a*) genetic effects, (*b*) shortening of lifespan, (*c*) induction of cancers, (*d*) destruction of tissue, (*e*) congenital malformation, and (*f*) effects upon young individuals. All these effects appear to be proportional to the exposure to radiation, and have been largely responsible for a recent downward revision in maximum allowable exposure to radiation.

THE GENETIC EVALUATION OF RADIATION EFFECT

Up to 1946 estimations of the genetic effects of radiation had placed the quantity necessary to double the mutation rate per generation in the fruitfly at about 50 r. (Muller, Stern), but with some uncertainty, so that the true value might have been 80 r. or 35 r. At that time, there had been relatively little comprehensive evaluation of the range of genetic sensitivity to radiation in mammals or man. At the present time, the mutation rate per generation for the fruitfly is known to be doubled over the natural rate by about 50 r. (Stern). Through genetic study of irradiated mice (Russell), the amount required to double mutation rate per generation in the mouse is partially established also at approximately 35 to 80 r. Wright has estimated from evidence now available that the mammalian mutation rate may be doubled by as little as 3 r. or as much as 300 r. The best current estimates place the mammalian mutation-doubling dose of radiation at about 50 r. (4).

As an approximation, each species appears to form in natural circumstances about one new mutation in a generation time. The fruitfly lives a short time in about the same radiation environment (estimated roughly at 0.1 r. per year) as man. In its life span of 20 or 30 days, it can accumulate only the minute quantity of 0.008 r. Thus, if 50 r. in the fly produces an additional number of mutations equal to those which occur naturally, radiation can account for only a part of the natural mutation frequency, namely, the fraction

$$\frac{0.008}{50}=\frac{1}{6{,}000}.$$

Hence, at background radiation, only 1 observed mutant in 6,000 is suspect of being induced by radiation. In humans, the life span up to average reproduction age is about 30 years, lived in the same environment of 0.1 r. per year, or a total of about 3 r. by average reproduction age. Thus, if 50 r. is estimated to double the human mutation rate, radiation from natural sources may be expected to account for

$$\frac{3}{50}=\frac{1}{17},$$

---

[3] National Academy of Sciences, The Biological Effects of Atomic Radiation—Summary report, 1956.

[4] National Academy of Sciences, The Biological Effects of Atomic Radiation—Report to the Public, 1956.

[5] British report, Radiation Hazards to Man, Cmd 9780.

[6] Willard F. Libby, Current Research Findings on Radioactive Fallout, Proc. National Academy, December 1956.

[7] M. Eisenbud and J. H. Harley, Radioactive Fallout Through September 1955, Science 124, 3215 (Aug. 10, 1956).

or approximately 6 percent of the naturally occurring mutations. If we accept the lowest possible value of 3 r. for the mutation-doubling dose, we would have as the fraction attributable to radiation

$$\frac{3}{3}=1,$$

and radiation could account for the entirety of mutation changes in humans.

The fallout of radioactive materials through 1956 has increased the radiation exposure of gonadal tissue by an amount estimated as approximately 0.004 r. per year (see table V–D) (largely from ingested cesium–137 (4) and deposits on the ground (6)). This is an increase of approximately 3 percent over natural radiation exposure.

The recommended limits of radiation exposure in man will be affected by information on the quantitative relationship between ionization and mutation and the understanding of the natural mutation burden. Should we estimate the level of radiation likely to double the natural mutation frequency in man as 25 r. or 3 r., we will be at least 2 to 20 times as concerned about the genetic problems associated with radiation exposure as we are under the current assumption that the human mutation rate is doubled to 50 r.

Genetic studies of irradiated Japanese have been carried out by the Atomic Bomb Casualty Commission at Hiroshima and Nagasaki. A 10-year study has been analyzed by S. V. Neel and W. S. Schull.[8] The principal result is that no measurable increase in mutation rates was observed. They measured biological characteristics that could reflect genetic state and genetic change, such as stillbirths, male-female birth ratios, and congenital malformations. The results of all observations of this kind can be interpreted either as demonstrating no measurable increase in these events, which are associated with mutations, or as showing that, had the true congenital malformation rate been doubled, there would be only 90 percent probability of discovering even this increase. Thus a small increase in congenital evidence of genetic change would not have been detected.

The results of the study of the Japanese indicate that the human genetic effect of radiation is acceptably consistent with the range of response estimated from mammalian genetic experiments; and it establishes with certainty that there are no catastrophic genetic effects at low to medium range of radiation exposure in human beings, although catastrophic effects are predicted at high levels of accumulated radiation exposure to whole populations. Many new mutations were probably produced in the Japanese exposed to the atomic bombs; but many of these may have been unobserved because of early lethality, and the rest are overwhelmingly diluted by the vast number of normal genes. This dilution was expected; and the statistical odds are known to be very greatly against the appearance of unfavorable and detectable combinations of mutant genes in any one generation of offspring.

Genetic change is, of course, basic to the concept of the Darwin principle of evolution. For this reason, it is possible that some increase in the mutation rate might be to human advantage in the long run by providing a greater pool of variance from which selection could take place, to our final advantage some thousands of years from now. Some brief speculation regarding the extreme limits of variation may be offered:

(*a*) Humans and other long-lived animals have, as a corollary of their longevity, a less frequent natural mutation rate per gene per unit of time than short-lived species. As an approximate rule, each species appears to have about the same mutation rate per generation time. Thus, it appears that species are in some balance between generation time (or lifespan) and stability of genetic structure.

(*b*) Testing biological capacity for survival under circumstances that increase genetic variation is possible only with species having relatively short generation times. They have a common feature of a potentially great ratio of progeny per parent. These species can therefore survive even if a relatively high proportion of conceptions are incapable of survival and reproduction. Humans in natural selection are at some disadvantage in comparison with species producing a large number of offspring per generation, such as the mouse or the fly. Thus, human

[8] Reported at the First International Congress on Human Genetics, in Copenhagen in August 1956.

genetic tolerance should not be judged from effects of radiation exposures on these more fertile populations.

(*c*) For survival of a species, the ratio

$$\frac{\text{Reproducing offspring}}{\text{Individual}} = \text{must exceed 1.}$$

In humans, lowering of infectious disease toll has brought this ratio to approximately 2. As a consequence, the human population is now doubling in numbers approximately every 40 years. Thus, humans have already achieved some protective reserve against genetic changes toward lower fertility.

(*d*) On a scale of catastrophic genetic misfortune, humans also have the protection of vast numbers of individuals. There are 2.6 billion inhabitants of the world. While this number is small compared with numbers of insects and small mammals, it is still a very large number compared with that of any previous age in the history of man. Radiation exposure, as a cause of genetic change and increase of genetic variance, would be expected to produce that change in a random way. Thus, even with a large increase in genetic variance induced by radiation exposure, if population numbers are sufficiently great, some individuals will remain relatively unchanged. If these individuals were favored in selection, they might replace the less fit, less fertile fraction of the population. Thus, if survival of mankind were the only consideration, population numbers might, through reduction and segregation, achieve selective retention of adequately functional humans.

Several approaches to evaluation of extreme tolerance of human populations to radiation exposure with respect to health and genetic constitution are presented in appendix B. These methods of estimation are difficult and speculative; but they indicate that an additional 2 r./per year (or 50 r. per generation time) of chronic radiation exposure to the average individual in the human population would eventually cancel health and lifespan gains we have achieved recently. Such estimations of impairment of health and estimations of the cost of increasing the genetic variance suggest that human population cannot afford the biological cost of this intensity of chronic radiation exposure, and that there should be extreme caution at this time against increasing the radiation exposure to all people by 10 times over its natural level.

Evolutionary benefits?

It seems possible that human evolution is occurring in some optimal balance between mutation tendency and genetic stability. Fertility, length of life, death rate, and individual usefulness may be highly affected by the number of accumulated new genes,[9] which both add to favorable evolutionary drift in average human vigor and add to the pool of undesirable genes to be selected against. At low radiation levels, such as 10 percent or 1 percent above the natural radiation background (the range of fallout effect), it seems unlikely that long-range genetic disturbances can become an appreciable problem, since the natural radiation background appears to account for only 10 percent of the change in genetic structure per generation. One may speculate further that, in the long run, man may be beneficially affected by good genes yet to be formed, so that increasing radiation exposure and the mutation rate may operate to human advantage. Such an argument is unlikely to convince men who understand some of the dangers of too great a burden of undesirable mutants; it is analogous to an attempt to convince the experienced cook that the baking of her prize cake would be accomplished in half the time at higher oven temperature.

Penrose has evidence of indirect beneficial effect of some recessive lethal genes, which appear to enhance the effect of the functioning gene with which they are matched in individual combinations. This effect is one in which mutation may beneficially add some variance to genetic functional characteristics. On the whole, however, there is a strikingly large mass of information indicating that any genes that can disturb function should be kept to an absolute minimum.

Unfortunately, there are still many unanswered questions facing geneticists on the topic, "What is the effect of undesirable genetic burden on the quality of humans?" Fully satisfactory experimental measures have yet to be applied to this problem. One approach that has led to considerable speculation is through

---

[9] Transformed genes are, with rare exception, nonfunctional, lethal, or undesirable.

estimations of the numbers of undesirable mutations carried by the average person. Estimations of this burden place it within the small range of 5 to 15 undesirable genes per average individual[10] (4). This value is the equilibrium resulting from approximately one such gene gained and one lost in each generation.[11] Thus it has been pointed out that if, through increase in radiation exposure, the genetic gain of undesirable genes increased from 1 per generation to 2 per generation, there would be a relatively great reduction in the quality of the best 25 percent of individuals (assuming that reduction in quality of offspring is proportional to the number of undesirable mutations per individual). Because of speculative—but reasonable and cautious—arguments of this nature, geneticists have uniformly cautioned against allowing any major proportion of the population to accumulate radiation as high as 50 r., which is the amount estimated to double the human mutation rate.

LIFE-SHORTENING EFFECTS

Life-shortening effects of radiation have been observed under a variety of experimental conditions. An experiment of particular significance because of the large numbers of animals and the range of exposure was the exposure of mice to nuclear detonation at "Operation Greenhouse" (Furth et al.). The fraction of life span lost per unit of radiation exposure appears to be essentially the same for a number of species, including the mouse, the rat, the guinea pig, the rabbit, and man. The largest number of experimental observations concerns the mouse. In the mouse, the fraction of the life span lost per unit of whole-body radiation exposure is acceptably constant over a wide range of variation in radiation exposure. The tentative conclusion is that radiation effect simulates aging itself, and that a unit of radiation exposure, regardless of the intensity and duration of exposure, produces approximately the same relative disturbance to body structure in adults of all mammalian species. On the human life span scale, these effects of radiation summarized from small-animal data suggest that 1 r. of radiation exposure is equivalent to 5 to 15 days of physiologic aging. This prediction is confirmed directly in man (with reasonable technical reservations) by Dr. Shields Warren's recent investigation of life span of radiologists compared with physicians not using radiation in their practice of medicine. The average age at death is approximately 6 years less for radiologists than for physicians in general practice or for pathologists, both selected as being relatively unexposed to radiation. The estimation of accumulated radiation exposure in radiologists is uncertain, but has been approximated as 300 to 500 r. Thus, the life span loss, if attributed to radiation, is

$$\frac{-6 \text{ years} \times 365 \text{ days/year}}{300 \text{ r to } 500 \text{ r}} = -7 \text{ to } -4 \text{ days per r of whole-body exposure}$$

Such a number is still subject to considerable possible revision; but many different estimates give values of 1 to 30 days lost per roentgen of radiation exposure, and the probable value for humans is in the range of 5 to 10 days lost per roentgen.

A question exists whether we can justifiably extrapolate effects such as life-span lost per roentgen from measures that are mostly determined in the range of 100 to 1,000 r. The evidence is that, over the range that can be tested, the effect is linearly proportional to the radiation exposure; and the information fits an extrapolation to zero shortening of life span at zero artificial radiation exposure. There is additional evidence in the effects of radiation upon cells (as distinguished from entire organisms), in which lethal damage to cells per roentgen also appears to be proportional to total radiation exposure. Such estimates agree for cells in the mouse, the rat, the rabbit, the guinea pig, and man. This

---

[10] Some individuals may have none. The fraction having none or very few diminishes steeply with increasing average numbers of undesirable mutations. Thus, doubling the burden of mutations may reduce the numbers of individuals having desirable genetic combinations to rare events.

[11] The average mutation frequency of 1.5 spontaneous mutations of human genes per generation, as summarized by Penrose, corresponds to 30 mutations per million genes per generation, assuming that humans have about 50 ;000 genes :

$$\frac{30 \text{ mutations per generation} \times 50{,}000 \text{ genes per individual}}{1{,}000{,}000 \text{ genes}} = 1.5 \text{ mutations per individual}$$

per generation.

The average mutation rate may be less than this estimate, since one may suspect that the genes usually observed to mutate are perhaps 10 times as mutable as the average gene.

experimental evidence that effect of radiation on cells is in linear proportion to radiation exposure of from 15 to several thousand roentgens provides a reasonable basis for understanding the life-shortening effects of radiation.

Furthermore, the life-shortening effects are consistent in order of magnitude with the genetic effects of radiation upon cells (2 to 3 cells affected per 1,000 cells per roentgen). The genetic effect of radiation has been shown to be acceptably proportional to radiation exposure from 25 r to 8,000 r.

The sum of systematic evaluations of such effects of radiation as mutation induction, cell destruction, and life-span shortening indicates that these effects are permanent and represent the quantum interactions of radiation randomly affecting body cellular structure. The concept of quantum interactions with matter justifies extrapolation to the probability that a single quantum of radiation reacts with an individual molecule.

Although all recent evidence suggests that radiation effects is proportional to radiation exposure, such effects must be viewed together with other common environmental factors that modify health. A scheme is used here in which the effects upon health is expressed as an induction of aging (this is expressed as loss in physiologic lifetime, or minus time, written "$-n$ years") or as a postponement of aging (expressed conversely as lifetime gained, or plus time, "$+n$ years").[12] These factors all appear to have a general action upon disease tendency, and the effect is about the same at any adult age. The list of relative displacements of physiologic age (table I) is given for factors that accentuate aging or loss of life span (expressed as minus time) or retard aging (expressed as plus time). These measures are derived directly from human records. They are grouped according to whether they appear to be reversible or permanent. Most of the effects that are not partial measures of the same state are apparently additive, in the few instances that can be tested for this property. (See p. 1107.)

Certain of these circumstances that modify health are partially interrelated, others may be independent of one another. Estimates of effect upon physiologic age may be additive, depending upon the extent to which they are independent. Thus country against city dwelling may be suspected to include the factor estimated as exercise benefit. The lipoprotein test already contains information that can be estimated partially by relative overweightness, and the lipoprotein tests already accounts for a portion of the smoking effect. Familiar inheritance is independently estimated from each ancestor; male against female differences are equally added to city against country effects, and presumably each separate disease sign in the impairment study is additive.

In further support of the additive nature of effects upon health, each morbidity circumstance that can be quantitatively estimated produces an effect proportional to the intensity of the circumstance. Examples of proportional change in mortality risk with morbidity severity are:

(*a*) Overweight: —0.17 year for each percent overweight
(*b*) Smoking: —0.45 year per cigarette used per day
(*c*) Radiation: —5 to 10 —days per r. of whole body radiation
    3 cells killed per 1,000 cells per r. (marrow and lymphatic tissue)
    4 cells with chromosome breaks per 10,000 cells per r.
    1.4 percent increase in lukemia per r.
(*d*) Atherosclerosis, diabetes, nephritis: End effects are proportional to severity of metabolic error
(*e*) Accidents are proportional to exposure risks

A somewhat similar tabulation can be made of an estimation of the cost of industrial and transportation progress in this century in terms of years of life span lost by accident death, distributed to the average individual in the population of the United States (table II). These values are approximately comparable to the preceding values based upon changes in physiologic age. (See p. 1108.)

In about the same way, we can tabulate the effects on life span of radiation received (table III).

---

[12] This estimation of life span lost or gained is in terms of relative physiologic age change. Change in life expectancy may be estimated by determining life expectancy at a given age in terms of a given age$+n$ years' change in apparent age. Thus, a person of age 40 has a normal life expectancy of 31.1 years. If his physiologic age is 50 (because of a sum of factors predicting —10 years age over the average), his life expectancy (from life tables) is 22.8 years, or an average loss of life span of 8.3 years. Thus the life expectancy lost in somewhat less than physiologic time lost.

TABLE III.—*Estimation of radiation effect upon health and life span*

Radiation received	Life shortening (in years)	
	If l. r.=−5 days [1]	If l. r.=−10 days [1]
(r.)		
50	−0.7	−1.4
100	−1.4	−2.7
200	−2.7	−5.5
400	−5.5	−11.0

[1] 2 columns are given because of uncertainty whether l. r.=−5 days or −10 days.

Thus it is observed that, although the estimated effect of radiation upon life span is a number worth attention, its magnitude of effect at low accumulated dosage is slight compared with many public health problems. It must be remembered that major problems such as smoking and overweight and fat metabolism are so subtle that they are estimated and established not by clinical methods but rather by statistical (actuarial) researches involving large population samples. The effect of smoking 1 pack of cigarettes per day, for example, appears equivalent in reduction of health and life span to the effect of between 200 and 400 r. of accumulated whole-body radiation. This is several times as great as the 50-r. limit currently recommended for occupational exposure; and 50 r., in turn, is on the order of 10 times as much as the individual would accumulate through fallout. If the life-span loss is estimated as 5 to 10 days per r. of whole-body exposure, the loss due to 50 r. falls within the range of −0.7 year to −1.4 years of life span. This effect is greatly exceeded by the magnitude of the smoking problem; the obesity problem; the problems of atherosclerosis, diabetes, and all the chronic diseases; the benefits of marital status; etc. The effect of 50 r. of whole-body exposure to the general populace can also be viewed as being in the same category of life-span loss as that which results in the population of the United States from use of the automobile. This estimation, however, does not include the problem of the mutation burden in the next generations following such radiation exposure.

SUMMARY OF THE FALLOUT PROBLEMS ON A GLOBAL BASIS

On a global basis, the fallout intensity of radioactive materials is no more than one-millionth of the high-level fallout that occurred by mishap in the vicinity of a thermonuclear explosion in the Marshall Islands in Ooctober 1954. Current estimations made directly in humans throughout 1953–56 place the fallout exposure from strontium 90 as being, on the average; sufficient to produce an irradiation effect of approximately 0.004 r./year to human bones. This is a small quantity of radiation—2 percent of naturally occurring bone radiation—and estimates of effects derived from this additional tissue burden will be correspondingly small compared with other human problems.

At the present time, according to the Libby report (October 1956), there is in the stratosphere about 2.2 megacuries of Sr–90,[13] and a similar quantity of cesium 137.[14] If all the material in the stratosphere (in the fall of 1956) were to descend upon the surface of the earth uniformly, the amount of either Sr–90 or Cs–137 would be about 12 millicuries per square mile. The time of retention by the stratosphere of highly dispersed fission products is on the order of many years. Measurements indicate approximately 10 percent fallout per year and 2.5 percent radioactive decay. As about 25 percent has been added to the stratospheric reservoir of dispersed fission products during the past 2 years, the level in the stratosphere has remained nearly constant over that time. The quantity of Sr–90 in the soil of the United States is somewhat greater than expected from the fallout estimated on an average global basis; in the far west it is

[13] Strontium 90 has a half life of 25 years and decays by emission of a β particle of 0.54 Mev. maximum energy to produce yttrium 90. Yttrium 90 is short lived (half life 65 hours) it decays to the stable zirconium by emission of a β particle having a maximum energy of 2.24 Mev. Because of the short half life of the daughter product and the probable insoluble chemical form of yttrium, the radioactivity of strontium 90 is equivalent to both its own beta decay and that of yttrium 90.

[14] Cesium 137 has a half life of 33 years and decays by β emission (0.52 Mev. maximum energy) with associated γ emission (0.66 Mev. energy.

23 m C of Sr–90 per square mile. This is due to the heavier fallout in the near vicinity of a nuclear exposion.

Strontium 90 distribution to September 1955

	mC/square mile
Worldwide except the United States and Pacific Islands	3. 4[6]
United States, except Utah, Colorado, New Mexico, and bordering	4. 9[6]
regions	12. 5[5]
Utah, Colorado, and New Mexico	20–23

The specific ratio of Sr–90 to normal calcium is a convenient way of expressing the Sr–90 problem.[15] This is because strontium closely follows calcium in chemical behavior. The levels of Sr–90 directly measured in young human bones during the period up to October 1956 are in the vicinity of 0.0038 r/year to the bone. Strontium 90 is deposited preferentially in the bone by a factor of more than 100 over the soft tissues, so that only the bones need be considered with regard to this isotope.

The Libby report estimates, on the basis of a balance between accumulated fallout of Sr–90 into the soil and uptake by cattle and man, that in America the human ratio of Sr–90 to calcium may eventually become 10 to 30 percent of that observed in the topsoil. The report estimates that Sr–90 now held by the stratosphere, in descending to the earth over the next 4 years, will produce a human Sr–90 concentration of from 0.016 to 0.038 r./yr. (0.004 to 0.010 MPC[16]), assuming that no further Sr–90 is added. The range of this expected gain of radiation exposure is equivalent to the extra cosmic radiation exposure experienced by individuals dwelling at altitudes of 5,000 feet (e. g., Denver, Colo.) compared with individuals at sea level. The estimation assumes that there is a selection factor[17] favoring calcium over strontium in uptake from soils into the plant and into the cow and into the human bones, so that 70 to 90 percent of the soil strontium is rejected in favor of calcium.

Both human adults and stillborn babies have similar concentrations of Sr–90 (i. e., similar Sr–90/Ca ratios). This is to be expected, since the developing child draws its calcium from the maternal calcium pool, which is in partial equilibrium with maternal bone. Both these human sources of measured Sr–90/Ca have been placed during 1954 and 1955 at approximately one-sixth of the value for cow's milk; the resultant adult human bone irradiation value for this period is about 0.0019 r. yr. (0.0005 MPC) from the Sr–90 content. Reported values for adults

[15] A convenient concept, established by relating irradiation of bone to bone cancer, is that a maximum permissible concentration (1 MPC) of strontium 90 is equal to 1 μC Sr–90 per 1,000 grams of calcium. The concentration of calcium in the bones is such that 1 MPC can also be expressed as 1 μC Sr–90 per 7,000 grams of bone. The concentrations of radioactive strontium are usually expressed in units of 0.001 MPC; the equivalence is 0.001 MPC$=1.4\times10^{-7}$ μC Sr–90 g of bone, corresponding to 0.0038 r/year.

[16] MPC=maximum permissible concentration.

[17] Harrison et al. have evidence that elemental strontium-to-calcium ratios, compared in food, blood plasma, and bone, are strikingly different; for man they are:

	g Sr/g Ca	Proportional units
Food	17 $\times10^{-4}$	7
Plasma	4 $\times10^{-4}$	2
Bone	2. 5$\times10^{-4}$	1

This is confirmed by Comar in observations using radiostrontium and radiocalcium simultaneously added to the diet. In Comar's observations for milk, the discrimination achieved against strontium in the deposition ratio of Sr/Ca may be less than that for other food sources, in which strontium and calcium may have different chemical binding.

The problem of a protective discrimination for humans against the uptake of the maximum Sr–90/Ca ratio is presented in the Libby report. At this stage of understanding, this apparent reduction of Sr/Ca in bones of humans compared with soil, plants, or animals seems to reside partly in the large calcium pool of the adult cow's body, which constantly dilutes incoming strontium and calcium so that milk, at present, is always intermediate in Sr/Ca ratio between the cow's bones and the forage. Similarly, the human calcium pool dilutes incoming Sr/Ca (largely from milk products) so that human bones at this time always have a lower Sr–90/Ca ratio than cow's milk or cow or calf bones. The content of children's bones is much higher than in adult or stillbirth material. There is some evidence for atomic discrimination between strontium and calcium, but the problem needs further study to determine how much of Sr–90 uptake by bone is lessened at fallout equilibrium. If only dilution operates, with little or no discrimination, humans will develop a higher Sr–90 level than is now expected.

did not exceed 0.004 r. yr. in the sample studied, except for one individual measured at 0.008 r. yr. This is a very small number in terms of radiation effect.

If, in the fallout to be expected, the discrimination against Sr–90 in its course from soil to plant to human bone is by only a factor of 50 percent instead of a factor of 70 percent to 90 percent, Libby's estimate of the future Sr–90 concentration would have to be increased to 0.075 r. yr. (0.020 MPC), based on the present stratospheric and soil burdens. This level of Sr–90 would represent an additional radiation exposure to the bone, equivalent to the additional cosmic radiation experienced by those who dwell at 10,000 feet in this latitude.

Libby has estimated, from soil calcium levels, that if the entire Sr–90 burden reached the soil and humans came into equilibrium with the top 2 inches of average soil, humans would eventually approach a maximum value of 40 $\mu\mu$C Sr–90/g Ca, or about 0.15 r. yr. of bone irradiation. Such a value would approximately double bone irradiation over natural radiation.

ESTIMATION FROM HUMAN BONE ASSAYS OF FUTURE HUMAN BONE CONCENTRATIONS OF STRONTIUM 90

The uptake of Sr–90 has been directly measured in human bones as a function of age, and of location and time of collection (Libby, (5) Kulp et al.). The following summary conclusions can be drawn from analysis of this information:

1. Strontium 90 content of the bones in human stillbirths is increasing and, on the average, is estimated from Libby as follows:

United States of America	$\mu\mu$C Sr-90/g. Ca	Percent increase per year
December:		
1953	0.14	
1954	.30	114
1955	.66	120
1956	[1](1.3)	(100)

[1] Extrapolated.

2. The bones of stillborn humans have a much lower Sr–90 content than those of year-old children. The Sr–90 content of children's bones, which may be averaged from the Libby report, is given in table IV. This table is representative of the Sr–90 concentration observed in children of early ages at two study intervals, namely September 15, 1954, and August 1, 1955, average collection dates. Newborns (stillbirths) have a much lower Sr–90 concentration, because the uterine source of Sr–90/Ca has some intermediate value between dietary Sr–90/Ca and adult tissue-bone Sr–90/Ca. The value for stillbirths, as of January 1955, is 0.31 $\mu\mu$C Sr–90/g Ca; at this same time, growing children, age 0 to 5 years, are laying down Sr–90 at 2 $\mu\mu$C Sr–90/g Ca. Thus, the fetal tissues appear to have available to them only 0.31/2=0.16 as much Sr–90 as the growing child. This is a reasonable fraction, considering the lesser relative amount of milk products consumed by the average mother and the fact that her tissue stores of calcium are largely from the prefallout era. The growing child at each interval of growth (i. e., 0 to 1 year, 1 to 2 years, etc.) dilutes the entering Sr–90/Ca by the existing quantity of Sr–90/Ca already present in the body. However, analysis of the increment increase in Sr–90 content shows that children of all ages are consuming and laying down equivalent concentrations of Sr–90/Ca, and that in January 1955, this concentration was approximately 2 $\mu\mu$C Sr–90/g Ca.

On this date, three sources of milk showed the following ratios:

Radiostrontium content of milk samples, January 1955

	$\mu\mu$C Sr–90/g Ca
Foreign cheese (5)	2.0
Chicago milk (5)	1.9
New York milk (7)	1.6

Since growing children have milk as their chief source of Sr–90, it is as expected that the value of milk closely approximates the concentration of Sr–90/Ca being deposited in growing bones. These values imply that, should milk remain as it was in January 1955, all children born close to this date

will eventually have in their bones an average concentration of Sr–90 of 2 μμC Sr–90/g Ca.

However, the milk Sr–90/Ca is increasing, and has been increasing since monitoring of milk was begun in 1953. Eisenbud's report[18] gives the following.

TABLE IV.—*Strontium 90 content of cildren's bones (from Libby report)*

Age	Weight		Sr-90 content		Sr-90 content in newly formed bone [1] (corresponding to January 1955)
	Average at measurement	Δ/yr.	Aug. 1, 1954, to Nov. 1, 1954	June 1, 1955, to Oct. 1, 1955	
	kg	*kg*	*μμC Sr-90/g Ca*	*μμC Sr-90/g Ca*	*μμC Sr-90/g Ca*
Birth	3.3		0.25	0.53	
1 year	7.2	3.9	.54	1.16	2.2
2 years	9.6	2.4	.43	.87	2.1
3 years	11.5	1.9	.39	.68	2.2
4 years	13.4	1.9	.35	.54	1.7
5 years	15.1	1.7	.33	.44	1.3
Average					1.9

[1] See the following:

$$\frac{\Delta\ \mu\mu C\ Sr^{90}}{\Delta\ g\ Ca}=\left[\frac{Sr^{90}/Ca_{(t_2)}\ x\ wt_{(t_2)}-Sr^{90}/Ca_{(t_1)}\ x\ wt_{(t_1)}}{\Delta\ wt_{(t_2-t_1)}}\right]\times\frac{12.0.}{10.5}$$

(The 12.0/10.5 is the correction factor for 10.5-month time interval Sept. 15, 1954, to Aug. 1, 1955.)

Sr–90/Ca content of milk in the New York area

Date:	*μμC Sr–90/g Ca*
June 1954	1.2
January 1955	1.6
June 1955	2.0
January 1956	2.7
September 1956	5.0

The minimum estimate of future average human burden of Sr–90, then, is that 5 μμC Sr–90/g Ca will be present in the bone. This corresponds to the latest reported value for milk concentration and to the fact that bone acquisition of Sr–90/Ca in growing children is very similar to milk Sr–90/Ca.

A difficult current problem is the estimation of future Sr–90/Ca in milk. The level of Sr–90/Ca in milk is increasing, and, by linear extrapolation, may be expected to raise the Sr–90 concentration in a year's time (by September 1957) to about 7 μμC Sr–90/g Ca. At this date, accumulated fallout of Sr–90, based upon the quantity estimated at the time of the Libby report, may be about 25 percent[6] to 50 percent[5] of the amount initially dispersed in the atmosphere. Since the Libby report was written, other nuclear detonations have occurred, so that it would be very reasonable to assume that fallout; by some 10 years from now, should have increased milk levels significantly. For lack of better information, we may assume a factor of, say, 3 to 5 times as much as September 1957 (allowing for residual hold-up in the atmosphere and for decay of Sr–90). Thus, milk levels and human bone levels by 1967 may be 20 to 35 μμC Sr–90/g Ca.

An additional factor must be considered, which may require that these future estimates be even higher. Cows, in body content of Sr–90/Ca, may be expected to lag several years behind the plant and soil levels. This is because of the large calcium reservoir in their bones and other tissues, and bcause the start of growth to milk-producing stage preceded current time by 4 or more years; moreover, the food consumed by dairy cows is customarily stored for many months before it is eaten. It is difficult to estimate that point in fallout time that corresponds to current milk values; it seems likely, however, that the Sr–90/Ca content of the bones of pasture-fed calves approximates the Sr–90/Ca level that adult cows

[18] Merril Eisenbud, Global Distribution orf Radioactivity From Nuclear Detonations With Special Reference to Strontium 90, Washington Academy of Sciences, fall symposium, November 15, 1956, Washington, D. C.

would secrete in their milk, were they in more rapid equilibrium with fallout. The following table, derived from values averaged from Libby's and Eisenbud's reports, shows that calf bones are approximately 60 percent higher in Sr–90/Ca content than milk. Thus, future estimations of Sr–90 levels should be at least 60 percent higher than the 20 to 35 $\mu\mu$C Sr–90/g Ca estimate, or, say, 30 to 50 $\mu\mu$C/g, in round numbers.

Strontium 90 content of various materials (in $\mu\mu C/g$ Ca)

	Mid-1953	Mid-1955	Mid-1956
Milk	1.1	2.1	3.6
Calf bones	1.4	3.5	5.7
Alfalfa (Wisconsin)	6.7	18.0	----
Soil (Wisconsin):			
2 to 6 inches depth	----	9.0	----
0 to 2 inches depth	----	35.0	----

It appears that the Sr–90/Ca of cow's milk is a close index of the concentration of strontium in newly acquired human bone. Current milk levels suggest that children's bones in the next decade will approach an average concentration of approximately 50 $\mu\mu$C Sr–90/g Ca. This is in close agreement with the estimation by Libby of a minimum average concentration of Sr–90 of 10 to 40 $\mu\mu$C Sr–90/g Ca. These estimates do not consider local variance in the United States, nor, with respect to future concentrations, the special problem of high-rainfall or low-calcium areas.

The upper value of approximately 40 $\mu\mu$C Sr–90/g Ca has been set by Libby upon the consideration that this is the projected specific concentration ratio when all the fallout is complete and mixed with the average calcium content of 2 inches to topsoil. There does not seem to be a way of independently confirming the upper average limit of radiostrontium concentration from observation of milk or bone. The biological concentrations are increasing rapidly with respect to time, approximately following the level of total accumulated fallout, and 40 $\mu\mu$C Sr–90/g Ca may not truly be a limit.

Whatever the speculation concerning future levels of Sr–90 in humans, we can be certain that current values (1956) represent a low level. If we translate a small dose such as 0.0038 r./year (0.001 MPC) into numbers predicting an increase in leukemia mortality (an estimate may be based upon tentative data that leukemia tendency may be doubled by 50 r. whole-body exposure [19]), an increment of

$$\frac{0.0038 \text{ r./yr.} \times 50 \text{ years mean life span}}{50 \text{ r./tumor doubling}} = 0.004,$$

or 0.4 percent increase in leukemia, is estimated. Since there are only approximately 8,000 cases of leukemia deaths reported in the population of the United States per year (plus 2,000 cases of bone-tumor deaths, which may be similarly affected by radiation), such a radiation burden is equivalent to an increase of 40 cases per year after 50 years' equilibration with this level of fallout. If radiation fallout and uptake of Sr–90 in human bones were to increase by a factor of 10, one could estimate 400 additional cases of bone tumor and leukemia induced per year after a 50-year period, in comparison with 1 million deaths from all causes and 10,000 expected deaths from leukemia and bone tumors. Both above numbers are small in comparison with overall public health problems.

Although there are some sizable uncertainties regarding Sr–90 burdens during the next 10 to 20 years, it seems from the average human values that Sr–90 may increase and become a public health problem if levels should rise to 50 $\mu\mu$ CSr–90/g Ca (equal to about 0.2 r./yr. to bone). There is time—but not much time—for a reevaluation of many unsatisfactorily estimated aspects of this problem, including the extent to which radiation exposure induces leukemia and bone tumors, and more precise estimation of the strontium levels in humans.

[19] This number may be high, since it is based upon whole-body radiation exposure, while induction of leukemia by Sr–90 exposure is the result of direct irradiation of bone and marrow, the specific tissues involved in the leukemia change.

At the reference level of 1 MPC of Sr–90 burden, which is 4 r./yr. to bone, an estimated increase in bone tumors and leukemia is

$$\frac{4 \text{ r./yr.} \times 50 \text{ years}}{50 \text{ r. per doubling of incidence of tumors}},$$

or an approximately fourfold increase in natural expectancy of these neoplasms with respect to the radiation-related component of their origin. This level may be reached by humans as a result of Sr–90 fallout. At some such value, reason argues against further exposure. The 1–MPC value based on radium exposure is consistent with a prediction of a fourfold increase in natural incidence of tumors. It would be difficult to observe a fourfold increase above natural incidence of bone tumors in animal-colony studies with radium, but not at all difficult in large human populations.

In summarizing their opinion for the British Report Cmd 9780, Mayneord and Mitchell write, "It appears however that each unit quantity of radiostrontium absorbed by bone confers a certain probability of bone-tumor formation, the tumor development time perhaps decreasing and the tumor incidence increasing with the dose. On the whole, the experiments seem in favor of a proportionality between the frequency of tumors produced in a given length of time and the amount of radioactive material in the body even at low-dose levels."

The problem in the experimental animal is that the frequency of bone tumor appearance is so slight that statistically significant increases in the frequency are not to be expected as a result of irradiation. The human problem is similar in that osteogenic sarcoma and leukemia are relatively unlikely occurrences, together causing about 1 percent of adult deaths in the United States, so that a small percentage change in incidence caused by radiation could not be distinguished from random fluctuation, and a relatively large fractional increase in the number of these cancers would not appreciably increase the total death rate.

No gross evidence of osteogenic sarcomas has been observed following administration of P–32 (approximately 100 rep to bones) to polycythemia vera patients. However, these patients do have a high incidence of leukemia. This leukemia tendency is probably attributable to both the radiation exposure and the nature of the basic disease of the blood-forming system in these patients.

Special phases of the Sr–90 problem need additional examination:

(*a*) In several areas of the world, Sr–90 concentration exceeds the average world values by more than a factor of 10 (4, 5). This excess poses questions as to the origin of the enhanced concentration. To a reasonable extent, it is explained by Libby as calcium deficiency of soils in such areas. Rainfall variation also leads to variation in fallout. It will be useful to know more about these anomalous effects. Current worldwide sampling is perhaps far from representative of the world as a whole, because special effort was made to seek out low-calcium high-rainfall areas.

(*b*) There may be a factor-of-8 difference between Sr–90/Ca concentrations in soil and in humans, resulting from discrimination in favor of calcium (Libby); this must be further studied.

(*c*) Some factor of uncertainty must be allowed for in the prediction of levels today and in the early future of Sr–90 in humans, considering that the most recent of these measures are based on early 1956. These uncertainties may amount to a factor of somewhat more than 10.

(*d*) Although it is unlikely that all these factors would reach their maximum, nevertheless, the total uncertainty in the estimated human burden of Sr–90 throughout the world could mean an upper limit of $10 \times 8 \times 10 \times$ Libby's lower estimate of exposure in the near future, 0.02 r./year, which works out to about 15 r./year or 4 MPC. This possibility indicates that the Sr–90 fallout problem urgently calls for further attention.

CESIUM–137 FALLOUT

The Cs–137 problem is quantitatively similar to that of Sr–90. These two fission products are present in the air and in fallout in approximately equivalent quantities (5), and they have similar decay rates. Whereas strontium is a bone seeker, cesium is found in approximately equal quantities throughout the body, though less in bone than in soft tissues. Its distribution roughly approximates that of potassium. Furthermore, cesium is not retained by the body. Thus, the cesium burden at any given time rapidly reaches equilibrium with the rate of fallout, in the potassium pool in plants and animals.

Marley, in the British report (4), writes (p. 124), "The highest body-activity detected so far in the United States is found to be $4\times10^{-8}\mu C$. This activity if maintained would produce a total body irradiation of 0.0006 r per year or about one-thirtieth of the dose due to naturally occurring potassium–40 in the body." Since this time in early 1956, the fallout level and fallout rate of Sr–90 have been increased only slightly, so that we may assume that the Cs–137 level in man, which is more reflective of immediate fallout, may have risen by as much as a factor of 2. It should remain nearly at this level, estimated as a maximum of 0.0012 r/year, for an indefinite period.

Cesium–137 body burden at 0.001 r/year is certainly not to be considered an adult hazard. With a linear relation between effect and dosage, 0.001 r/year over a lifetime would be less than 0.1 r, and irreversible accumulative effects of radiation, such as leukemia, might be increased by less than

$$\frac{0.1\text{ r.}}{50\text{ r. leukemia-doubling dose}}=0.002\text{, or }0.2\text{ percent.}$$

Stated in terms of life span lost or of the total tendency toward disease, 0.1 r Cs–137 dose $\times$—10 days of life span per r amounts to 1 day lost from the life span. A loss of 1 day is very small compared with health-modifying factors that are measured in years instead of days. Thus, in comparison with the smoking problem, the long-term effect of Cs–137 is approximately one-forty thousandth as deleterious. Only this extraordinary method of estimation by extrapolation of effect can convince the human reason that there is any such effect at all; even the best statistical procedures could not detect it through study of the most accurate data on the 160 million people in the United States. A 0.2 percent increase in leukemia (which is approximately $0.002\times8{,}000$ cases per year) is just 16 additional cases. This 8,000 expected normal incidence can fluctuate by random interplay of chance factors by plus or minus 1 percent, equal to 80 cases per year; thus, 16 cases of increased incidence cannot be detected.

THE LEVEL OF RADIATION EXPOSURE FROM FALLOUT

The total increase in background radiation on a global basis, as a consequence of radioactive fallout, has been very slight. In the preatomic age, natural sources of radiation produced an average radiation exposure of 0.1 to 0.2 r/year. The variation is due to slight geographic differences, to differing radioactive content of earth and buildings, and to the variation of cosmic radiation with altitude. At 5,000 feet above sea level, cosmic-ray intensity (measured by numbers of ionizations produced in matter) is increased to 1.5 times the sea-level intensity of cosmic rays; at 10,000 feet, the cosmic-ray ionization is 3 times that at sea level.

The increased human-tissue irradiation due to fallout and ingestion of radioisotopes is approximately as follows:

	Soft tissue irradiation (r/year)	Bone irradiation (r/year)
1955–56:		
Cs–137	0.0009	<0.0004
Sr–90	0	[1] 0.002 [2] 0.004
Predicted future values:		
Cs–137	0.0012	<0.0006
Sr–90	0	0.04 to 1.5

[1] Adult.
[2] Young.

Table V lists human radiation exposures from a number of sources.

TABLE V.—*Human radiation exposure (r./year)*

A. EXTERNAL EXPOSURE TO WHOLE BODY

Natural radiation exposure		Exposure from fallout from nuclear detonations	
1. Cosmic radiation at—		1. Fallout to earth, October 1952 to September 1956:	
Sea level	0.038	Salt Lake City	0.050
2,000 feet	.043	All other United States cities and other countries	.003
5,000 feet	.056		
10,000 feet	.112		
15,000 feet	.214		
2. Radiation from the earth:			
England	0.04		
Berkeley hills	.12		
Sweden	.09–0.16		
Average	.10		

B. INTERNAL EXPOSURE

	To gonads and soft tissue	To bone		To gonads and soft tissue	To bone (1955)
Potassium 40	0.020	0.005	Strontium 90	[1]	[2] 0.002 [3] .004
Carbon 14	.001	.001			
Hydrogen 3	[1]	[1]	Cesium 137	0.001	[1]
Radium	.002	.120	Iodine 131	[1]	[1]
Average	.023	.126	Average	.001	[2] .002 [3] .004

C. TOTAL EXPOSURE

Prefallout:			Now:		
At sea level	0.164	0.264	At sea level	0.165	0.269
At 5,000 feet (Denver)	.179	.282	At 5,000 feet (Denver).	.183	.287

D. INTERNAL EXPOSURE UNDER OTHER CONDITIONS

1955:			Predicted for individuals born now (if no additional nuclear detonations):		
Strontium 90:					
Average value, USA	------------	0.0019	Minimum average value predicted by Libby—0.004 MPC	------------	0.016
Highest value reported	------------	.0075	Maximum average value predicted by Libby for United States—0.010 MPC	------------	.038
Certain low-calcium areas	------------	------------	Certain low-calcium areas	------------	0.16–0.38
If humans at equilibrium should approach the Sr/Ca ratio of plants rather than 10 to 30 percent of plant Sr/Ca ratio	------------	------------	If humans at equilibrium should approach the Sr/Ca ratio of plants rather than 10 to 30 percent of plant Sr/Ca ratio	------------	0.10–1.5
Iodine 131:			1956:		
Thyroid	[4] 0.004	------------	Iodine 131	<0.001	------------
Thyroid (maximum measured by Van Middlesworth)	[4] 0.004	------------	Probably	.0002	------------
Other than thyroid (estimated)	<0.000001	------------	Estimated	< .000001	------------

[1] Too small to be considered in this tabulation.
[2] Adult.
[3] Young.
[4] Possibly the true value is 0.001 or less.

The problems of radioactive fallout may also be examined in comparision with other ways of acquiring exposures to radiation (English values for radiological exposure are generally much less than in America (4)). (See table VI.)

Thus, it is possible that, from common use of X-ray-generating devices, the average person in the United States has already begun to accumulate an exposure to radiation effect that is sizable compared with the fallout problem. That no gross evidence of disease has become evident during these past few years of increasing radiation exposure does not disprove the existence of slight average effects of radiation. For example, at current estimation of leukemia induction by radiation, about 20 percent of the relatively rare cases of leukemia (0.5 percent of adult fatalities) may be attributable to natural radiation. There is no difficulty in believing that supplementary radiation resulting from our modern activities may have been responsible for the other 80 percent of known cases of leukemia; the average additional artificial radiation exposure per year would only have had to be 0.8 r. to account for this difference. Considering the generous use of unshielded and unfiltered X-ray equipment in dental offices and shoestores alone, and the lack of public and professional appreciation of need to minimize radiation exposure, it is even reasonable to conjecture that the addition of artificially created radiation exposure to natural irradiation may essentially account for leukemia. Faber has analyzed 828 cases of leukemia registered in Denmark in the period 1950–53 with regard to the amount and type of irradiation each patient received for 20 years prior to development of leukemia. The incidence of previous incidental X-ray or radiation exposure for the chronic lymphatic leukemia cases was 18 percent, for myeloid leukemia, 30 percent, and for acute leukemia, 32 percent. It appears that both acute leukemia and myeloid leukemia can be induced by radiation; and the traceable X-radiation induction may account for a sizable percentage of current cases in Denmark. Faber's information does not rule out that lymphatic leukemia may also be induced by radiation. The analysis of leukemia incidence in followup of three groups of individuals who had had varying exposures to X-rays or other radiation strongly suggests that the radiation induction of leukemia is proportional to the radiation exposure, and that for whole-body radiation exposure the number would be entirely consistent with an estimation that 50 r. doubles the chance of development of leukemia.[20]

TABLE VI.—*Common means of exposure to radium*

Source	Exposure	
	Directed to the specific body region	Scattered to the whole body (dose per use)
Routine chest X-ray [1]	0.05 to 2 r./exposure	
Fluoroscopic examination [2]	10 to 20 r./min	1/200 to 1/1000 of local dose.
Cinefluorography [2]	25 r. per examination	
Dental X-ray [1]	10 to 150 r. per whole-mouth series.	0.01 to 1 r.
Shoestore fluoroscopy [1 2] shoe-fitting unit	50 to 150 r./min. to feet	1 to 10 r./min.
Radium-dial watch [1] μ C/watch	7. r/yr. to the wrist	0.01 r./year.
Radium and X-ray [1 2 3] technicians (throughout the world).		p.1 to p.3 r./week, 5 to 15 r./yr.
AEC maximum permissible dose for 20 years' exposure.		15 r./yr. at 0.3 r./week.
Average accumulated exposure of 10 most highly exposed individuals over 5-year period—U. C. Radiation Laboratory.[4]		0.1 r./week, 5 r./yr.

[1] William Nolan.
[2] AEC Report to Congress.
[3] Jones.
[4] University of California Radiation Laboratory records.

RADIOIODINE FALLOUT

Of all the problems that we can currently evaluate, the radioiodine fallout problem is disposed of most readily. Radioiodine is produced in thousands of curies by some of the nuclear detonations, and, in falling to the earth's surface, it contaminates grass and is eaten by foraging animals. In its fallout, it is

[20] Court-Brown and Doll, Summary of Leukemia Induction, British Report (4), pp. 84–89.

greatly diluted and does not at any time become a human problem. The herbivorous animal, however, eats large quantities of grass; and in the cow, for example, essentially all the iodine 131 ingested accumulates in the thyroid gland. Over a few days' time, several hundred pounds of grass may be eaten, and all the iodine contained becomes concentrated in the 15 to 30 grams of thyroid tissue. Following nuclear detonations of the last 2 years, the thyroid concentrations of radioactive iodine in pastured cattle reached as high as 0.001 to 0.003 mμC/g (depending upon the quantity of fallout), and the average radiation exposure, as measured over 3 years, was about 1 r./year to the thyroid tissue. This would be of genuine concern to man at similar human burdens of I–131, because it is now known that thyroid tissue is especially sensitive to radiation induction of tumors. However, cattle fed principally in feed lots have only 1/100 (or less) as much I–131 as range-fed cattle. Further careful measurement of fresh human thyroid material has been routinely made during the last 2 years by techniques that are sensitive and reliable for estimation of I–131 content. Direct measurement shows that human thyroid, at any time of high uptake of I–131 by bovine thyroid, has less than 1/5000 of the bovine I–131 content. It is possible that human thyroids had less than 0.0006 mμC/g during the latter part of 1956, when range cattle had 1 to 2 mμC/g. It is certain that the human thyroid exposure during the 1956 period did not exceed 0.001 r./year, and the probable value is 0.00016 r./year or even less. (Interestingly, one human thyroid showed an activity comparable with bovine thyroid content of I–131; the case, when traced to its source, proved to be from a man who had previously been given a small tracer dose of I–131 in the Donner Laboratory. The observed quantity of I–131 was accounted for by the magnitude of the dose, the estimated excretion, and the radioactive decay.)

Up to this time, radioiodine from worldwide fallout is not a problem of concern to humans; and it is not expected that it will become a problem in the future.

SUMMARY

1. This paper reports a broad examination of the levels of radiation exposure incurred from fallout. The discussion is limited to Sr–90, Cs–137, and I–131, the only radioactive isotopes reported to become associated with human environmen in detectable quantities.

2. The worldwide effect of radiation from fallout is now far less than that of naturally occurring radiation from cosmic rays and from radioactive elements normally contained in earth, buildings, and body tissue. The inescapable minimum of natural radiation exposure, for all people, is about 0.1 r./year. The average person at sea level in the United States is probably receiving about 0.16 r./year.

3. During 1954–55 the Sr–90 concentration in human bones (both in adults and in stillborn infants) produced an average exposure to the bones themselves of 0.002 r./year. (Only the bones—not the soft tissues—are exposed to measurable levels of Sr–90 irradiation.) At current fallout trends, the irradiation of human bone by Sr–90 will increase to 0.016 r./year, perhaps even to 0.038 r./year (Libby). The maximum value projected in this discussion is 0.2 r./year. (These are average predictions for the northern hemisphere and for the major population densities of the earth.)

4. Radioiodine (I–131) activity has been measured in humans during periods of likely fallout exposures. Radiation exposure from fallout I–131 is essentially nil for humans.

5. Any analysis of the fallout of radioactive materials on a worldwide basis shows that it does not even remotely approach the threshold for acute radiation effects, which cannot be recognized below 100 r. in a single exposure. Radiation predicted from future fallout is still far less than natural radiation background. Increases in the internal radiation exposure of 0.1 r./year are not meaningful in comparison with acute radiation damage. Attempted comparisons are responsible for most misunderstanding of the fallout hazard to humans.

6. Life-span changes, cancer or leukemia induction, and cell changes appear to be proportional—as are genetic effects of radiation—to radiation exposure. Although these effects are not measurable in any individual exposed to fallout, they can be estimated, in terms of very small risks. The effects are dwarfed

in comparison with the adverse environmental hygienic factors that most persons regard as commonplace. For example:

Factor:	*Life-span loss per person (days)*
Smoking one pack cigarettes per day	3,000
Being 25 percent overweight	1,300
Having 25 percent elevated lipoproteins	2,500
Living in United States as a driver of an automobile	470
Working in industry (industrial hazard)	100

7. The evidence indicates that Sr–90 may eventually cause a worldwide increase in leukemia, accounting for about 2 percent of all deaths. Compared with the current accident rate, a 2 percent leukemia increase distributed throughout the entire population would be a life-span loss of about 1.0 year per person in the United States; all accidents account for a 2.3-year life-span loss per person, automobile use for 0.87 year. Thus the Sr–90 induction of leukemia is comparable with some of the mechanical mishaps we risk as a partial cost of the "advantages" of our mechanized and energized age.

8. The sum of evidence is that radiation has a deleterious effect upon man's health, but that the effects are extremely small at such slight radiation exposures as are involved in the worldwide fallout. Nevertheless, since radiation probably does affect man's health and progeny—even though minutely for minute exposures—incurring it should be treated as the equivalent of the spending of money or time, and should be allowed only for necessary gainful advantages.

BIBLIOGRAPHY

Fallout

1. E. P. Cronkite, V. P. Bond, and C. L. Dunham, Some Effects of Radiation on Human Beings, A Report on the Marshallese and Americans Accidentally Exposed to Radiation from Fallout and a Discussion of Radiation Injury in the Human Being (United States Atomic Energy Commission, July 1956).
2. National Academy of Science, The Biological Effects of Atomic Radiation—Report to the Public (Washington, D. C., 1956).
3. National Academy of Science, The Biological Effects of Atomic Radiation—Summary Report (Washington, D. C., 1956).
4. British Report, Cmd 9780, Radiation Hazards to Man (Her Majesty's Stationery Office, 1956).
5. Willard F. Libby, Current Research Findings on Radioactive Fallout, Proc. Natl. Acad., Dec. 1956.
6. Merril Eisenbud and J. H. Harley, Radioactive Fallout Through September 1955, Science 124, 3215 (1956).
7. Merril Eisenbud, Global Distribution of Radioactivity from Nuclear Detonations with Special Reference to Strontium–90, Washington Academy of Sciences Fall Symposium (Atomic Energy Commission, Washington, D. C., Nov. 15, 1956).

Kulp, Eckelmann, and Schubert, Strontium–90 in Man, Science 125, 219 (1957).

R. J. Bryant, A. C. Shamberlain, A. Morgan, and G. S. Spicer, Radiostrontium Fallout in Biological Materials in Britain, Atomic Energy Research Establishment, Harwell, England AERE-HP/R–2056, September 1956.

Blood-forming system

T. G. Hennessy and R. L. Huff, Depression of Tracer Ion Uptake Curve in the Rat Erythrocytes Following Total-Body X-Irradiation, Proc. Soc. Exptl. Biol. Med. 73, 436 (1950).

Hardin B. Jones, Some Physiological Factors Related to the Effect of Radiation in Mammals, in Symposium on Radiobiology, James J. Nickson, Ed. (Wiley, New York, 1952).

Lola S. Kelly, Effect of Radiation on DNA Synthesis in Mammalian Cells, in Progress in Biophysics, J. A. P. Butler and J. T. Randall, Eds. (Pergamon Press, London, 1957).

L. O. Jacobson and E. K. Marks, Plutonium Project: Hematological Effects of Ionizing Radiations in Tolerance Range, Radiology 49, 286 (1947).

O. A. Trowell, The Sensitivity of Lymphocytes in Ionizing Radiation, J. Path. Bact. 64, 688 (1952).

Harvey M. Patt, Factors in the Radiosensitivity of Mammalian Cells, in Ionizing Radiation and the Cell, Ann. N. Y. Acad. Sci. 59, Art 4, p. 649 (1955).

John H. Lawrence, The Treatment of Chronic Leukemia, Med. Clin. N. Amer. 38, 525 (1954).

Henry Kaplan, On the Etiology and Pathogenesis of the Leukemias: A Review, Cancer Res. 14, 535 (1954).

Genetics

W. P. Spencer and Curt Stern, Experiments to Test the Validity of the Linear r-dose/mutation Frequency in Drosophila at Low Dosage, Genetics 33, 43 (1948).

E. Caspari and Curt Stern, The Influence of Chronic Irradiation with Gamma Rays at Low Dosage on the Mutation Rate of Drosophila, Genetics 33, 95 (1948).

D. Uphoff and Curt Stern, The Genetic Effects of Low-Intensity Irradiation, Science 109, 106 (1949).

Liane B. Russell and W. L. Russell, An Analysis of the Changing Radiation Response of the Developing Mouse Embryo, in Symposium on Effects of Radiation and other Deleterious Agents on Embryonic Development (Wistar Inst., Philadelphia, 1954), p. 103.

S. V. Neel and W. S. Schull, Effects of Exposure to the Atomic Bombs on Pregnancy Termination in Hiroshima and Nagasaki (National Academy of Science, National Health Institute, Washington, D. C., 1956.) (An Analysis of Genetic Studies Carried Out During 1946–1955 by the Atomic Bomb Casualty Commission at Hiroshima and Nagasaki, reported at 1st International Congress on Human Genetics, Copenhagen, Denmark, Aug. 1956.)

L. S. Penrose, Evidence of Heterosis in Man, Proc. Roy. Soc. (London) 144, 203 (1955).

J. Gordon Carlson, An Analysis of X-ray-Induced Single Breaks in Neuroblast Chromosomes in the Grasshopper, Proc. Natl. Acad. Sci. U. S. 27, 42 (1941).

Life-span-shortening effects

A. Radiation effects:

R. D. Boche, "On Permissible Exposure to Radiation," Address at Symposium on Low-Level Irradiation, Argonne Natl. Lab., Chicago, Oct. 19, 1948.

Henry A. Blair, Biological Effects of External Radiation (McGraw-Hill, New York. 1954).

Austin M. Brues and George A. Sacher, Analysis of Mammalian Radiation Injury and Lethality, in Symposium on Radiobiology, James J. Nickson, Ed. (Wiley, New York, 1952).

H. B. Jones, Radiation and Other Factors Influencing Health and Life Span; Presented Am. Assoc. Advance. Sci., Dec. 1954.

H. J. Muller, Radiation Damage to the Genetic Material, in Science in Progress, 7th Series, George A. Baitsell, Ed. (Yale Univ. Press, New Haven, 1951).

George A. Sacher, On the Statistical Nature of Mortality with Especial Reference to Chronic Radiation Mortality, Radiol. 67, 250 (1950).

Shields Warren, Longevity and Causes of Death from Irradiation in Physicians, J. Am. Med. Assoc. 162, 464 (1956).

B. Effect of smoking:

E. C. Hammond and D. Horn, The Relationship between Human Smoking Habits and Death Rates, J. Am. Med. Assoc. 155, 4 (1954).

C. Effect of overweight:

L. I. Dublin and H. H. Marks, Mortality Among Insured Overweights in Recent Years, Presented at 6th Annual Meeting, Association of Life Insurance Medical Directors (Metropolitan Life Ins. Co., Oct. 1951).

D. Fat metabolism:

John W. Gofman, Serum Lipoproteins in the Evaluation of Atherosclerosis, in Experimental Methods for the Evaluation of Drugs in Various Disease States, Ann. N. Y. Acad. Sci. 64, Art 4, 590 (1956).

John W. Gofman and Hardin B. Jones, Obesity, Fat Metabolism and Cardiovascular Disease, Circulation 5, 514 (1952).

Gofman, deLalla, Glazier, Freeman, Lindgren, Nichols, Strisower, and Tamplin, The Serum Lipoprotein in Transport System in Health and Metabolic Disorders, Atherosclerosis and Coronary Heart Disease, Plasma (Milano) 4, 413 (1954).

Gofman, Jones, Strisower, and Tamplin, Evaluation of Serum Lipoprotein and Cholesterol Measurements as Predictors of Clinical Complications of Atherosclerosis, Appendix A, Circulation 14, 725 (1956).

Gofman, Strisower, deLalla, Tamplin, Jones, and Lindgren, An Index of Coronary Artery Atherogenesis, Modern Med. 21, 119 (1953).

Jones, Gofman, Lindgren, Lyons, Graham, Strisower, and Nichols, Lipoproteins in Atherosclerosis, Am. J. Med. 11, 358 (1951).

Lyon, Yankley, Gofman, and Strisower, Lipoproteins and Diet in Coronary Heart Disease, A Five-Year Study, Calif. Med. 84, 325 (1956).

E. Childhood disease: Hardin B. Jones, A Special Consideration of the Aging Process, Disease, and Life Expectancy, in Advances in Biological and Medical Physics, Vol. 4 (Academic Press, New York, 1956).

F. Physical impairment: Society of Actuaries, Impairment Study (Peter F. Mallone, Inc. N. Y., 1951).

G. Occupational exercise: The Registrar General's Decennial Supplement, England and Wales Occupational Mortality Part I, 1951 (Her Majesty's Stationery Office, London, 1954).

H. Diabetes: E. P. Joslin, H. F. Root, P. White, and A. Marble, The Treatment of Diabetes Mellitus, 9th Ed. (Lea and Febiger, Philadelphia, 1952).

I. Sex differences, urban rural differences: Vital Records of Denmark, Great Britain, Holland, and Sweden.

J. Accidents: Vital Statistics of the U. S., 1949, Federal Security Agency, Public Health Service, and National Office of Vital Statistics (U. S. Govt. Printing Office, Washington, D. C., 1951).

Interpretation of the Atomic Bomb Casualty Commission Report of Physical Growth of Hiroshima Children Exposed to the Atomic Bomb

Hardin B. Jones

Hiroshima children 5 to 19 years old (approximately 4,000, divided into 4 categories—males and females, exposed and unexposed) studied 5 years after exposure to the atom bomb are significantly shorter and lighter in weight than unexposed children, according to the report of Earl L. Reynolds to the Atomic Bomb Casualty Commission. Statistically the effect is slight in either sex, since weight is depressed about 2 percent and height is depressed about 1 percent (the standard error is 0.35 percent for groups of this size). Average individual variation in height or weight is about 12 percent (standard deviation) in Hiroshima children; therefore, the effect on height or weight of average radiation in the exposed group is approximately $\frac{\text{1 to 2 percent}}{\text{12 percent}} \times 100$, or about 10 percent of the effects of factors generally contribuating to lower-than-average individual differences in grows.

The effect of atom bomb exposure upon growth is measurably correlated with distance from the hypocenter of the explosion. The actual relationship observed is slight but significant, the correlation coefficient being about +0.1 for relationship of children's size and distance from the bomb. Since it is very likely that true exposure dose is rather poorly related to distance from the bomb at exposure and correction of this defective information should increase the correlation, the true effect of exposure upon children's growth is probably larger.

An improved estimation of quantitative relationship between exposure dose and growth in these children is possible by using groupings according to initial severity of symptoms, on the hypothesis that indeterminate shielding factors protected those who suffered fewer symptoms than would be expected from the theoretical dose. In comparisons by age and sex groupings, the asymptomatic groups were significantly larger in average physique, whereas the groups with definite irradiation exposure symptoms were significantly smaller. Such comparisons lead to a rough direct estimation of correlation between exposure and suppression of growth. Chi-square values show that the growth depression associated with exposure is significant. When chi-square values are converted to correlation coefficients, the result is equivalent to a correlation of approximately −0.4, indicating that the true relationship between irradiation exposure and growth, in doses sufficient to produce acute effects, may be larger than the above estimate; the square of the correlation coefficient indicates that 16 percent of the observed difference in growth is probably due to the effects of irradiation. Roughly estimated, the growth-depressant effect in humans should be equal to:

$$\frac{\text{growth change}}{\text{roentgen exposure}} = \left\{ \begin{matrix} \text{correlation coefficient} \\ \text{of growth vs. exposure} \end{matrix} \right\} \times \frac{\text{standard deviation of growth}}{\text{standard deviation of radiation exposure}}$$

as derived from the regression equation. The computed value in this case is:

$-0.4 \times \frac{\text{10 percent}}{\text{80 r.}} = -0.05$ percent per roentgen, all values being rough approximations.

The association observed between size and radiation exposure in Hiroshima is strikingly similar to that observed by Russell and Russell when mice were exposed to varying single doses of X-rays at 11.5 days after conception. Size of mice at birth decreased proportionally to radiation exposure from 100 r. to 400 r. The estimated effect upon embryonic growth per roentgen is 0.25 percent depression of size per r. It is noted that those children in Reynolds' study who were exposed in utero have twice the effect of decreased size seen in children exposed after birth; however, since the Reynolds study considered size 5 years after exposure rather than size differences at birth, as in the Russell study, the relative size difference at birth may have been even greater. Although the number of children irradiated before birth was too small to make the difference statistically significant, it is interesting to note that only a small correction factor would make the Hiroshima overall growth-depression ratio of 0.05 percent per roentgen comparable to the embryonic growth-depression ratio of 0.25 percent per roentgen observed in mice. These studies suggest that radiation effect upon both young mice and humans is the same with regard to suppression of growth.

Earle L. Reynolds, The Physical Growth of Hiroshima Children Exposed to the Atomic Bomb. Document submitted to the Atomic Bomb Casualty Commission, National Research Council, 1954.

Liane B. Bussell and W. L. Russell, An Analysis of the Changing Radiation Response of the Developing Mouse Embryo. Symposium on Effects of Radiation and Other Deleterious Agents on Embryonic Development, Biology Division, Oak Ridge National Laboratory, Oak Ridge, April 20, 1953.

Representative HOLIFIELD. Now I suggest that we have a discussion. We have certainly had a heavy diet today of philosophy on genetics and facts presented, and theories. I am sure now we can probably focus on some of the points we think should be focused on at this time.

I am going to ask Mr. Hollister to lead off with some questions.

DISCUSSION BY DRS. JAMES F. CROW, BENTLEY GLASS, A. H. STURTEVANT, HERMANN J. MULLER, W. L. RUSSELL, AUSTIN BRUES, AND HARDIN JONES

Mr. HOLLISTER. The first question, to Dr. Glass. Do we know specifically of any mutations in man that are radiation caused?

Dr. GLASS. No, you can't identify the individual mutations as being radiation caused or as having occurred spontaneously. The same kinds of mutations can occur through the action of radiation as may occur spontaneously. So you can't identify the particular mutations. There is aways a probability that even if the exposed person—the parent of the mutant person—received radiation, still the mutation might have occurred anyway. All we can go by is the increase in the frequency of mutations, which is thought to occur with a particular increase in dose.

Mr. HOLLISTER. So the radiation mutations produced in man or in any other organism are indistinguishable by kind.

Dr. GLASS. Exactly.

Mr. HOLLISTER. From any other sort of mutations.

Dr. GLASS. If we looked at the chromosomes we might find that a larger proportion of the radiation induced ones were visible losses, as has been brought out today. But just judging from the ordinary clinical symptoms, no, you could not distinguish them in kind from the spontaneous ones.

Representative HOLIFIELD. Are there any comments, or are you in general agreement? I see by your nodding that you are in general agreement.

Mr. Hollister. I would like to ask, Dr. Muller, if you know of any mechanism seriously proposed that would predict a nonlinear effect for the mutation dose relationship.

Dr. Muller. At high doses; yes. For what we call chromosome aberrations, but not for what we call gene mutations or point mutations.

Dr. Russell referred to a result of his and we, at our laboratory, obtained a similar one in flies, in which there was a less than linear apparent effect at very high doses, owing, as we judged, to the fact that the cells that had been worse hit were killed off more so that we lost the cases. But I do not see any way of getting a fundamentally nonlinear effect, especially at low doses. If the process takes place in any way like what we think it does, that is.

Representative Holifield. If any member of the panel wishes to comment, if you will raise your hand, I will recognize you. Dr. Russell.

Dr. Russell. I should like to add to that that I think the possibility does still exist, if there is killing of cells at low doses. That is, down to a level at which cells are killed I think the possibility of departure from linearity might still exist. This level may be lower than we might tend to think from experiments on sperm. The spermatogonia seem to be sensitive.

Dr. Muller. Might I add one word to that? If that is true, then it would work the other way from the way the people argue who believe in a threshold, because it would mean a relatively greater effect at low doses.

Dr. Russell. Yes. I tried to bring this out, that it would be an increase in the effect, rather than less. But I do not think departure from linearity would be expected at such low levels as 0.1 roentgens, for I don't think there would be enough killing of cells at this dose level to make any difference. This would perhaps apply at the 10-roentgen level or something of this order. Down at the very low doses I would agree 100 percent with Dr. Muller that linearity would be expected.

Representative Holifield. Dr. Jones.

Dr. Jones. It is certainly true that several ionization events may be necessary for critical change within the cells. The ravages that can take place in the cell with time are so similar to radiation-induced events that even though one ionization occurs and several are needed, perhaps the preceding natural aging events have made the internal climate such that one ionization could produce that change.

In this sense we perhaps have a further reason for accepting the hypothesis of a proportional change down to very, very small doses. Of course, at these small doses we have very small effects.

Dr. Russell. Could I add one more point to this? My point is that at the very low doses you would still expect linearity, but the slope of the line at these low doses might be steeper than would be predicted from our present data at the 300 and 600 r. points.

The question is, Have we measured the full effect at the present time? We think we need to measure the mutation rate at lower doses to be sure.

Representative Holifield. Thank you.

Mr. Hollister. I would like to ask if it is not true that you as geneticists—when you draw a line between a good and a bad muta-

tion—do this almost entirely on the basis of how the mutation affects reproduction. Who would like to answer that. Dr. Crow?

Dr. Crow. From the standpoint of some theoretical computations that are made in assessing damage in the long-time future, this kind of an assumption is made. But in general, although we realize that most harmful mutations have some harmful effect on survival or reproduction, I think most of us—probably all of us—would be agreed that this is not really the point which as humans we are interested in. We are interested in the pain, disease, misery, all of which are associated with the reduction in survival value.

Mr. Hollister. In other words, it is possible you could get into a situation, for example, where by process of continued mutation and selection, you could achieve a population that would reproduce very prolifically, but would have all sorts of other defects, that is, susceptibility to diseases and shortened life span and so on.

If we apply this criteria of good and bad mutation, you could do this?

Dr. Crow. As a very remote theoretical possibility. I don't think that is a likely possibility at all.

Mr. Hollister. Dr. Crow, another question for you. This is the question of numbers of individuals limiting in the quantitative studies of natural mutative rates either in man or other organisms. Is the size of the population that you have to work with a limiting consideration in the kinds of experiment that you can perform or the kinds of data you can obtain?

Dr. Crow. In the study of human populations?

Mr. Hollister. Either in humans, fruitflies, or mice.

Dr. Crow. I would say the numbers are the principal limiting factor. If one studies humans one cannot make experimental matings and that is serious. The organisms that are preferred for research purposes are those that you can grow cheaply and in large numbers, and that is why the fruitfly has been used.

Is that the point of your question? I am not quite sure.

Mr. Hollister. My understanding is that you had postulated somewhere that there was some size or some number of individuals, perhaps a million or perhaps 10 million, that was a practical maximum size that an experimenter could work with.

Dr. Crow. I think you are confusing me with the U. N. committee. I cannot think of the man's name. Yes, Dr. Appleyard.

Mr. Hollister. That is possible.

Dr. Crow. I think these are statements from Appleyard who has done some computations on the size of population that would have to be studied in order to detect differences of a certain small magnitude.

Mr. Hollister. Is it not true that in some of these studies he has concluded that the populations have to be enormous?

Dr. Crow. That is correct. One can reach those conclusions even before seeing his figures.

Dr. Muller. Could I interpose something since we mentioned the U. N. committee? I received information from someone in the audience in regard to a question that was raised or that I raised during my talk about the composition of the U. N. Scientific Committee. I stated that Dubinin had been scheduled to appear from Russia. I got information that he did not show up. Also, that he had not been

scheduled to be a delegate, but an adviser or consultant. Also, that at the most recent meeting there were various advisers present from other countries who were geneticists, not delegates, however. The geneticists who were delegates were confined to those I listed. Even though most of the discussion was done by the consultants rather than the delegates, it is an unfortunate situation if geneticists are not actual delegates, because although they were evidently given free rein during the discussions on the effects on posterity, nevertheless, as I indicated before, the effects on the exposed generation itself are also very important, and are very possibly closely related to the genetic subjects. Therefore, the committee should have geneticists to discuss those effects also.

Representative HOLIFIELD. Without objection, the record will be corrected.

Dr. MULLER. Yes, sir; it should be corrected. Our country had two good geneticists as consultants.

Mr. HOLLISTER. Dr. Muller, do you suppose that something analogous to the uncertainty principle in physics could exist in genetics with regard to this threshold question? For example, presumably we have to have an effect, tested in a certain number of cells. Leukemia, bone tumor, these have to involve more than one cell for us to observe. If this is true, does not this of itself indicate a possible threshold when in fact there might not be one. That is, we cannot observe a leukemia in 1 cell, can we, or a bone tumor involving 1 cell? Presumably it involves many cells.

Dr. MULLER. If the individual is unlucky enough by reason of the cell being lucky enough, then the one cell that became leukemic could give the individual leukemia.

Mr. HOLLISTER. But you would not know this until it involved more than one cell?

Dr. MULLER. No. There could be a mutation in one cell if our conception is right.

Mr. HOLLISTER. You would not know this experimentally?

Dr. MULLER. You would know it experimentally if that cell was in position in which it could express the tendency given to it by the mutation to divide or to reproduce or multiply in an uncontrolled manner. After a while it would crowd out the other cells and the person's blood would be full of these white cells, and he would have leukemia.

Mr. HOLLISTER. The thing that you would measure would be the presence of leukemia in the person after some of the multiplication had occurred.

Dr. MULLER. That is right.

Mr. HOLLISTER. So you would not know he had leukemia until it showed up as a result of many cells being pathologic?

Dr. MULLER. I think the question is on the same basis as all other genetic questions. All you get is a more or less random sample. You have to judge by that sample. That is why you need large statistics.

Representative HOLIFIELD. Dr. Glass has something to say on that.

Dr. GLASS. May I speak to that question, too? The more we press back into the knowledge of how the genes produce their effects, the more possible it becomes to detect the nature of those effects in individual cells. Thus even in a tissue culture of human skin epithelial

cells, if a mutation occurred in the one gene that we know of that controls the production of pigment, and changed it to an albino type, you would not have to have a thousand cells or a whole individual to know that mutation had occurred. You could spot it in that one cell.

If we knew enough about the biochemistry of different cells and enzymes, we could easily detect this in single cells.

Senator ANDERSON. Dr. Crow, do you agree with that? It looked as if you did not.

Dr. CROW. Let me get into this act, too. Back to what I think maybe you have in mind. At the present time we can demonstrate a linear effect perhaps down to 25 r. We could do a very large experiment and perhaps demonstrate a linear effect down to 10 r. We could do an enormously large experiment and demonstrate it down to 5 r. One cannot continue indefinitely. If that is what you mean by an uncertainty principle, I think there is something here. One cannot do a large enough experiment to demonstrate linearity down to an arbitrarily low value. Having said that, I would like to say that about this time we start relying on purely physical considerations of the kind that other people such as Dr. Pollard have been mentioning.

Mr. HOLLISTER. How about some of the testimony that Dr. Brues gave, which I am not sure I understood perfectly, and do not have in front of me—and if he is here, he might want to comment himself—to the effect that to cause cancer in body cells more than one cell would have to be affected by a dose of radiation before this effect would occur.

This might imply a threshold, although in fact a threshold might not exist.

Representative HOLIFIELD. Dr. Brues, have you been correctly quoted? Will you come forward? Pull up a chair and defend yourself.

Dr. BRUES. Was the question addressed to me?

Mr. HOLLISTER. I think you can help us first to make sure I paraphrased what I thought you said correctly.

Dr. BRUES. I shall in that case rephrase it to say this: I should not necessarily assume that a somatic mutation would be the basis of cancer a priori. But if it is, it still might be a little more complicated than the genetic situation, where just one cell is involved. I can think of at least two different ways in which that might occur, but I shall not take the time to go into them.

Mr. HOLLISTER. But these complications that you speak of involve the notion that more than one cell would have to be affected by the radiation; is that correct?

Dr. BRUES. That would be correct, yes. This is not proven or disproven, but it is a suggestion which I think is as likely as the other one which has been made, rather categorically.

Mr. RAMEY. Do the complications make for the threshold, then?

Dr. BRUES. I beg pardon?

Mr. RAMEY. Do the complications arising mean that you have to have more dose before you would get some sort of threshold effect?

Dr. BRUES. Yes. I think the suggestion I made rather specifically, and this was based on many things which have been observed in cancer pathology, rightly or wrongly, is the fact that cancer rather tends to arise in a tissue which has been generally disarranged. As for a general disarrangement or disturbance of the blood vessel supply of the tissue, this, I think, is not linear with radiation dose, but

like the erythema produced by irradiation on the skin, the old method of measuring dose, this appears to have some sort of threshold. If that is necessary as well as something else that radiation does, then we will not have a linear response. That is the point I made.

Representative HOLIFIELD. Is there any comment on that? Observing no hands raised, we will go to the next question.

Mr. HOLLISTER. Dr. Crow, do you know if experimentally, a population has ever been destroyed genetically?

Dr. CROW. I cannot think of an example. You mean by accumulating so large a number of mutations that ultimately it was killed off?

Mr. HOLLISTER. Yes.

Dr. CROW. Dr. Russell has reminded me of an example. One of Dr. Bruce Wallace's populations died out presumably as a consequence of very heavy radiation. Is the theoretical point here whether it is possible to induce a large enough number of mutations to kill off posterity without killing off the first generation?

Mr. HOLLISTER. Presumably this experiment proves it is possible.

Representative HOLIFIELD. I think we have time for one more question. We will allow each one of you, regardless of the time element, to comment on this. Are conclusions in the field of genetics being arrived at too far in advance of the data?

Dr. STURTEVANT. It seems to me, sir, that we have to draw some conclusions. We have to do something, because not doing something is equivalent to doing nothing. We therefore have to proceed on the basis of the best information we have. This is a common enough human experience; it happens to all of us every day. We have to reach decisions as to what to do or not to do without all the information we should like to have. I don't think that the situation here is any different from that which is usual. We have to proceed on the basis of the best information we can lay hands on.

Representative COLE. You concur with Dr. Crow when he said it is better to guess wrong than not to guess at all?

Dr. STURTEVANT. I think it is not only better to; it is necessary to. You have to make some kind of guess in order to live at all in this world.

Representative HOLIFIELD. Would there be any other comment on that?

Dr. JONES. I think the same thing applies to the question of life span effects. Here we simply do not know how life span effects operate in the small-dose range. We may be a long time in finding out information that applies directly in the small-dose range. In the absence of that, we have to make some estimate of the effect. If we relate this problem to fallout, we may estimate fallout to be now about 2 percent of natural radiation. Natural radiation is estimated to cause a life span loss of 25 to 50 days, if we live as long as 70 years. So the effect of fallout is only about 1 day or half a day on this basis, if we assume proportionality. This loss is extremely small but may be worth keeping in mind, even though it may be as small as one twenty-five thousandth of man's life span.

Dr. RUSSELL. I should like to make two comments on this. I think the geneticists on the National Academy committee have faced up to this problem, and in some sense should be complimented on this. I am a member of the committee, so I do not want to compliment myself, but the others. Most of them were experimental scientists and

they were very reluctant to come out with figures based on what they would consider, in some respects, inadequate evidence. However, it was necessary to face up to drawing a conclusion.

I don't think anyone should be reprimanded for drawing a conclusion when a conclusion was requested.

Representative HOLIFIELD. I certainly do not want you to think that this committee is reprimanding.

Dr. RUSSELL. No. Other people have.

Representative HOLIFIELD. It is very salutary that you brought this out. We believe it is in line with your scientific integrity to point out danger signs, whether you are sure how great they are or how many.

Dr. RUSSELL. I did not mean to apply my remark to this committee. I believe geneticists have been blamed for making too definite statements based on the evidence, perhaps mostly by medical specialists.

The other point I have is that we perhaps know more about this genetic hazard than we did about many other hazards we have experienced in the past. Many hazards man has been encountering were not known to be dangerous until many humans died from them. For example, many industrial poisons and even radium in the first place. I think the genetic hazard represents a situation where we know in advance a good deal more about it than we have done for some other things, including, I might say, some medical treatments. Some of these have been found to be hazardous only after several people have suffered from them.

Representative HOLIFIELD. Of course, the magnitude of the threat of nuclear radiation from war is the compelling factor in this matter.

Dr. RUSSELL. Yes.

Representative HOLIFIELD. Dr. Crow, I am sure you want to say something.

Dr. CROW. I agree heartily with the two people who have spoken.

Dr. GLASS. I agree, but I would like to add just one very brief comment. We know there is a genetics hazard. We don't know the exact amount of that hazard. We think that it is better to overestimate it than to underestimate it, and play safe, than to underestimate it and reap irreparable damage.

Representative HOLIFIELD. Mr. Ramey has a question, I understand.

Mr. RAMEY. I believe most of you gentlemen sat in on yesterday's discussion of the pathological or somatic effects. As biologists rather than geneticists, do you think that this linear effect that was brought out today for genetics applies in the somatic pathological field?

Representative HOLIFIELD. Who would be so bold as to answer that?

Dr. CROW. I will be so bold as to make an answer, but it will not be very definite. I believe most geneticists are convinced that at least some of the somatic effects of radiation are of a linear nonthreshold sort. I don't think anybody would be so dogmatic as to state that all such effects are or even what the fraction is.

Representative HOLIFIELD. Dr. Muller, what do you think about that? Would you have any opinion on that?

Dr. MULLER. My opinion is, as I said before, that the most important effects, those from which the human race when exposed to radiation suffers by far the most damage, and that is the shortening of

life effect, and probably leukemia and some related things, are in all probability linear without a threshold.

Might I also say with regard to the other question of whether we are going too far beyond the evidence, that it was not, of course, possible in these discussions to present the details of the evidence, and the reasoning involved, but that the estimates that were presented as what we regarded as probable were not in any sense guesses or speculations, but arrived at as a result of an enormous amount of work and calculations. Not only that, but that they were arrived at by more than one method. There was a totally different method used recently by a number of geneticists in arriving at the frequency of mutations in man. It was remarkable that at the end it was in very good agreement with the estimate reached by the first method that we had used.

I would make this qualification only of what Dr. Glass said, that we were not trying in the main to show the maximum effect. I would regard the preferred estimates as minimum estimates.

Senator ANDERSON. My question was not too serious an inquiry. I was just wondering if geneticists had a union, guild or gang, or something that teaches you to hang together? This is not only the most agreeable group of seven scientists, but certainly the most agreed group I have seen. I commend you of the fact that you have been able to hang together as long as you have through a rather long day.

Dr. STURTEVANT. I would like to say that I think it would have been very difficult to get together a group that would have disagreed with most of what has been said here among practicing geneticists.

Dr. MULLER. Might I take the occasion to thank the members of the committee on behalf of all of us for their having put on these hearings on this subject.

Representative COLE. Mr. Chairman, I am not sure that the question can be answered, but at least I am curious enough about it to pose the question.

Are there any other firm conclusions that can be reached based on data and experience with respect to the danger or hazard of radiation other than the one that was just voiced by Dr. Glass, that it does constitute a hazard? I am directing the question at all of them and I prefaced it by saying I was not sure it could be answered, but I was going to ask it anyway.

Dr. CROW. I find it hard to answer, Mr. Cole, because of the difficulty of deciding what we really mean by firm in a case like this. I think the conclusion that any effect of radiation is harmful is about as firm as a scientific conclusion ever is. Of course, the quantitative figures are much less firm.

Representative HOLIFIELD. From the field of physiology?

Dr. JONES. I would echo Dr. Crow's opinion that it looks as though we can definitely say that some effects do occur, and at very small dosages they are undoubtedly small effects. But they seem nevertheless to be effects, and we cannot say with certainty what the relative orders of magnitude of these effects are. I don't think there is any reason to be more concerned than to try to get better information as soon as possible. There is no public hazard at the moment compared to usual concepts of public hazard. We certainly do owe it to ourselves to find out what these effects are.

Representative HOLIFIELD. On behalf of the committee, gentlemen, I wish to express our collective thanks for this participation in this set of hearings. I am sure they will be read by a great many thousands of people with great interest. Your audience will be large. I think these hearings will be the year's best seller.

Tomorrow we will have Dr. William B. Looney as the leadoff witness, followed by Dr. Libby, Dr. Ralph Lapp, and Dr. Walter Selove, in the Senate caucus room at 10 o'clock.

(Thereupon at 5:10 p. m., Tuesday, June 4, 1957, a recess was taken until Wednesday, June 5, 1957, at 10 a. m., in the Senate caucus room.)

THE NATURE OF RADIOACTIVE FALLOUT AND ITS EFFECTS ON MAN

WEDNESDAY, JUNE 5, 1957

Congress of the United States,
Special Subcommittee on Radiation of the
Joint Committee on Atomic Energy,
Washington, D. C.

The special subcommittee met, pursuant to recess, at 10:05 a. m., in the caucus room, Senate Office Building, Hon. Chet Holifield, chairman of the subcommittee, presiding.

Present: Representatives Holifield, Durham (chairman of the Joint Committee), Price, Cole, Van Zandt; Senators Anderson, Hickenlooper, and Bricker.

Also present: Professional staff members: James T. Ramey, executive director; George E. Brown, Jr., Hal Hollister, staff technical adviser, and Paul C. Tompkins, consultant.

Representative Holifield. The committee will be in order.

The subcommittee will continue its hearings on the effects of radiation on man, and today we will take a step back toward the pathology section of our witnesses. Our witness this morning is Dr. William B. Looney from the John Collins Warren Laboratories of the Huntington Memorial Hospital of Harvard University, Massachusetts General Hospital, who has been for the past 7 years conducting experiments and partaking in clinical work directly with patients who have had radiation damage. We should have had Dr. Looney on 2 days ago when we had the pathology section, but we could not fit him into the program because of the time limitation. We are mighty happy to have him with us this morning.

I notice you have quite an extensive statement here, and it is your purpose to submit this for the record, I understand, and to discuss the highlights of your statement.

Dr. Looney. That is correct, sir.

Representative Holifield. You may proceed, Doctor.

STATEMENT OF DR. WILLIAM B. LOONEY, MASSACHUSETTS GENERAL HOSPITAL [1]

Dr. Looney. Mr. Chairman and members of the committee, first, I should like to express my appreciation for the opportunity to

[1] Date and place of birth: March 18, 1922, South Clinchfield, Va. Education: Bachelor of science, Emory and Henry College, 1944; United States Naval Academy, 1941–44; doctor of medicine, Medical College of Virginia, 1948; internship, Presbyterian Hospital, Chicago, 1948–49; assistant residency, internal medicine, 1949–50; AEC postdoctoral research fellow in medical scence, National Research Council, Argonne National Laboratory, 1950–52. Work history: Assistant in medicine, University of Chicago, 1950–52; Radioisotope Laboratory, United States Naval Hospital, Bethesda, 1952–55; Officer in Charge of Radioisotope Technicians School, National Naval Medical Center, Bethesda, 1953–55; subcommittee on radiobiology, National Research Council, 1953–55; Consultant to Egyptian AEC, Cairo, Egypt, 1954– ; specialist, Public Health Service Research, fellow of National Cancer Institute, 1955– ; clinical and research fellow, Massachusetts General Hospital, 1955– ; visiting fellow in physics, MIT, 1955– ; consultant in radiation protection to Surgeon General, Department of the Navy, 1957–. (Submitted by witness.)

present this information to the Joint Committee on Atomic Energy.

As was just previously mentioned, my studies primarily have been on the effect of radiation in man, and the importance of the information to be presented this morning on the effect of radiation in man by radium is related to the fact that both radium and strontium are deposited in the skeleton similar to calcium. We have a considerable amount of information about the effects of radium in man; however, we do not on strontium. Since both strontium and radium act similarly to calcium, having the knowledge of radium, we can make estimates of the effects of strontium on man by our knowledge and factual data of the effects of radium on man.

I should like to confine my testimony primarily to the available knowledge we have on the effects of radium in man, and then at the end of the statement, based on the estimates from man, make some estimates as to the radiation dose from strontium necessary to produce similar changes in man over a 70-year period.

Man is constantly confronted with toxic agents in his environment which, if present in sufficient quantities, may produce temporary or lasting changes. By the accumulation of clinical and experimental information about the quantity of these toxic agents which produce minimal changes, safety measures are established. The present maximum permissible concentrations for radio elements in use today are based primarily on the results of clinical and investigative studies made over the past half century.

The principal sources of material for the study of the effects of internally deposited radioactive materials in man have been individuals who were employed in the luminous-dial industry, and persons who received radium for medical purposes. Radium salts had been given orally and intravenously for hypertension, arthritis, anemia, and other medical disorders from about 1915 to 1930. The painting of watch dials with luminous materials containing radium, mesothorium, and radiothorium started in this country in about 1914.

Adequate safety measures were not taken until about 1925–27, following the deaths from bone tumors, anemia, and crippling bone lesions, or all three, which occurred in some of these workers.

The first report in regard to the maximum permissible concentration, which I shall refer to as MPC, for radium was made by the National Bureau of Standards in 1941.

Senator HICKENLOOPER. I hate to interrupt you, Dr. Looney, unless you are willing to be interrupted in your statement.

Dr. LOONEY. That is perfectly all right.

Senator HICKENLOOPER. I notice the statement about the safety measures taken in 1925–27, following the deaths from bone tumors, anemia, and crippling bone lesions, which occurred in some of these workers. Do you comment later on the fact of whether or not all of the workers who received substantially the same dosage had the same results?

Dr. LOONEY. Yes, sir; this will be the context of the report.

Representative VAN ZANDT. Dr. Looney, when radium was first used, was there the same concern regarding the safety factor as there is today for radiation?

Dr. LOONEY. Well, this is a very fascinating history. The initial use of radium medically was based on the finding that radon, which is the first daughter produced by radium, was found in the waters of

the health springs in Europe, and a few biological experiments were done to show that radon or radium had beneficial effects. It was first believed that radiation had primarily a beneficial effect. As a result of the biological experiments and the finding of radon in the health springs, numerous people were treated. Historically, we now have the spectrum shifting from one side to the other.

Representative VAN ZANDT. One more question.

Dr. LOONEY. Yes.

Representative VAN ZANDT. I would like to ask about X-rays. Are you familiar with the development of X-ray from the time it was first used and was concern shown for the safety factor?

Dr. LOONEY. I think Dr. Taylor, who is in the audience here, would be certainly much more competent to comment on the general history. I think he is far more familiar with the overall details.

I will say, though, that certainly throughout the history of the uses of radiation, there certainly has been concern about its deleterious effect.

Representative VAN ZANDT. Thank you.

Dr. LOONEY. To go back to the report, the first report in regard to the maximum permissible concentration for radium was made by the National Bureau of Standards in 1941. Seven individuals having from 0.02 and 0.5 micrograms of radium in their bodies from 7 to 25 years had no noted changes referable to the deposition of radioactive materials. However, death had occurred in patients having as little as 1.2 micrograms of radium. The maximum permissible level of body burden for the amount of radium was established at that time as one-tenth microgram of radium. This was after the initial rapid elimination of radium from the body.

If radium is taken in, almost all of it is eliminated, but the fraction remaining in the bone is eliminated rather slowly. So the maximum permissible concentration for radium was set in 1941 at one-tenth microgram.

There have been two large studies, one made at the Argonne National Laboratory in Chicago, with which I was associated, primarily as coordinator in the evaluation of the clinical aspects with biophysical effects of the investigation. The other was made at the John Collins Warren Laboratories of the Huntington Memorial Hospital of Harvard University, Massachusetts General Hospital, Boston, and the Massachusetts Institute of Technology. These studies are continuing, and I have in the acknowledgments at the end made note of the people involved in these studies.

I would like to emphasize this was a very large study in which I was primarily interested in the clinical aspects, and I shall incorporate in the report the acknowledgment of the large group of people participating in these two studies.

This report is primarily a summary of these two recent investigations of people at or near the present maximum permissible concentration of radium.

Of the 78 patients that have been examined, the mean age of the luminous dial workers was about 21 years, and the mean age of the patients was about 34 years when they received radium. The average time of retention for both groups has been about 25 years with a

range of about 10 to 35 years. The average of these people in 1951 was about 50 years.

To summarize, we have information on people beginning about 15 to 20 years of age, and the major part of these patients have had radium in their bodies for 25 years. Many of these people are in the older age group, although the majority of the people are 50 or 60 years of age. We would like to have for a more complete picture additional information of people in the older age groups, and also people in the younger age groups. We have in the middle of the life span more information, but at either end the information diminishes.

Representative HOLIFIELD. Is there any information that the younger age group, with the same dose, is affected more deleteriously than the older age group?

Dr. LOONEY. Comparison of the effects of skeletal radiation in the unselected luminous dial workers and patients who received radium medically was made. It is not possible to draw conclusions on the present information in these two groups of patients. However, based on the available clinical information one might consider that the luminous dial workers were less affected after beginning employment at ages 15 to 20 years, than the patients who received radium medically in the 1930's.

Representative HOLIFIELD. Is that on comparative doses?

Dr. LOONEY. On comparative doses, yes, sir. As I say, I think this data is inconclusive. This is just based on the information we have available at present in man.

Senator ANDERSON. Do luminous dial workers go to work at 15?

Dr. LOONEY. Fifteen to twenty years of age; yes. Some of them started at 14 and 15. This was in 1915.

Representative HOLIFIELD. And they absorbed this radium, as I understand, by putting the tip of the brush in their mouths to point up the brush in order to paint the numerals on the watches. Was that the way they received it mostly?

Dr. LOONEY. I think this, plus the fact there was radium in the atmosphere and they inhaled it. There were no known effects at that time. In 1924 a dentist discovered destruction of the mandible of some of these people. This established the fact that the ingestion and inhalation of radium produced deleterious effects in man. This very tragic experience is the foundation upon which some of the maximum permissible concentrations of the most important radioelements in use today are based.

There is one other point I think should be brought out in regard to these patients. Ten of these fifty radium patients, and sixteen of the luminous dial workers were selected because of symptoms. In other words, they were discovered because they were having trouble. It is apparent that, if we had a thousand people and we saw only the people who were having difficulty, we would have a biased or selected group of people from the population of radium patients, and luminous dial workers. About one-third of these people were found because they had symptoms. This would tend for one to overemphasize the effect of ardioactive materials based on the present information. This is something the committee should keep in mind.

Thirteen of these seventy-eight patients having between one-hundredth and four-tenths micrograms of radium had no changes attributable to the effects of radium, with two exceptions. One patient

having 0.15 microgram of radium had minor areas of decreased density demonstrated roentgenographically in his teeth. These are minor changes; however, this patient was discovered because of the changes. These findings are characteristic of the dental changes in people we have seen with more radium in their body. The other patient was an 83-year-old woman who had very severe arthritis, was incapacitated, and had minor changes in her skeleton. Old age, plus the incapacity, might have contributed to the changes.

Sixty-five patients had between 0.5 and 23 micrograms of radium. All of these patients had either skeletal changes or symptoms attributable to radium, or both, which we could reasonably attribute to radium, with 3 exceptions; these people had between five-tenths and 1 microgram of radium. We should keep in mind the one-tenth microgram as the present MPC for radium, then we can mentally refer to the changes.

These patients began to have difficulty with about 10 times the amount of radium greater than the maximum permissible concentration.

Now I would like to go into the mechanism of deposition of radium in the skeleton, the pathological changes seen, and then relate these pathological changes to the amount of radium in the body. Radium is deposited in small areas of high concentration, so that we have an irregularity of deposition. This is an important factor to take into consideration, because in the last analysis we are interested in what happens in certain areas. If radium is concentrated in the area of bone tumor formation, then we are not as interested in the average value of total bone, as we are interested in these areas of concentration.

Analyses of samples of bone from some of these people have been made. In samples taken from different bones and from several parts, the same bones, we have found that these concentrations vary as much as by a factor of 10, and maybe greater. When we talk of average values of one-tenth microgram, we must realize that these people have areas of concentration which may be much greater than that. This has to enter into the consideration of the MPC of radium as well as strontium.

If you will go back to figure 2 at the end of my statement, there is an autoradiograph which shows how the radium is deposited in one of the bones. It is the picture here [indicating], showing the actual radium deposition in the bone. You will see that the dark areas represent areas of radium. (See p. 1176.)

In considering the effects of radium, you must consider the effect in the small areas of concentration in relation to the pathological changes.

Now, the changes that have been shown to develop in these people are areas of abnormal bone formation, which occur usually at the ends of the bone. When an X-ray is taken they show up as areas of increased density. The effect of radium causes abnormal bone to be produced, and it is generally considered that tumors develop in and around these areas of abnormal bone formation.

The other characteristic change is areas of decreased density in the shaft, or the middle of the bones, and these show up in the X-rays as areas of decreased density. When we examine people who have had a sufficient amount of radium, we see these areas of either increased or decreased density which are scattered throughout the skeleton, and

these characteristic changes are one of the most reliable clinical methods we have at present for determining the early effects, or the first detectable effect, of radioactive materials in man.

Chairman DURHAM. What kind of a dose, Doctor, was that received by the bone structure in figure 2 of your statement?

Dr. LOONEY. This patient had 1.3 micrograms of radium, sir. This is about 13 times the present accepted MPC for radium.

In figure 4 you will note that this is a very detailed microscopic picture of radium being deposited in one of the fundamental units of the bone, known as the Haversian system. You will note the alpha tracks from the radium coming from those two Haversian systems, and this relates to the black area you saw on the autoradiograph of the whole bone. This is a microscopic picture showing the radium deposited in the small area. (See p. 1178.)

Representative HOLIFIELD. Do you have that same picture on the screen?

Dr. LOONEY. Yes, sir, I do. This is the picture here [indicating]. I might point out that here [indicating] is the area of the increased density that you see at the heads of the bone here and here [indicating]. The areas of decreased density are in here and here [indicating].

The areas that you will notice here [indicating] are the areas of destruction in the bone, and this is an X-ray of the bone showing the areas of increased density and the areas of decreased density, and these are changes characteristic of radium deposition. It is the best clinical method we have of determining the effects of radium.

Representative HOLIFIELD. Is it your judgment that the strontium 90 would have the same effect?

Dr. LOONEY. It is the best means we have of comparing the effects of strontium 90 to the effect of similar radio elements in man, and we have to go on the best available evidence we have.

Representative HOLIFIELD. Have you taken pictures of bones of mammals that have been subjected to strontium 90 exposure?

Dr. LOONEY. This has been done in some of the atomic energy laboratories. I have not been directly connected with this, and I am sure that some of the people in the audience could comment on that. Strontium and radium and calcium are deposited in the skeleton in similar manner; this is the basis of comparing the radium data with the present level for strontium.

This [indicating] is the picture of the alpha track, or the actual radiation from radium in one of the fundamental units of the bone, and this [indicating] is the underlying bone, showing an area of destruction, and another area showing no change.

Here [indicating] it shows the normal bone, and this is a typical osseous tissue adjacent to the normal bone, which results in these areas of increased density seen roentgenographically. In animals we see this atypical osseous tissue formed following plutonium, strontium, and radium administration, and in man similar changes are produced by radium. We consider that the mechanism is similar in the production of these changes.

Chairman DURHAM. What kind of rays are those, Doctor?

Dr. LOONEY. These are alpha rays. This is actually a picture taken by photographic emulsion over the bone itself. In other words, the track was coming up in the photographic emulsion, and it is like

taking a photograph, and this is the photograph of the alpha track.

Representative HOLIFIELD. No one has used "micrograms" before in the presentation. Will you relate that to a microcurie?

Dr. LOONEY. Yes. The microgram of radium and the microcurie are the same, because the microcurie was established from radium. So if I speak of micrograms or microcuries, as far as radium is concerned it would be the same.

Representative HOLIFIELD. That is what I wanted you to say for clarification of the record.

Dr. LOONEY. Yes.

Representative HOLIFIELD. Are the alpha or gamma rays which might be emitted of the same intensity and range in strontium 90 as in radium?

Dr. LOONEY. No, sir, they are not, because most of the radiation from radium is alpha radiation, and the radiation from strontium is from beta radiation.

Now you have heard the term "relative biological effectiveness" used. The RBE of radium has not been taken into consideration in the determination of the present MPC for strontium 90. Tho RBE [indicating] may be more than unity [indicating], so that this would permit the raising of the strontium level based on the radium data, if we were to determine that the effectiveness of irradiation from radium was greater than strontium in producing biological changes. In other words, it is generally considered that the alpha rays are more effective in producing biological changes per unit of energy dissipated to the material. These may be inherent safety factors that can be incorporated in present considerations of the MPC for strontium.

Representative HOLIFIELD. So if there would be a difference, it would be weighted toward the greater effectiveness of radium?

Dr. LOONEY. Yes, it would be weighted. Any change would be to the raising of the strontium MPC.

I would like briefly to comment about the hematological changes in these patients. The changes have been minor, and the only changes we have found have been minor changes in the size and shape of the red cells. In a few exceptions there have been anemias that have occurred in these people, but the hematological findings have been very minor changes compared to the skeletal changes.

I should like to go on to symptoms, and probably I should define what I mean by symptoms in these discussions. I shall refer to symptoms as the time when the patient becomes subjectively aware of these skeletal changes which we see. In other words, we first see skeletal changes in many of these people who have no symptoms at all. When skeletal changes progress to the point that the normal configuration of the bone is destroyed, symptoms usually occur the patient may have a fracture of the femur, or he may have destruction of the hip, and he limps, then these denote symptoms. The patient is aware these skeletal changes are present.

In going over these patients, there have been 11 patients of the 78 who have had destruction of the hip, the head of this [indicating] hip bone, so that they walked with a limb. All of these 11 patients have had seven-tenths micrograms or more of radium in their bodies.

There have been five patients with fractures of the femur. Some of these fractures have occurred from very minimal trauma. I remember one patient we examined had a fracture of the femur after

her husband came to a sudden stop at a stoplight, and the pressure of her foot on the floorboard of the car caused this fracture of the femur. So that the skeletal system does become more fragile, after a period of time, with these people in this range.

Representative HOLIFIELD. How did that particular patient receive that does of radium?

Dr. LOONEY. This patient was a luminous dial worker, sir, and she worked at Ottawa, Ill.

If you will turn to chart 1, you will note that this is the summary of the X-ray findings of 32 patients who had a complete skeletal survey. These patients were arranged in the order of increasing amounts of radium in the body. The frequency with which these characteristic skeletal changes, which I have shown to you, have occurred, were plotted as a function of the amount of radium in the body. You will note from the left hand vertical bar that there were 15 patients having between five-tenths and 1 microgram of radium in the body. You will notice that about 15 percent of the total bones that could be involved had these characteristic changes. (See p. 1174.)

There were 8 patients between 1.1 and 2 micrograms of radium, and you will notice that the frequency goes up to about 55 percent, and that between 2.1 and 14 micrograms of radium it goes up to about 60 percent.

If you will turn to table 1, you will notice that in the first group of patients, the average age was 49; the average age of the second group was 52; the average time of retention in the first group was 20 years; the average time of the second group was 22 years; the average age of the third group was 61 years; and the average time of retention was 22 years. (See p. 1174.)

We have a group of people who are about the same chronological age, who have radium retained for about 20 years, in which we can show a correlation between the frequency of these characteristic changes and the amount of radium in the body.

I think this is probably one of the most significant clinical observations that has been made—the correlation an objective clinical change with the amount of radium in the body within a specific dose range. I want to emphasize that extrapolation of the result of this clinical data either one way or the other would present many difficulties.

Chairman DURHAM. How did these patients receive this, Doctor?

Dr. LOONEY. With two exceptions, all of the people received this for medical purposes.

Chairman DURHAM. For treatment of other diseases?

Dr. LOONEY. Yes, sir; for the treatment of other diseases.

An important point to bring out here is the fact that people with 10 and 15 micrograms of radium did not have a proportionate increase in skeletal changes. In other words, we did not see people with greater amounts of radium in the body having greater changes. This would be consistent with the hypothesis that the changes that we see are the result of the dynamic interrelationship between the destructive processes and the reparative processes of the body. If we had radium with some other destructive skeletal disease, we might see changes at a much lower level than if the patient had no disease. Radium, plus condition A plus condition B might produce changes at 1 microgram. Radium, plus condition B might produce changes at 5 micrograms, and radium alone might produce changes at 10 micro-

grams. This is consistent with the hypothesis that the body is constantly repairing itself from destructive changes. When the body can no longer repair these changes, then permanent changes occur, in regard to radium, the skeletal destruction can be seen on roentgenographic examination.

Representative HOLIFIELD. As a term of common reference, would you say that any effect upon the bone is usually referred to as a tumor or cancer of the bone?

Dr. LOONEY. No, sir.

Representative HOLIFIELD. Or is there a differentiation between those two?

Dr. LOONEY. Yes, sir; there is quite a differentiation between those two.

Representative HOLIFIELD. Will you explain that for the record?

Dr. LOONEY. Yes, sir. These changes which we have seen—these changes here [indicating]—are minor changes, and are from abnormal bone formation. The areas here [indicating] are from small areas of destruction in the bone, and these can be seen in other conditions but usually not with the distribution and the characteristics seen in the radium patients. In other words, this is not diagnostic of radium, but it is very characteristic, and there are very few other medical conditions which will produce the same picture. I might say other nonmalignant medical conditions.

Chairman DURHAM. Doctor, did any of these patients in this group develop cancer from the normal treatment of other diseases from this radium?

Dr. LOONEY. I want to go into the number of tumors that have developed in these people, sir. I could not say whether these developed tumors from other conditions. All I could say is that a very large number of these people developed bone tumors, and out of proportion to other groups of people.

Of these 78 patients, 15 people have developed tumors.

If you will refer to figure 14, you will see the distribution of tumors that have occurred in these patients. You will notice that they usually occur at the end of the long bones, in the same areas in which you see these areas of abnormal bone formation.

I would like to read from that portion of the statement (p. 1170) entitled "Bone Tumors."

The 15 malignant tumors which developed in the 78 patients recently evaluated were found in individuals containing from 0.5 to 10 micrograms of radium in their bodies. The patient having 0.5 microgram of radium was a luminous-dial worker. It is reasonable to assume this patient ingested mesothorium and radiothorium. The patient with the lowest radium concentration, who had received radium medically, had 0.9 microgram of radium. The patient with the lowest concentration of radium, which was considered not to be contaminated with members of the thorium decay series, and who had developed a bone tumor, had 3.6 micrograms of retained radium.

It is important to emphasize that the luminous-dial workers used not only radium, but mesothorium and radiothorium. We have only measured radium, so we have not taken into consideration the radiation dose from either mesothorium or radiothorium.

The accumulated radiation dose with 3.6 micrograms of radium was estimated to be about 5,000 rads during the 25 years from radium administration until tumor formation.

Some of these people were selected because of symptoms. The number of bone tumors in the patients which were not discovered because of symptoms were located in a review of the records. We find that there is a frequency of about 2 percent in this unselected group as compared to a frequency of 14 percent of the entire group of 78 people. Although this is not conclusive, I do think it should be considered as it suggests this may be a biased group of people with whom we are dealing.

Because of the major interest in the possibility of bone tumor induction by radiation, I have taken two comprehensive articles in which the investigators have summarized the cases in which bone tumors have been produced following external radiation.

Vaughan in 1956 reported 39 cases of sarcoma arising in bone following external radiation have been recorded in the literature. The latent period between receiving radiation and the development of the tumor was 3 to 11 years. The radiation dose was not known in all of these cases; however, in most recorded cases it was estimated to be usually greater than 3,000 roentgens (1,500 to 7,000 roentgen range).

Cruz et al. in 1957 reported an additional series of 11 cases in which the bone tumors developed from 4 to 24 years after external radiation. The total radiation dose given ranged from about 1,000 roentgens to 5,000 roentgens, and was given over a period varying from 1 month to 9 years.

I should like to comment now on the possibility of bone-tumor production from strontuim 90, based on this information of tumor induction following external and internal radiation in man.

The assumption is often made that the incidence of the effect of strontium 90 is proportional to the magnitude of the dose. This assumption has been used to estimate the bone tumors which may be produced from the low concentrations of strontium 90 in the skeleton from fallout. There is no exact evidence either proving or disproving this assumption, but there is some clinical evidence which suggests that this assumption is overcautious.

The 50 bone tumors which have been known to have been produced in man from external radiation, and reported, were summarized in the previous section. The skeleton in the localized area of tumor induction received at least 1,000 roentgens, and usually more than 3,000 roentgens of radiation. If it is assumed that the radiation dose to bone is greater by a factor of 2 than the measured skin dose—in other words, the radiation going into the bone would absorb more radiation than the dose measured at the skin. So we are assuming it may be more than a factor of 2, maybe greater than this, but we are saying this as a reasonable estimate—then the minimum observed carcinogenic dose from external radiation would be about 2,000 rads, with the majority of the tumors being produced by more than 6,000 rads of external radiation.

Representative COLE. I would like to ask Dr. Looney, Mr. Chairman, to explain his statement that there is some clinical evidence which suggests that the assumption is overcautious, the assumption being that the incidence of the effect of strontium 90 is proportional to the dose. Now what do you mean when you say "there is some

clinical evidence which suggests that this assumption is overcautious." In what way?

Dr. LOONEY. I am saying that the minimal carcinogenic dose that we have reported for tumors to be produced in man is in the order of 2,000 rads. Based on the present clinical evidence we have today, we cannot prove or disprove that smaller doses would produce tumors, but I am just presenting to you the present evidence we have in man known to produce tumors. I am just saying that this assumption might be overcautious.

Representative COLE. What do you mean by "overcautious"? That is what I want.

Dr. LOONEY. May I come back to this at the end of these comments? Maybe this will clarify this statement.

What I have attempted to do is to present the order of magnitude of radiation we are dealing with. The following estimates on strontium 90 are based on the assumption that strontium 90 will be present in the body over a life span of 70 years. If we assume it is in equilibrium with bone or there is a constant intake of strontium 90, it would tend to approach a uniformity in bone.

Estimates which we can make from this information about the levels of strontium 90 necessary to produce bone tumors, skeletal roentgenographic changes, and total skeletal radiation from background radioactivity are as follows: Ten microcuries of strontium deposited in the skeleton for 70 years would give an estimated dose of about 2,000 rads. This is the minimum radiation dose recorded which has produced a bone tumor in man. This should give some idea of the magnitude of strontium levels which may produce bone tumors in man. You will notice that 6,000 rads is the estimated amount of radiation known to produce most tumors. The amount of strontium 90 which would deliver 6,000 rads to the skeleton over a life span of 70 years would be in the order of 30 microcuries.

The minimum radiation known to produce tumors in man is in the order of 2,000 rads, which would be 10 microcuries. The present MPC for industrial workers is 1 microcurie, and this would give 200 rads over a 70-year period. (The tenth of a microgram of radium would also give about 200 rads.) The total dose from natural radiation, based on the estimates of Dr. Robert Dudley of Massachusetts Institute of Technology, from all sources of radiation to the skeleton over a life span of 70 years, would be in the order of 10 rads. The sunshine unit, 1 times 10 to the minus 3 microcuries of strontium, would deliver about two-tenths of a rad to the skeleton in 70 years.

I will leave this up on the board so you can refer to it as the order of magnitude, and I will try to keep all of the units in rads so that this will give a basis for comparison.

I should like to go back to my statement in the section titled "Comments on * * * Bone Tumor Formations * * *." (See p. 1171.)

The patient with the smallest total body radium known to induce tumor formation, in which the possibility of contamination of the thorium series is unknown, died from a bone tumor in 1952. The estimated total body radium was 0.9 microgram. The time after administration is unknown, however, it is reasonable to assume that it was about 25 years. Based on the estimates above, the patient would have received a total accumulated does of about 1,800 rads during the 25-year period.

Histopathological changes have been demonstrated by roentgenographic examination of the skeletons of radium patients prior to development of the tumors. It is generally considered that the bone tumors developed in or around areas of atypical osseous tissue formation. Bone tumors have been shown to develop in or around the abnormal bone formation in animals given plutonium, strontium, and other radio elements.

If it could be shown that these histopathological changes are preliminary steps to bone tumor formation, then it could be assumed that as long as the body reparative processes prevented the abnormal bone formation, bone tumors would not develop. It should be emphasized that the associated histopathological changes seen prior to bone tumor formation in the radium patients may be coincidental findings. Proof of a correlation must await a better understanding of tumor induction by radiation.

Now there is one other bit of information in which I must emphasize little reliance can be placed. However, it is the best available evidence we have in man. This information pertains to the latent period of tumor development in relationship to the magnitude of the dose.

In reviewing the latent period for tumor induction in the luminous dial workers reported by Martland in 1931, it was found that the latent period for tumor formation was about 5 to 10 years in 6 patients who developed tumors. Only 3 of these patients had estimates of total body radium reported, and these estimated were 6, 15, and 50 micrograms.

The 8 luminous-dial workers who were examined in the recent Boston-Chicago investigations, and who died from bone tumors, lived for an average of 25 years after beginning employment. The average total body radium was 3.4 micrograms—range of 0.5 to 10 micrograms.

We have very meager data, but there does seem to be an inverse relationship between the latent period and the radiation dose necessary to produce bone tumors. The estimates based on this would be in the same order of magnitude as the estimates we have by the known radiation dose that produces bone tumors in man.

Therefore, the available clinical information we have at present indicates that the radiation dose for bone tumor production in man from both internal and external radiation is in the order of 2,000 rads.

The present MPC for radium, as I mentioned previously, was established in 1941. The fact that we are finding changes now at four-tenths of a microgram, which is four times greater than the MPC, is probably offset by the confidence in the larger group of patients which we have studied for longer periods of time. It is reassuring that it has not been necessary to change the MPC of radium over this long period of time, in view of the large amount of information that has been accumulated since it was established.

I have listed some of the factors which may permit the raising of the present levels for strontium 90. As I have pointed out, estimation of the mesothorium and radiothorium content in luminous-dial workers is being carried out. If it is found that the mesothorium and radiothorium contribute significantly to the dose, this would permit raising the present levels.

The characteristic changes we have seen may occur in the normal population, and further studies may permit us to obtain a better

understanding of how these changes are produced, thereby permitting a raising of the MPC.

As I mentioned in the introduction, most of these people were studied beginning about 15 or 20 years of age, so we have a gap in our knowledge of the younger age groups, which might necessitate lowering the present level. The present MPC is based on our present methods, and knowledge of the clinical changes produced by radioactive materials. It is possible that other subclinical effects may occur which may necessitate lowering the MPC.

In summary, it is considered that the best estimates which can be made in regard to the effects of strontium 90 over a life span of 70 years on the present incomplete information on the effects of radium in man are as follows:

The skeletal content of strontium 90 necessary to produce a bone tumor in a life span of 70 years would be in the order of 10 microcuries of strontium.

The skeletal content of strontium 90 necessary to produce significant changes, such as destruction of the hip would also be in the order of 10 microcuries of strontium over a life span.

The skeletal content of strontium necessary to produce minimal skeletal changes, which were demonstrated roentgenographically, would be in the order of 2 microcuries of strontium.

It should be emphasized again that these estimates of the concentrations of strontium 90 which may produce skeletal damage are the result of estimates based on the available information at present.

Representative HOLIFIELD. Have you finished, Dr. Looney?

Dr. LOONEY. Yes.

Representative HOLIFIELD. Are there any questions of Dr. Looney?

Representative VAN ZANDT. Mr. Chairman?

Representative HOLIFIELD. Mr. Van Zandt.

Representative VAN ZANDT. Dr. Looney, you have spelled out for us the effect of radium on the skeleton and explained how it produced bone cancer. Has radium in any way, shape, or form affected the blood to the point of developing leukemia or arthritis?

Dr. LOONEY. As I mentioned, the hematological changes in these people have been minor. In one case the patient died from renal insufficiency and there was a question of leukemia, but I would have to look this up.

Representative VAN ZANDT. What about arthritis?

Dr. LOONEY. Arthritis—I suppose the effects of radium might be considered a destructive form of arthritis. These people get destruction of the hip so this is, in a sense, a form of destructive or degenerative arthritis.

Representative VAN ZANDT. Dr. Looney, to your knowledge, has radium produced any other effects to the human body other than bone cancer?

Dr. LOONEY. That I know of, no sir. The major effects are the skeletal changes, and the bone tumors.

Representative HOLIFIELD. Dr. Looney, your presentation has been very valuable, and particularly the well-documented written presentation, which will be included in toto in the record.

I appreciate particularly having the pictures of the bone structures that are represented by your clinical and experimental experience in the record.

Thank you very much.

(The prepared statement together with "A Study of the Dynamics of Strontium and Calcium Metabolism and Radioelement Removal" submitted by Dr. Looney follows:)

THE BASIS FOR THE PRESENT MAXIMUM PERMISSIBLE CONCENTRATION FOR RADIUM AND ITS RELATION TO THE MAXIMUM PERMISSIBLE CONCENTRATION FOR STRONTIUM 90

A statement perpared for the Joint Congressional Committee on Atomic Energy on the subject "The Nature of Radioactive Fallout and Its Effects on Man," June 4, 1957, by William B. Looney, M. D.,[1] the John Collins Warren Laboratories of the Huntington Memorial Hospital of Harvard University, Massachusetts General Hospital, Boston, Mass.

INTRODUCTION

Man is constantly confronted with toxic agents in his environment, which, if present in sufficient quantities, may produce temporary or lasting changes. By the accumulation of clinical and experimental information about the quantity of these toxic agents which produce minimal changes, safety measures are established. The present maximum permissible concentrations for radioelements in use today are based primarily on the results of clinical and investigative studies made over the past half century.

The principal sources of material for the study of the effects of internally deposited radioactive materials in man have been individuals who were employed in the luminous dial industry and persons who received radium for medical purposes. Radium salts had been given orally and intravenously for hypertension, arthritis, anemia, and other medical disorders from about 1915 to 1930. The painting of watch dials with luminous materials containing radium, mesothorium and radiothorium started in this country in about 1914. Adequate safety measures were not taken until about 1925–27, following the deaths from bone tumors, anemia, and crippling bone lesions which occurred in some of these workers (41).

The first report in regard to the maximum permissible concentration (MPC) for radium was made by the National Bureau of Standards in 1941 (9). Seven individuals, having from 0.02 and 0.5 micrograms of radium in their bodies from 7 to 25 years had no noted changes referable to the deposition of radioactive materials. However, death had occurred in patients having as little as 1.2 micrograms of radium. The maximum permissible level of body burden for the amount of radium which remains after early rapid elimination was considered to be 0.1 micrograms.

Two investigations of 50 radium patients and 28 luminous-dial workers were made in Boston and Chicago and recently reported (3, 25). As a result of these two investigations, greater reliance can be placed in the MPC for radium. These investigations have given information about the effects of radium deposited for long periods of time in quantities at or near the present MPC. One of the most important results of the recent investigations is that a relationship between an objective clinical finding (skeletal roentgenographic abnormalities) and the physical estimate of total body radium could be made within a specific dose range.

This report is a condensation of a review article published recently in the Journal of Bone and Joint Surgery (27), which summarizes the results of studies made on the radium patients and the luminous dial workers over the past 40 years.

The clinical course following the deposition of varying amounts of radium has been divided arbitrarily into two categories in this report.

A. Patients with total body radium content at or near the MPC of 0.1 micrograms with either no detectable clinical effects or minor skeletal changes

Thirteen of the seventy-eight patients studied in the Boston-Chicago investigations had between 0.01 and 0.4 micrograms of radium, and with the exception of 2 cases listed below, none had either symptoms or skeletal changes which

[1] Clinical and research fellow in medicine, Harvard Medical School; visiting fellow in physics, Massachusetts Institute of Technology; special U. S. Public Health Service fellow of the National Cancer Institute.

could be attributed to radium. One patient with 0.4 micrograms of radium, age 83 years, was incapacitated with arthritis, therefore it was difficult to differentiate between the skeletal changes which may have been produced by radium from those which may have been produced by the skeletal disorder and disuse atrophy. Roentgenographs of the teeth of the other patient demonstrated areas of resorption in the teeth similar to those seen in other patients with greater amounts of radium. This patient was 32 at the time of examination. He had received radium water as a tonic between the ages of 8 to 10 years. The total body radium was only 0.15 micrograms of radium. It was considered that the early age of administration of radium may account for the dental changes with only a 50 percent larger radium content than the MPC of 0.1 micrograms.

B. Chronic effects of radium

1. Patients studied during the first and second decades following the initial medical and industrial use of radium (1915–35).—These patients were symptom free for about 10 years (31). Following the latent period, various changes relating to the skeleton began to develop. Death usually occurred from tumors of the skeleton, anemias or crippling bone lesions, or all three. Twenty-five of these cases were reported in the two decades which followed. Estimates of the radium content of the body began to be reported on these patients; however, the reliability of these estimates is hard to ascertain. These estimates ranged from about 5 to 100 micrograms of radium.

2. Patients studied during the third and fourth decades following the initial medical and industrial use of radium (1935–55).—Sixty-five of the seventy-eight patients evaluated in the recent Boston-Chicago investigations, having between 0.5 and 23 micrograms of retained radium, had either roentgenographic changes or symptoms, or both, which could reasonably be attributed to radium. The exceptions to this were three patients having betwen 0.5 and 1 microgram of radium having no skeletal abnormalities demonstrated roentgenographically. There was no proportionate increase in either the frequency or severity of the roentgenographic changes and symptoms with increasing amounts of radium in the body. The wide variation in the clinical abnormalities is consistent with the hypothesis that the changes observed are the result of a dynamic relationship between the reparative processes of the body and the destructive processes of the body acting in conjunction with the releterious effects of radium.

The mean age of the 28 luminous dial workers in the Boston-Chicago study was 21 years at the time of employment, and the mean age of the 50 patients at the time of radium administration was 34 years. The average time of retention was about 25 years in both groups, with a range of about 10 to 35 years. The average age of the patients in 1951 was about 50 years (range from 40 to 80 years). Seven of the 50 patients given radium have died; 5 died from tumors, 1 from anemia, and 1 from multiple causes; 11 of the 28 luminous dial workers are dead; 8 died from tumors, 1 from broncho-pneumonia, 1 from a urinary disorder, and another from renal insufficiency and leukemia. The average age at death for the radium patients was about 65 years, and the average age at death for the luminous dial workers was about 45 years. The average time of deposition of radium until death of the 18 patients was about 25 years, and average total body radium was about 5 micrograms.

As far as we have been able to determine, 10 of the 50 radium patients and 16 of the 28 luminous dial workers were discovered because of symptoms. It is apparent that these patients represent a biased sample from the population of radium patients and luminous dial workers. Correction for this biased sampling should be made in any attempt to relate the frequency of bone tumors and bone destruction in these patients with estimates of the frequency of these changes with comparable amounts of strontium 90.

RADIUM ELIMINATION

Radium is considered to be dispersed throughout the soft tissues and the skeleton when it first has been taken into the body (43). Most of the radium is eliminated during the first week (42, 43) (fig. 1). Fecal excretion is the major route of elimination in early and late cases. From 90 to 97 percent of the radium which is eliminated is eliminated in the feces and only 3 to 10 percent is eliminated in the urine (2, 35, 42). It has been reported that the rate of elimination following inhalation or ingestion is more rapid than that following intravenous administration (40, 42). Results from recent studies, however, indicate that these differences in the rate of elimination are not so pronounced as it was previously thought (26, 30, 35). Within a matter of

months, 90 to 99.9 percent of the radium is eliminated (40, 43). The age, the total amount of radium injected, and the estimates of the injected dose in 19 patients at 6 months, 12 months, and 20 years, have been reported (35). The average amount of the injected radium remaining at the times these estimates were made was 4.7 percent, 2.2 percent, and 0.8 percent, respectively.

The amount of the remaining radium eliminated daily becomes less and less (26). The available clinical data on the coefficient of elimination [2] of radium are given in figure 1. The marked change in the coefficient is elimination at 1 week has been considered to be the result of the elimination of the principal portion of radium from the soft tissues and the gastrointestinal tract and the change at 1 year has been considered to be the result of the elimination of the principal portion of radium from the more accessible parts of the skeleton. It is reasonable to conclude that the decreasing rate of elimination of radium is from the more inaccessible parts of the skeleton after 1 year.

About 15 to 35 percent of the absorbed radium is eliminated the first day. The amount of the remaining radium eliminated daily after 1 week is less than 1 percent. After about 10 years, it varies from 0.002 to 0.009 percent of the body content. The coefficient of elimination after about 20 years varies from about 0.002 to 0.016 percent of the body content (35, 43).

THE MANNER OF RADIUM DEPOSITION IN THE BODY

A. Autoradiographic study of radium deposition

Entire bones were sectioned serially and a comprehensive picture of distribution throughout the entire bone was obtained by placing the sections on roentgenographic film (3, 23). The precise manner of the deposition of radium was obtained by detailed autoradiography (1, 3, 23). Histological sections were covered with a photographic emulsion. After the emulsion and staining had been developed, simultaneous study of radium deposition and histopathological change was made.

Radium was found in small areas of high focal concentration irregularly distributed in both compact and cancellous bone. In compact bone only a small percentage of the Haversian systems and interstitial lamellae had appreciable concentrations of radium. In some instances radium was concentrated in 1 or 2 concentric lamellae, in other instances it was deposited around the central canal or periphery of the Haversian system.

The areas of radium concentration in travecular bone were usually 5 to 15 micra in the greatest dimension. However, there was a wide range in size and shape of these areas, and they were found at any depth within the trabecula. In some instances linear concentrations ran parallel to the curvature of the trabecula for 50 to 100 micra. Heavy and rather uniform concentrations of radium were present at the junction of the articular cartilage and the traveculae of the long bones in the gross autoradiographs. In the study by gross autoradiography it was found that frequent small highly concentrated areas resulted in outlining the bone contours (fig. 8). Some cementing lines were clearly outlined by heavy concentrations of radium.

In some sections exposed for long periods of time there was a much less concentrated and a much more uniform distribution of radium. These findings are in agreement with existing theories that radium has more than one principal mode of deposition. The small highly concentrated areas may have been areas in which bone formation was taking place at the time of administration or redistribution. The more uniform and less dense distribution may be the result of inorganicion exchange (23). (See figs. 2, 3, 4, 5.)

HISTOPATHOLOGY

The histopathological changes which are most important diagnostically are: (1) the formation of an atypical osseous tissue in the trabecular spaces of cancellous bone, and (2) well differentiated areas of destruction in compact bone.

Martland divided the changes in the skeleton into three stages, all of which are primarily concerned with the bone marrow (31). In stage III, however, he did state that bone absorption and considerable decalcification did occur along with the replacement of bone marrow and noncellular fibroblastic tissue.

---

[2] The coefficient of elimination is equal to the amount of radium eliminated per unit time (days) divided by the amount of radium in the body at the time the elimination measurements were made.

Studies of the skeletal changes produced in animals following the deposition of radioactive elements clarify the mechanism of production of the histopathological changes that have been found in human material. Heller and Bloom and Bloom concluded from their studies of such bone-seeking radioactive elements as strontium, yttrium, plutonium, and radium that similar changes were produced by all of these radioactive elements.

Following radium injections in mice the most spectacular change was a dense atypical bone in the metaphysis. This started from the proliferation of spindle cells which formed a dense fibrous tissue. Areas of calcification occurred in this fibrous tissue to form an atypical osseous tissue. It was also found to lesser degree in the shafts of the long bones and in the vertebrae. This was accompanied by the disappearance of osteoblasts, swelling and degeneration of cartilage cells, and death of most of the osteocytes. Repair began along with the destricutive effects of radium. In the lower dose ranges, reversible changes occurred so that in a matter of months normal bone was found on histological examination. Larger amounts of radium producted more severe and lasting changes. The epiphyseal cartilage varied tremendously in width, and the entire metaphysis was abnormal in appearance. The atypical osseous tissue remained in these animals at all intervals up to the termination of the experiment at 5 months.

The femoral shaft contained varying degrees of empty lacunae and dead oseocytes. The bones of the shaft varied from the relatively smooth contour to greater or lesser irregularities on the endosteal surface with projection into the marrow cavity.

Historical specimens have been obtained from 3 luminous-dial workers and 6 patients who had received radium. The radioactive elements had been present in the skelton from 12 to 35 years preceding biopsy and autopsy. Two luminous-dial workers and two patients who had received radium had bone tumors.

The histopathological changes were similar in certain respects to those changes found by Martland, Heller, and Bloom and Bloom (4). Atypical osseous tissue was present in cancellous bone. It was usually found near the articular surface of such bones as the humerus and femur, in addition to being found in the metaphyseal area. The atypical osseous tissue was laid down adjacent to the trabeculae in some areas. In some areas there was hyperplasia of the trabeculae, while in others destruction of the trabeculae was present. The trabecular spaces were usually filled with an acellular fibrous tissue. In general there was an absence of radioactivity in the atypical osseous tissue and accellular fibrous tissue was found in the autoradiographic study. In most cases, there was a prominent absence of osteocytes in the lacunae, and there was little evidence of bone regeneration.

In compact bone, the central canals showed a wide variation in size from normal to the nearly complete destruction of the entire Haversian system. A large number of the central canals were occluded with a dark staining material similar to the atypical osseous tissue in the trabecular spaces. There was usually an absence of cells in the lacunae. Minimal evidence of bone regeneration was observed, and areas of destruction usually were replaced with fibrous connective tissue.

In addition to the microscopic areas of destruction, macroscopic areas of destruction have been observed in compact bone 1 to 2 millimeters in width and 5 to 20 millimeters in length. Radium concentrations were rarely found in or around the macroscopic areas of destruction. Areas of transition in which both radium concentrations and microscopic change appear together have been found rather infrequently.

It has been shown that a correlation exists between the frequency of destructive changes and the amount of radium deposited in the body. It has been postulated that these macroscopic areas of destruction occur as the result of the fusion of adjacent central canals of Haversian systems undergoing destructive changes. It has been further postulated that the radium had been removed from the macroscopic areas of destruction by the time the changes occurred. If direct irradiation was the primary mode of production of the skeletal changes, more areas of transition should have been found in which both radium deposition and skeletal histopathological changes were present. It is evident that considerable difficulties are inherent in any attempt to reconstruct a pathological process which has been going on for 20 to 30 years from specimens taken at the termination of the process.

As a result of these observations, it is considered that radium deposited in the skeleton usually initiates a sequence of events which eventually produces patholog-

ical changes. These changes are probably the end result of many intermediate factors such as trauma, damage to blood supply, hormonal imbalance, decreased bone repair, and increased bone destruction from other causes. Evidence from the histological sections indicates that Haversian systems may undergo periods of resorption followed by periods of bone formation. There was a subnormal appearance of the bone in many instances. Some of the destructive changes may be the result of the inability of bone to maintain normal repair. It is evident that the relationship between radium deposition and skeletal change is complex. It is reasonable to conclude that when the destructive effects of radium and other deleterious intermediate factors become greater than the reparative processes of the skeleton, permanent alternations occurs (see figs. 6, 7, 8, 11, and 12).

HEMATOLOGICAL FINDINGS

In 1924, Castle, Drinker, and Drinker (8) reported the hematological findings of 22 luminous-dial workers. The erythrocyte count was below 4 million in 6 percent of these cases and above 6 million in 19 percent. The white-blood-cell count was below 7,000 in 27 percent of the workers. Abnormal erythrocytes occurred in 36 percent of the series. There was an increase in lymphocytes and monocytes and decrease in the polymorphonuclear neutrophils found on differential examination.

In 1943, the Public Health Service (41) made a study of 196 employees of luminous-dial-painting plants. Anisocytosis was found in 11 percent of these people, and poikilocytosis in 8 percent. The average erythrocyte count in the Public Health Service series was 4,300,000 as compared with an average of 4,500,000 in a series of 31 control patients.

Hematological data were available on four patients who died. Two of these patients died from malignant tumors—patient (R–24) and patient (B–7). Patient (R–44) died from aplastic anemia and patient (L–27) died from secondary infection and debilitation.

The marked pancytopenia that occurred in Martland's cases had not occurred in the patient evaluated in 1951. A short time before death, there were usually about 3 million erythrocytes per cubic millimeter and 10 grams of hemoglobin per 100 cubic centimeters. No hemorrhagic manifestations occurred in these patients. Some of Martland's cases had less than 1 million erythrocytes and less than 1,000 leukocytes per cubic millimeter shortly before death. He considered the anaemia which developed in his patients to be of a regenerative type resembling pernicious anemia, and he described the hematological changes in the following way: "The blood in this case showed a profound anemia, characterized by a large cell anisocytosis, by the presence of megaloblasts and by a marked leukopenia. There was not, however, a hyperbilirubinemia, and the van den Bergh tests were negative. The anemia is, therefore, not a hemolytic anemia. It is not an aplastic anemia, since there is marked embryonal blood formation. The fault lies in a long continued irritation of the hematopoietic system, the hemolytic or reticuloendothelial system being unaffected. There is a stage of stimulation followed later by sudden exhaustion of the erythroblastic and leukoblastic centers with the production of a rapid, fatal anemia with leukopenia, which fails to be influenced by any form of treatment." Red regenerative marrow was present in the femora of some of these patients.

These changes in the hematopoietic system are similar to those seen in the acute radiation syndrome. Most patients who lived less than 6 weeks following external radiation had hypoplastic marrow. Some patients who died at 4 to 5 months after exposure had diffuse myeloid hyperplasia which involved even such long bones as the femur. Occasionally the marrow appeared pink and gelatinous.

The bone marrow of patient (R–24) was studied in 1948, 18 years after radium administration. It was reported to be distinctly overactive. There was moderate erythroblastic activity with an excessive number of cells in mitosis without a shift to the left. The remainder of the cells were normal. The erythrocyte count was 5,300,000 per cubic millimeter and hemoglobin was 12 grams per 100 cubic centimeters at this time. The erythrocyte count was 3 million per cubic millimeter and hemoglobin was 9 grams per 100 cubic centimeters shortly before death in 1951. Small pink areas of hyperplasia was found in the femur and tibia at autopsy. Sternal marrow from patient (R–43) revealed extremely atrophic marrow shortly before her death in 1947. There were only small areas or erythropoiesis and the remainder of the marrow was composed principally of fat cells. No evidence of excess destruction, serious fat atrophy, or fibrosis

was observed. The hemoglobin at this time was 12.6 grams per 100 cubic centimeters and the erythrocyte count was 3,890,000 per cubic millimeter.

Patients who have over one microgram of retained radium are more likely to have anisocytosis, poikilocytosis, and hypochromia of the erythrocytes than those having under one microgram. However, significant hematological changes usually do not occur until late in the course of the disease.

In view of this additional information, it appears that little qualitative difference exists in the hematological response to internal or external radiation. If an individual had received large enough amounts of radioactive substances internally, hematological changes occurred which were similar to the hematological changes following exposure to large amounts of external radiation. In individuals who had received smaller amounts of either internal or external radiation, the hematological response at any given time was varied. The type and number of cells in the circulation at any specific time is the result of the natural survival of the cells and the balance between their radiosensitivity and their ability to recover from injury.

The red regenerative marrow that Martland described was in all probability an abnormal attempt of the hematopoietic system at increased production as a result of damage. Martland's term leukopenia anemia of regenerative type which was used to describe the hematological changes in the luminous-dial workers does not seem to be appropriate. The anemia that develops in these patients and the patients who had received radium would be more suitably placed in the category of primary refractory anemia in which either a hyperplastic or hypoplastic (aplastic anemia) bone marrow may be found. No leukemias have been found.

ROENTGENOGRAPHIC CHANGE

Areas of increased density have been found in cancellous bone on a skeletal roentgenographic examination. This roentgenographic change is the result of a typical osseous tissue in the trabecular spaces; infrequently it is the result of hyperplasia of trabeculae. Well-defined areas of decreased density were found in compact bone. These were the result of the destructive changes in the cortex of the bone.

These changes were divided into three groups, principally on a descriptive basis (24):

Group I: Areas of decreased density which were usually 1 to 2 millimeters in width and 5 to 20 millimeters in length in the long bones which gave a streaked appearance, and "punched-out" areas varying from 2 to 20 millimeters in the greatest dimensions present in the skull.

Group II: Areas of increased density usually associated with areas of decreased density with varying degrees of change in the trabecular pattern. These usually occurred in the femoral head, the humeral head, and the glenoid process, giving a mottled or motheaten appearance. There was an increase in frequency of biconcavities and collapse of the vertebras in patients having larger amounts of retained radium. Areas of increased density were found along the superior and inferior borders as well as small areas of increased density in the vertebral bodies.

Group III: The term "aseptic necrosis" has been used in referring to the changes in normal configuration. The heads of the femora, the bones of the feet, and the mandible were most commonly involved.

Serial roentgenograms of a few of these patients over long periods of time have been obtained. In most of the available serial roentgenographic studies, changes were not found until years after the deposition of the radioactive element. Periods of skeletal change may occur and may become stabilized or may improve. Later skeletal changes occur with increasing frequency.

For example, one patient (R–50) was given radium in 1922 (age 42 or 44). Ten years later skeletal roentgenographic changes characteristic of radium deposition occurred primarily in the mandible. These changes gradually improved. In 1940 (age 60) a significant increase in skeletal involvement occurred. There was a gradual increase in frequency and severity until her death in 1949. Roentgenograms of the skull of another patient (R–24) in 1948 demonstrated 12 areas of decreased density; the number of areas had increased to 22; however, there was minimal increase in the size of the areas. The size of these areas usually did not become greater than dimensions given in the description of the roentgenographic findings. Infrequently, however, they may be as great as 3 to 4 centimeters in diameter. The usual sequence of events was an increase in numbers, as well as an increase in the number of bones involved.

It is evident from these and other serial roentgenograms that these changes do not bcome detectable until many years after the deposition of radioactive elements. The possibility of skeletal histopathological changes being produced and remaining undetected reontgenographically for years must also be considered. There were no roentgenographic changes found on complete skeletal roentgenographic examination of patient (R–13) in 1950, with the exception of an aseptic necrosis of the right femoral head. However, minor histopathological changes characteristic of the deposition of radioactive elements were found in sections of the left fibula of this patient. In 1954, roentgenographic changes characteristic of the deposition of radioactive elements were present in the fibula and other long bones.

It has been shown that skeletal histopathological changes are reversible in animals following the deposition of radioactive elements. The history of another patient (R–50) demonstrates that skeletal changes as seen by roentgenogram are reversible in man following the deposition of radioactive elements. It is reasonable to assume, therefore, that reversible skeletal histopathological changes occur in man, as well as in animals, following the deposition of radioactive elements. When the reparative processes are able to be maintained at a level equal to the destructive processes, no detectable changes occur as seen by roentgenogram. If the ability to maintain skeletal repair is impaired or decreased, more and more permanent alterations probably result from the imbalance between the reparative and destructive processes.

Histopathological changes have been present to such a degree that roentgenographic changes have been found in many patients who are symptom-free. However, significant clinical changes usually do not occur in the absence of skeletal roentgenographic change. The results of skeletal roentgenographic, autoradiographic, and histopathological studies made on serial bone sections indicate that repeated skeletal roentgenographic examinations are the most satisfactory clinical methods for the early detection of skeletal alterations following the deposition of radioactive elements.

Thirty of the patients who were given radium for medical reasons and two luminous-dial workers were selected because each of these patients had had a complete skeletal roentgenographic examination. They were arranged in order of increasing amounts of retained radium from 0.5 to 14 micrograms, and the skeletal changes characteristic of the deposition of radioactive elements were tabulated. These patients were arbitrarily divided into three groups, those patients having between 0.5 and 1 microgram of retained radium, those having between 1.1 and 2 micrograms, and those having between 2.1 and 14 micrograms. Table I gives the age and sex of 32 patients 21 years after the deposition of radioactive materials.

Forty individuals were selected at random while undergoing physical examination at the Argonne National Laboratory for skeletal roentgenographic examination to act as a control group. It would have been desirable to have matched controls; however, the results of these roentgenographic examinations greatly minimized the possibility of the minor changes being present to any significant degree in the general population.

Five luminous-dial workers having between 0.02 and 0.4 micrograms of radium and 4 radium patients having between 0.01 and 0.4 of radium had complete skeletal surveys. One 83-year-old patient (0.4 micrograms of radium) had roentgenographic changes characteristic of radium deposition. The interpretation of these changes was difficult because of the marked arthritic abnormalities. Another patient with 0.15 micrograms of radium had areas of resorption of the teeth.

The five bones (skull, radius, ulna, tibia, and fibula) which were most frequently the sites of decreased density were first tabulated (chart I). The frequency of involvement was expressed as a percentage of the total number of bones that could possibly be involved. No distinction was made between unilateral or bilateral involvement, since the long bones were involved bilaterally in all but about 10 to 20 percent of the cases.

For example, in the 15 patients having between 0.5 and 1 microgram of radium, a total number of 75 bones could be involved; only 6 bones had changes characteristic of radium deposition or about 10 percent of the total number of bones. In group II, about 55 percent of the bones had characteristic changes, and in group III about 65 percent of the bones were involved.

Attempts were made to find some correlation between the severity of bone changes and increasing amounts of radium. The skeletal changes were divided into six grades of severity (27). With increasing amounts of retained radium

in the body, a proportional increase in roentgenographic changes does not occur. In addition, there is a marked individual variation in the amount of retained radium in the body. In some patients who had the greatest amounts of retained radium, only minor changes occurred, while other patients who had only one-fifteenth to one-thirtieth as much had more severe skeletal changes. Chart II graphically demonstrates this clinical observation in regard to the roentgenographic changes. Patients in group III had about 5 times as much radium as those in group II; however, there is only a 10 percent increase in the frequency of involvement. This lack of correlation between group II and group III is further emphasized by the fact that the average age of group III is 60 compared with an average age of 50 in group II. This finding and the finding of a significant increase in roentgenographic changes and symptoms at 1 microgram, even though the luminous-dial workers were 13 years younger, are suggestive that age at the time of deposition is not a major factor in the eventual production of roentgenographic changes. The fact that some of the patients had between 0.1 and 0.5 microgram of radium deposited for 25 to 30 years and had no roentgenographic changes is suggestive that time of retention per se may not be a major factor in the eventual production of roentgenographic changes. It is to be emphasized that these observations, suggesting that the time of deposition and the length of retention are not important, may well be the result of biased sampling of the patients, as well as an inadequate number of patients.

The most important result of these studies is that a definite correlation has been made between an objective clinical finding and the estimated amount of radioactive element retained in the body. For the first time, a semiquantitative relationship between the frequency of the roentgenographic changes and retained radium has been established within a certain dose range. No characteristic roentgenographic changes have been observed in patients having under 0.1 microgram of radium and relatively few changes have been found in patients having between 0.1 to 0.5 microgram of radium. Between 0.5 and 1 microgram of radium changes began to occur with increasing frequency and those patients having over 1 microgram of radium had a considerable increase in the frequency of the roentgenographic changes when compared with the patients having under 1 microgram of radium. (See figs. 9, 10, 11, 13.)

SYMPTOMS

Symptoms which can be reasonably attributed to radioactive element deposition usually result from destructive changes in the skeleton in patients with small amounts of radium. When symptoms do occur as a result of skeletal change, there are usually not severe in comparison with the extent of skeletal damage. In some instances, there is neither a progression of the skeletal lesions nor of the symptoms following the production of the skeletal change. For example, aseptic necrosis of the head of the femur in one patient developed in 1940 (age 30 years). This was followed by limping and discomfort on walking. However, during the period between the onset of symptoms and the examination in 1951, little progression of either limping or discomfort on walking occurred. There was also minimal discomfort following the aseptic necrosis of the head of the radius in another patient (R–23) since the beginning of symptoms in 1947. Another patient had aseptic necrosis of the head of the femur 22 years after the administration of radium at the age of 50 years (25, 27). She had no other symptoms or changes that could be related to the deposition of radioactive elements.

Skeletal changes usually occur in bones subject to weight-bearing or to repeated trauma (31). Aseptic necrosis usually occurs in the heads of the femora and the bones of the feet. Collapse of the veterbrae is occasionally found, while fractures almost always occur in the femoral shaft. One patient (L–27) fractured her left femur while pressing her foot to the floor of an automobile. Patients (L–14) and (R–35) sustained fractures of the shafts of the femur on minimal trauma. Healing of the fractures was delayed, but proper union occurred in all patients. Rarely, patients may have femoral fractures with permanent nonunion. Patient J. J. had collapse of some of the vertebral bodies with associated pain that required the wearing of a Taylor brace for control, while patient (R–44) had more marked vertebral changes with minimal discomfort.

Many of the patients did not have any symptoms which might be attributed to radium intake. Other than an increased risk of skeletal injury or tumor

formation, they may never have any recognized clinical changes as a result of radioactive element deposition.

In attempts to correlate clinical change with the amount of radium in the body, marked variations were found. The history of patient (R–49) illustrates this clinical observation. The only difficulty that he had was a march fracture of the foot which had healed uneventfully. The patient was asymptomatic at the time of examination in 1951 and only minor skeletal roentgenographic changes were present which could reasonably be attributed to the deposition of radioactive elements. Fourteen micrograms were present in his body, 15 to 30 times the amount present in some patients who had severe skeletal changes or tumor formation.

The time between the deposition of the radioactive elements and the onset of the first symptoms was analyzed in order to see whether any correlation could be made between this period and increasing amounts of retained radium. Twenty-one of the 25 luminous-dial workers had symptoms referable to radioactive-element deposition and 25 of the 50 patients who had received radium had symptoms that could reasonably be attributed to radioactive-element deposition.

The luminous-dial workers and patients who had received radium were divided into two groups. The first 10 patients of the luminous-dial workers who had symptoms and had between 0.1 to 1.5 micrograms of retained radium were compared with the 11 luminous-dial workers having between 1.6 and 18 micrograms of retained radium. The average time before symptoms occurred in the first group was 16 years and in the second group was 15 years. The 25 patients who had received radium who had symptoms referable to the deposition of radioactive elements were divided into 2 groups. The first 12 patients had between 0.7 and 4.2 micrograms of retained radium, the other 13 patients had between 5 and 22 micrograms. The average time before symptoms occurred in the first group was 17 years and in the second group was 16 years. There was no increase in the time interval before symptoms occurred in the first group, even though there was from about 10 to 100 times less radium in the body. It should be noted that the period of latency before the onset of symptoms that could reasonably be attributed to the deposition of radioactive elements varied from 1 to 32 years.

Since no relation between increasing amounts of radium and the time before symptoms occurred from deposition of radioactive elements was found, the frequency of involvement of the femur was examined to see if some correlation could be made with the retained radium. Nine patients who had received radium had aseptic necrosis of the head of one or both femora as did luminous-dial workers.

It is well known that aseptic necrosis of the femoral head usually occurs as a result of trauma or following fractures of the neck of the femur. It is also true that aseptic necrosis does occur in which the etiology cannot definitely be established. In these cases, it is considered that circulatory disturbances are the primary cause of the necrosis. The cause of the disturbance is poorly understood; it may be from gradual occlusion or from trauma (37). Unfortunately there is little information concerning the incidence of unexplained aseptic necrosis of the femoral head. Estimates of the occurrence of aseptic necrosis of the femoral head from all causes approximated 1 in 200 in an orthopedic practice.

The 14 percent incidence of aseptic necrosis of the femoral head in these patients seems unusually high in the absence of a history of antecedents trauma or fracture. It is interesting to note that all of the lesions occurred in patients having 0.7 microgram or more of radium in their bodies. The average time from the deposition of the radioactive elements until symptoms referable to the femoral head occurred was 15 years, approximately the same as that for symptoms in all other parts of the skeleton. The time of onset varied from 9 to 22 years.

Almost all of the fractures occurred in the shaft of the femur. Some of the patients fractured both femora, as well as sustaining refracture one or more times. Fractures following such minimal trauma as pressure of the foot on the floor of the car emphasize the fragility of the skeleton that may result from the deposition of radioactive elements.

Fractures of the femur occurred in 4 of the luminous-dial workers and in only 1 patient who had received radium. Again it should be noted that all of the fractures occurred in patients having 0.9 microgram or more of retained radium.

TUMOR FORMATION

The clinical course of patients in whom malignant tumors eventually develop is illustrated in the following case histories.

Patient (L–21) began to have symptoms as a result of skeletal changes 11 years after employment as a luminous-dial worker in 1934 (27). Several skeletal lesions occurred over a period of years following the deposition of the radioactive element. A biopsy specimen was taken from the left femur in 1950, following the clinical diagnosis of chronic osteomyelitis. The histological diagnosis at that time was a low-grade inactive fibrous osteomyelitis associated with recent slow-growing cancerous type of fibrous osteoma. In 1952 pain began to occur and to increase in frequency in the left femur, and the roentgenograms were interpreted as being suggestive of malignant changes in the medial condyle. The extremity was amputated and an osteogenic sarcoma was found throughout the femur on histological examination. The patient died a few months later.

Patient (R–24) had noted the onset of pain in the left foot 18 years (1948) after the administration of radium water (27). Roentgenograms of the foot a few months later revealed an area of decreased density in the left tarsal navicular. The condition became so severe that it necessitated the use of a cane. Roentgenograms at that time revealed marked destructive changes of the tarsal bones. Because of the marked increase in symptoms and the destructive changes in the foot in 1950, a biopsy was done and fibrosarcoma was diagnosed on histological examination of the specimen. The pain became generalized and required more and more analgesics to control; it was described as boring and burning in character. Intermittently, sharp knifelike pains occurred which lasted for a few seconds. The pain was not significantly influenced by motion, position, and temperature. The leg was amputated below the knee in February 1951. Examination of the amputated tibia revealed extension of the tumor. A sarcomatous lesion developed on the end of the stump a few weeks after the operation. The patient died in August 1951, as a result of generalized metastases. Areas of fibrosarcoma were found throughout the skeleton. In some parts of cancerous bone, it was difficult to distinguish between the fibrosarcoma and the fibrous connective tissue found in the trabecular spaces of patients without malignant involvement.

It is evident from the case histories of patients (L–21) and (R–18) that the transition from a benign to a malignant lesion may be similar to that in many skeletal diseases. Both of these patients were first considered to have osteomyelitis and then osteoid osteoma. In patient (R–17) the diagnosis of osteoid osteoma of the finger had been made 1 year prior to the diagnosis of osteogenic sarcoma. Characteristic histopathological changes due to the disposition of radioactive elements were found in reviewing the specimens from which these diagnoses were made.

The history of patient (L–8) emphasizes the need to follow these patients carefully. Patient has led a relatively normal life since 1934 as a result of prompt attention to symptoms occurring in the elbow. Following a biopsy and histological diagnosis of osteogenic sarcoma, the arm was disarticulated at the shoulder.

Figure 14 shows the sites of origin of the tumors that developed in 13 of the 78 patients. Eight of these patients were luminous-dial workers and five were given radium for medical purposes. Table II gives the age at the time of administration, the time from administration until the first symptoms occurred which could reasonably be attributed to the deposition of radioactive elements, the time from the first symptom until death, and the amount of retained radium. Since both groups of patients have approximately the same amount of radium retained in the body for about the same length of time it is not possible to make any differentiation between luminous-dial workers and patients who were given radium in relation to the estimated radium present. The average age at the time of employment in the luminous-dial workers was 17 years as contrasted with 39 years as the average age at the time of the administration of radium.

Most of the patients in whom malignant tumors eventually develop follow a similar course. There is a latent period followed by symptoms which usually developed from changes in weight-bearing bones and bones which are subject to repeated trauma. Later tumor formation occurs at a site which is usually not the site of the initial symptoms. When the malignant tumor develops, there may be a marked acceleration of the disease process and death usually occurs about 1 to 4 years after the symptoms began at the site where the tumor originated.

THE RELATION OF THE CLINICAL CHANGES TO THE AMOUNT OF RADIUM PRESENT IN THE SKELETON

A. Hematological changes

Minor changes occurred with greater frequency in the erythrocytes in patients having more than 1 microgram of radium than those patients having less than 1 microgram. The marked anemias found in the earlier cases were not present in the 78 cases studied recently. Two patients developed anemias a short time before death. Only one leukemia has been reported.

B. Roentgenographic changes

One of the most important results of the recent Boston-Chicago investigations was the ability to correlate the frequency of occurrence of roentgenographic changes characteristic of radium deposition with the physical estimates of total body radium. Thirty-two patients [3] having complete skeletal roentgenographic examinations were arranged in order of increasing amounts of radium and the changes tabulated (review section on roentgenographic changes.)

C. Symptoms

Symptoms [4] which can reasonably be attributed to radium occur as a result of changes in the skeleton. Symptoms which could reasonably be attributed to radium did not occur in any of the 78 patients who had under 0.4 microgram of radium. Nine patients who received radium medically and two luminous-dial workers developed destruction of the femoral head. The average time for symptoms to occur was 15 years (range 9–22 years). All of the patients who had aseptic necrosis of the femoral head had 0.7 or more micrograms of radium in the body. The 5 patients who had fractures of the shaft of the femur had 0.9 microgram or more of radium in the body.

The results of these investigations indicate that the relation between the destructive effects of the radioactive elements remaining in the body for long periods of time and the clinical changes produced, is a dynamic relationship between the destructive and reparative processes of the body. For example, some of the patients had aseptic necrosis of the femoral head with less than 1 microgram of radium, while others, with 10 micrograms or more, had no deleterious effects. It is reasonable to assume that these patients had other disturbances in the femoral head, such as an unsatisfactory blood supply. Another example of this dynamic relationship was demonstrated in the patient who had increased changes during pregnancy which subsided with the delivery of the child. Still another possibility is that certain periods of increased bone destruction or reduced bone formation may occur from disease processes which later revert to normal. One patient had rather severe changes in the mandible which later subsided.

D. Bone tumors

The 15 malignant tumors which developed in the 78 patients recently evaluated were found in individuals containing for 0.5 to 10 micrograms of radium in their bodies.[5] The patient having 0.5 microgram of radium was a luminous-dial worker. (It is reasonable to assume this patient ingested mesothorium and radiothorium.) The patient with the lowest radium concentration, who had received radium medically, had 0.9 microgram of radium. The patient with the lowest concentration of radium, which was considered not to be contaminated with members of the thorium decay series, and who developed a bone tumor, had 3.6 micrograms of retained radium. The accumulated radiation dose with 3.6 micrograms of radium was estimated to be about 5,000 rads during the 25 years from radium administration until tumor formation.[6]

---

[3] One patient which was evaluated later with 0.15 microgram of radium had dental changes.

[4] Symptoms is a term used to denote subjective awareness of an abnormality. When skeletal changes progress to such a degree that destruction of the hip or fractures occur—symptoms are noted by the patient.

[5] See fig. 14.

[6] The estimates of continuous radiation dose is based on the assumption that 0.1 microgram of total body radium will deliver a dose of 3 rads per year to the skeleton. It was found in the radium excretion studies made in the Chicago investigations that the retention of radium could be expressed as an approximate power function of time (27). From this information Brues and Tyler derived an expression for the estimation of the cumulative radium dose from the estimation of the instantaneous dose rate at the time of measurement. Based on the equation of Brues and Tyler, the total cumulative dose would be approximately twice the dose estimated at the time of measurement.

Only 2 bone tumors have developed in the 52 of the 78 patients who were not discovered because of symptoms arising from the skeleton. The frequency of bone tumors in this group is 2 percent, in contrast to a frequency of 14 percent for the entire group of 78 patients.

Vaughan (45) (1956) reported 39 cases of sarcoma arising in bone following external radiation have been recorded in the literature. The latent period between receiving radiation and the development of the tumor was 3 to 11 years. The radiation dose was not known in all of these cases; however, in most recorded cases it was estimated to be usually greater than 3,000 roentgen (1,500–7,000 roentgen change).

Cruz et al (10) (1957) reported an additional series of 11 cases in which the sarcoma of bone occurred from 4 to 24 years after external radiation. The total radiation dose given ranged from about 1,000 roentgens to 5,000 roentgens, and was given over a period varying from 1 month to 9 years.

COMMENTS ON THE POSSIBILITY OF BONE TUMOR FORMATION FROM STRONTIUM 90

The assumption is often made that the incidence of the effect of strontium 90 is proportional to the magnitude of the dose. This assumption has been used to estimate the bone tumors which may be produced from the low concentrations of strontium 90 in the skeleton from fallout. There is no exact evidence either proving or disproving this assumption, but there is some clinical evidence which suggests that this assumption is over cautious.

The 50 bone tumors which have been known to have been produced in man, and reported, were summarized in the previous section. The skeleton in the localized area of tumor induction received at least 1,000 roentgens, and usually more than 3,000 roentgens of radiation. If it is assumed that the radiation dose to bone is greater by a factor of 2 than the measured skin dose, then the minimum observed carcinogenic dose from external radiation would be about 2,000 rads, with the majority of the tumors being produced by more than 6,000 rads of external radiation.

The patient with the smallest total body radium known to induce tumor formation, in which the possibility of contamination of the thorium series in unknown, died from a bone tumor in 1952. The estimated total body radium was 0.9 microgram. The time after administration is unknown, however, it is reasonable to assume that it was about 25 years. Based on the estimates above, the patient would have received a total accumulated dose of about 1,800 rads during the 25-year period.

Histopathological changes have been demonstrated by roentgenographic examination of the skeletons of radium patients prior to development of the tumors. It is generally considered that the bone tumors develop in or around areas of atypical osseous tissue formation. Bone tumors have been shown to develop in or around the abnormal bone formation in animals given plutonium (22). If it could be shown that these histopathological changes are preliminary steps to bone tumor formation, then it could be assumed that as long as the body reparative processes prevented the abnormal bone formation, bone tumors would not develop. It should be emphasized that the associated histopathological changes seen prior to bone tumor formation in the radium patients may be coincidental findings. Proof of a correlation must await a better understanding of tumor induction by radiation.

In reviewing the latent period for tumor induction in the luminous-dial workers reported by Martland in 1931 (31), it was found that the latent period for tumor formation was about 5 to 10 years in 6 patients in this study who developed tumors. Only 3 of these patients had estimates of total body radium reported and these estimates were 6, 15 and 50 micrograms. (See table II.)

The eight luminous-dial workers who were examined in the recent Boston-Chicago investigations, and who died from bone tumors, lived for an average of 25 years after beginning employment. The average total body radium was 3.4 micrograms (range of 0.5 to 10 micrograms).

It is considered that little reliance can be placed in estimating the minimum total body content for tumor induction over a life span from this meager data. However, if we do estimate the total body radium which would have a latent period for tumor induction greater than the average life expectancy (80 years)

from the 2 average radiation doses and latent periods given above, it would be 0.3 and 0.4 micrograms of radium respectively.[7]

If we assume that 0.5 micrograms of radium retained for 70 years would be the minimal body burden necessary to cause skeletal sarcoma, this would mean that a cumulative radiation dose of about 2,000 rads would be necessary to induce tumor formation. It is considered that the most reliable estimates of the radiation dose necessary to induce bone tumors over a life span of 70 years, should be equivalent to the total radiation dose necessary to induce carcinogenesis in 5 to 30 years (2,000 rads). Therefore, the estimates of the total radiation dose from strontium 90 necessary to cause bone tumor formation in man during a life span of 70 years will be based on the best available estimates from human data of about 2,000 rads. It may be calculated that 1×10^{-9} curies of strontium 90 will give a radiation dose to the skeleton of 3 millirads per year, or 0.2 rads in 70 years under equilibrium conditions. Based on the preceding assumptions, the total skeletal radiation dose from 1×10^{-9} curies of strontium 90 over a 70-year period would be in the order of one ten-thousandths total skeletal radiation dose known to produce tumors in man, either from external or internal radiation.

COMMENTS ON THE PRESENT MPC OF RADIUM

Results of the Boston-Chicago investigations have shown that clinical changes begin to occur in patients with 0.4 micrograms of radium. The fact that changes are found to occur in man with 4 times the present MPC of 0.1 microgram of radium instead of 12 times the MPC when it was established in 1941, is offset by the greater confidence in the MPC by the studies of a much larger group of patients with radium present in their bodies for longer periods of time.

It is reassuring that it has not yet been necessary to change the MPC of radium of 0.1 micrograms following the large amount of information that has accumulated since it was first established. There are certain considerations, which, if sufficient information becomes available, may permit raising, or may necessitate lowering, the present MPC. These are as follows:

A. Factors which may permit the raising of the MPC for radium

1. The contribution of the thorium series to the dose from radium.—It is well established that mesothorium I (half life 6.7 years) and radiothorium (half life 1.9 years) were used in the luminous-dial paints, and mesothorium has been found in patients given radium medically. The radiation dose from mesothorium in some of these patients has been significant. It has been the major source of the cumulative radiation dose in some of the luminous-dial workers.

The time relation in this question is very important. It is assumed that reversible changes are produced by the presence of mesothorium, then its major clinical effect should have occurred before the changes began to occur in these patients. Roentgenographic changes do not begin to occur until 15 to 30 years after the deposition of the radioactive elements. However, once changes begin to develop in these patients, there is usually a gradual increase in the frequency and severity rather than a decrease. If the changes produced during the period immediately after deposition are irreversible and contribute significantly to the changes that are being seen in these patients 20 to 30 years later, then it may be possible to elevate the MPC. Further work is in progress in an attempt to clarify the relationship of mesothorium, radiothorium, and radium in the clinical changes observed.

2. Controls.—Even though the roentgenographic changes found in these patients are characteristic of radium deposition, further control studies to estimate more reliably the incidence of these changes in the general population should be carried out. The skeletal roentgenographic findings may eventually prove to be the most reliable method for the early detection of clinical changes from the deposition of bone-seeking radioelements. A better definition of the normal limits of skeletal variation is needed, in order that a more precise evaluation of the skeletal changes in the radium patients may be made.

The destruction of the head of the femur is another clinical finding in these patients which emphasized the need for further control studies; 11 of the 78 patients had destruction of the hip. This frequency of aseptic necrosis is

[7] These estimates are consistent with the findings of animal data of Brues[6], in which the latent period was found to vary inversely with the square root of the dose. The value of 0.3 micrograms of radium is obtained from assuming that the latent period of 10 years is increased by a factor of 8, and the average radium content is 25 micrograms. The value of 0.4 micrograms is obtained from assuming that the 25 years' latent period is increased by a factor of 3, and the average radium content of the patient is 3.4 micrograms.

unusually high in the absence of a history of antecedent trauma. Proper evaluation of this condition in the radium patients must await a better understanding of the cause of aseptic necrosis, as well as more reliable information as to its occurrence in the general population.

B. Factors which may necessitate lowering the MPC of radium

1. Age at the time of administration and duration of retention of radium.—The average age of the 50 patients at the time they received radium medically was 34 years. The average age of the luminous-dial worker at the time of employment was 17 years. Almost all of these patients either received radium, or were exposed to radium from 1920 to 1930. It is evident that the clinical information on the effects of radium deposited in the body for more than 40 years is lacking. Since the findings of the recent investigations suggest that clinical changes are the result of an imbalance between reparative and destructive processes of the body, it may be found that small amounts of radium in persons of the older age groups may produce significant clinical changes. This may result from either diminished reparative abilities, or an increase in skeletal destruction from other debilitating diseases of the older age groups.

There are only a few patients available for study of the long-term effects of radium who received the radium before the age of 15 to 20 years. One of these patients had the smallest amount of total body radium in which dental change characteristic of radium deposition has been observed.

The problem of bone tumor production in the younger age groups is probably the most important question in this regard. The greatest incidence of osteogenic sarcoma is considered to occur in the second and third decades, and almost no clinical information is available on the effects of skeletal radium deposition before this increase in the incidence of bone sarcoma occurs. It is possible that the increased susceptibility to skeletal tumor induction might necessitate a significant lowering of the MPC for children.

2. Undected subclinical effects.—The present philosophy for the establishment of the MPC is based on the correlation of physical estimates of the quantity of the radioelement in the body with detectable clinical changes or abnormalities in clinical tests. As more refined tests are developed to detect abnormalities in body function, it may be discovered that the present MPC is producing changes which are not detected by present methods of clinical evaluation.

Another important consideration is that subclinical changes may cause a reduction in the reserve function of organs. This may go undetected in most instances. However, the combined effects of an intercurrent disease and the reduced function from the effects of radiation may cause more severe effects than either the disease or the radiation separately.

COMMENTS ON THE PRESENT MPC OF STRONTIUM 90 BASED ON THE INFORMATION GAINED FROM THE EFFECTS OF RADIUM IN MAN

The present estimates of the MPC of strontium 90 are based on the assumption that both radium and strontium are distributed in a similar manner in bone. It has been shown that the radium concentration in different parts of the skeletons of the radium patients and luminous-dial workers may vary by a factor of 10 and probably greater. The present MPC for strontium 90 is based on the comparison of the average skeletal does of strontium and radium. Since under equilibrium conditions the strontium 90 distribution would tend to approach uniformity, comparison of the maximum skeletal dose in the localized areas of high radium concentration in the radium patients and the maximum skeletal dose in the strontium 90 patients might permit raising of the present MPC of strontium 90 by as much as a factor of 10.

The best available estimates indicate that biological effectiveness of radium is 1 to 4 times that of strontium 90 (i. e., the radium RBE[8] = 1 to 4) when compared on an equivalent energy basis. This work is based on the results of animal experimentation (15, 16). Comparison of the radiation dose necessary to produce bone tumors in man from internal and external radiation indicates that the RBE of radium may be somewhat nearer to unity. However, the estimation of radiation dose from external radiation might be in error by as much as a factor of 2. If it can be established that the RBE of radium is greater than unity then it might be possible to increase the MPC of strontium 90 by as much as a factor of 4.

---

[8] Relative biological effectiveness: The ratio of gamma of X-ray dose to the dose that is required to produce the same biological effect by the radiation in question.

It is considered that the best estimates that can be made in regard to the effects of strontium 90 over a life span (70 years) on the present incomplete information of the effects of radium in man are as follows:

1. The skeletal content of strontium 90 necessary to produce a bone tumor in a life span of 70 years would be in the order of 10 microcuries (100 times the present MPC of 0.1 microcurie of strontium 90 for the general population).

2. The skeletal content of strontium 90 necessary to produce significant changes, such as destruction of the hip, would also be in the order of 10 microcuries (100 times the present MPC of strontium 90).

3. The skeletal content of strontium 90 necessary to produce minimal skeletal changes which could be demonstrated roentgenographically, would be in the order of 2 microcuries (20 times the present MPC of strontium 90).

It should be emphasized that these estimates of the concentrations of strontium 90 which may produce skeletal damage are the result of estimates based on the best available information at present.

TABLE I.—*Age and sex distribution of 32 patients having between 0.5 and 14 micrograms of radium present in their bodies on an average of 21 years after deposition*

	Group I (0.5 to 1 microgram)	Group II (1 to 2 micrograms)	Group III (2 to 14 micrograms)	Total
Sex:				
Male	4	2	5	11
Female	11	6	4	21
Total	15	8	9	32
Age:				
30 to 40	1	0	0	1
40 to 50	9	4	2	15
50 to 60	3	3	2	8
60 to 70	0	0	3	3
70 to 80	2	1	2	5
Average age (years)	49	51.9	60.9	----------
Average time (years) since administration	20.3	22.2	22	----------

CHART I

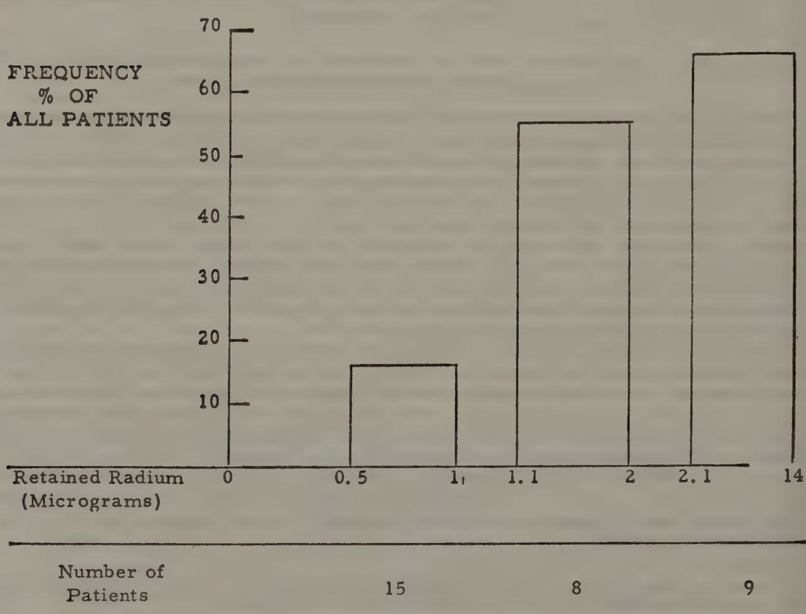
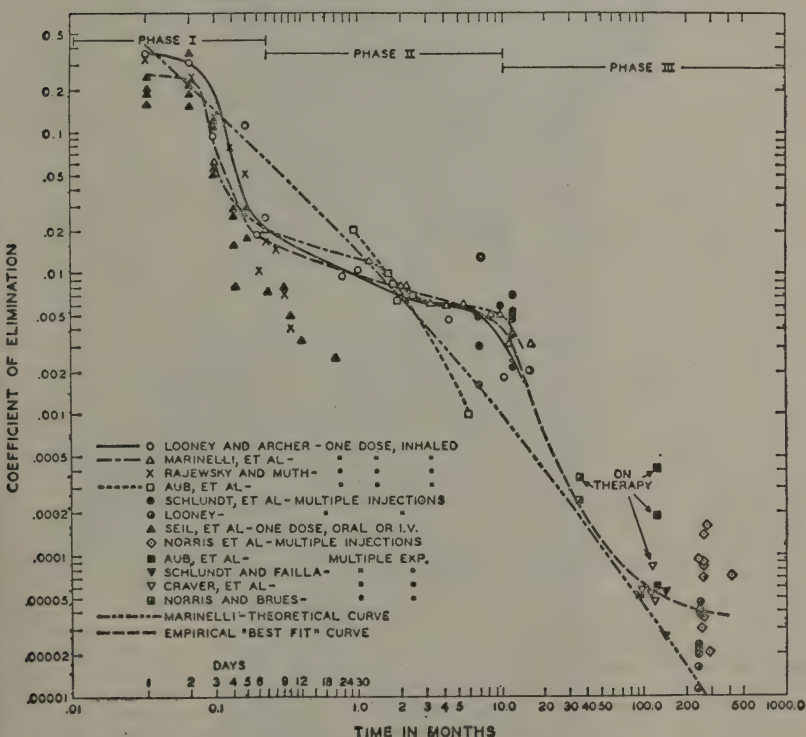


TABLE II.—*Time interval between deposition of radio-elements and occurrence of symptoms, tumor formation, and death in the 1931 and 1951 investigations*

	Number of patients	Average age at administration (years)	Average time from administration to death (years)	Average retained radium (micrograms)
Luminous-dial patients	3	17	10	23
1931 series				(6–50)
Range, years		16–21		
Luminous-dial patients	8	17	26	3.4
1951 series				(.5–10)
Range, years		15–21		
Radium patients		39	26	3.1
1951 series	5			(.8–8)
Range, years		25–49	1–8	

FIGURE 1

CHANGE OF ELIMINATION COEFFICIENT WITH TIME



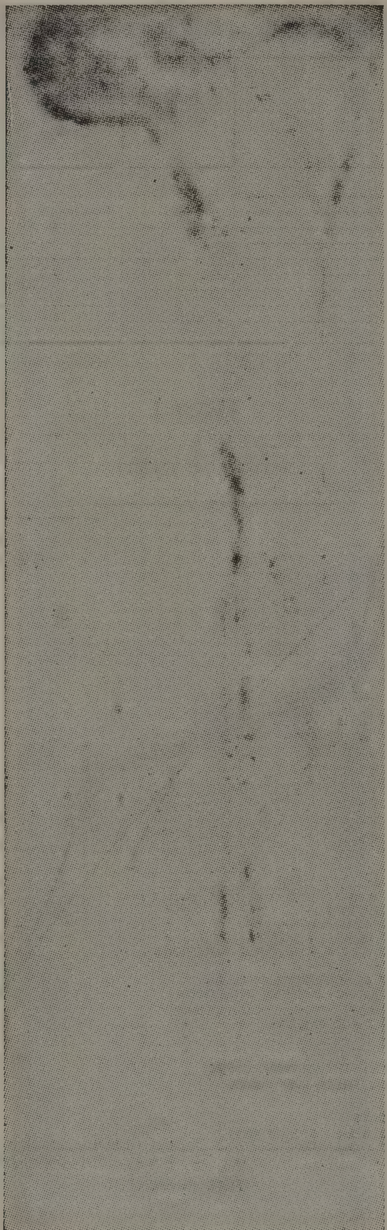


FIGURE 2.—Gross autoradiogram of a longitudinal section of the femur of patient M. K. Note the irregular distribution of radium in the cancellous bone of the upper end of the femur and the small areas of focal concentration in the cortex. The contour of the head is outlined by small concentrations at the junction of the articular cartilage and cancellous bone. The medial portion of the shaft is outlined by the small highly concentrated areas near the periosteum and the endosteum. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

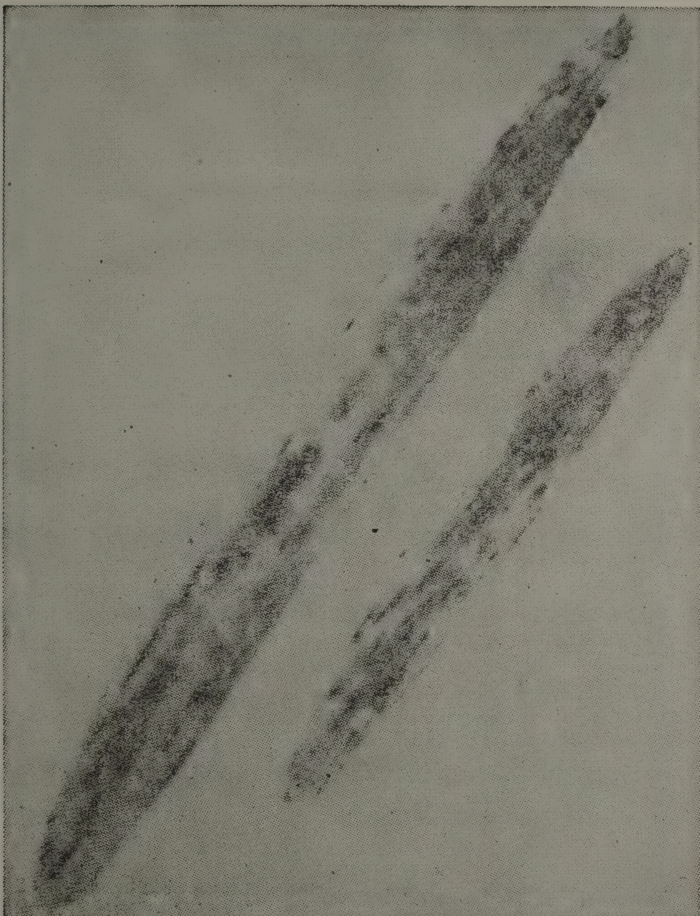


FIGURE 3.—Gross autoradiograms of two longitudinal sections of the cortex of a femur from patient R–24 (5-week exposure). Smaller section was taken 2 millimeters from the periosteum and the larger section was cut just medial to it. Areas of concentration are more frequent in the smaller section and the lower end of the larger section, which are nearer periosteum. The large area of darkening in the center of the larger section is the result, probably, of an endosteal concentration of radium, since this section was on the periphery of the narrow cavity. (Reproduced courtesy of American Medical Association, Archives of Pathology.)

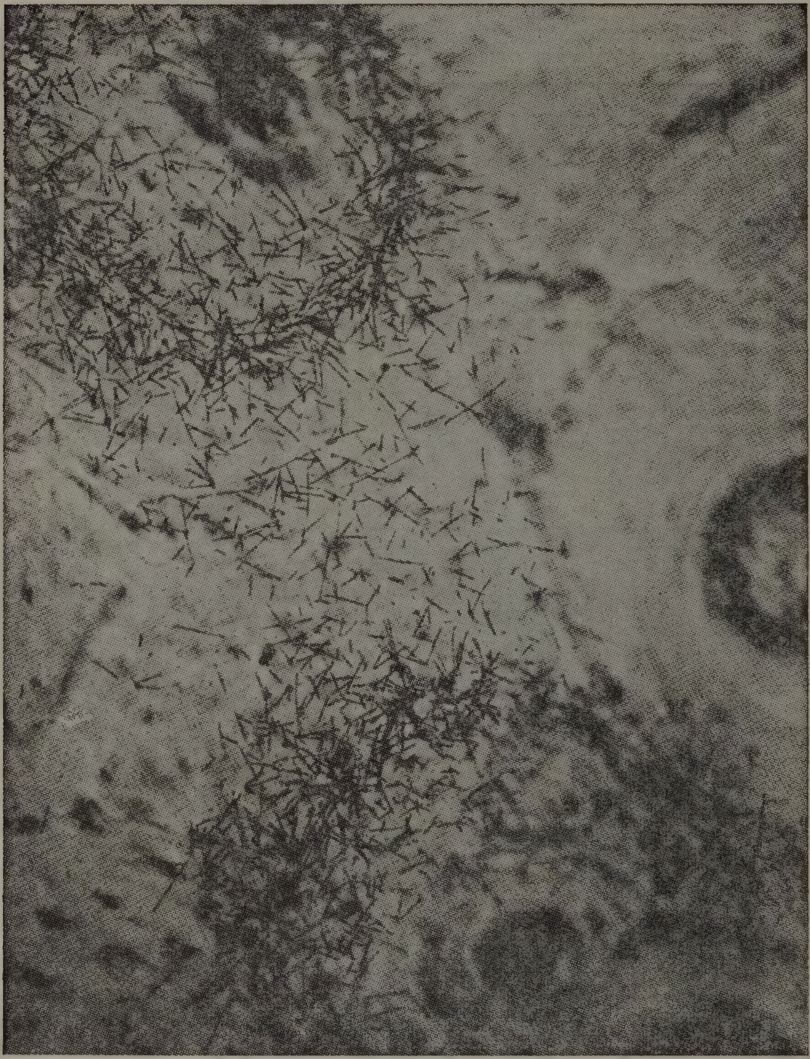


FIGURE 4.—Detailed autoradiogram of a cross section of cortical bone from the humeral shaft of patient R–24 (5-week exposure). Two of the small number of Haversian systems have radium concentrated in this section. The greatest concentration of radium is on two concentric lamellae in the center of the Haversian system. Alpha tracks are less dense in remainder of the two Haversian systems and interstitial lamellae between the systems. Observe that the activity suddenly falls off around these areas of concentration and that the rest of the photomicrograph is free of alpha tracks (X 207). (Reproduced courtesy of American Medical Association, Archives of Pathology.)

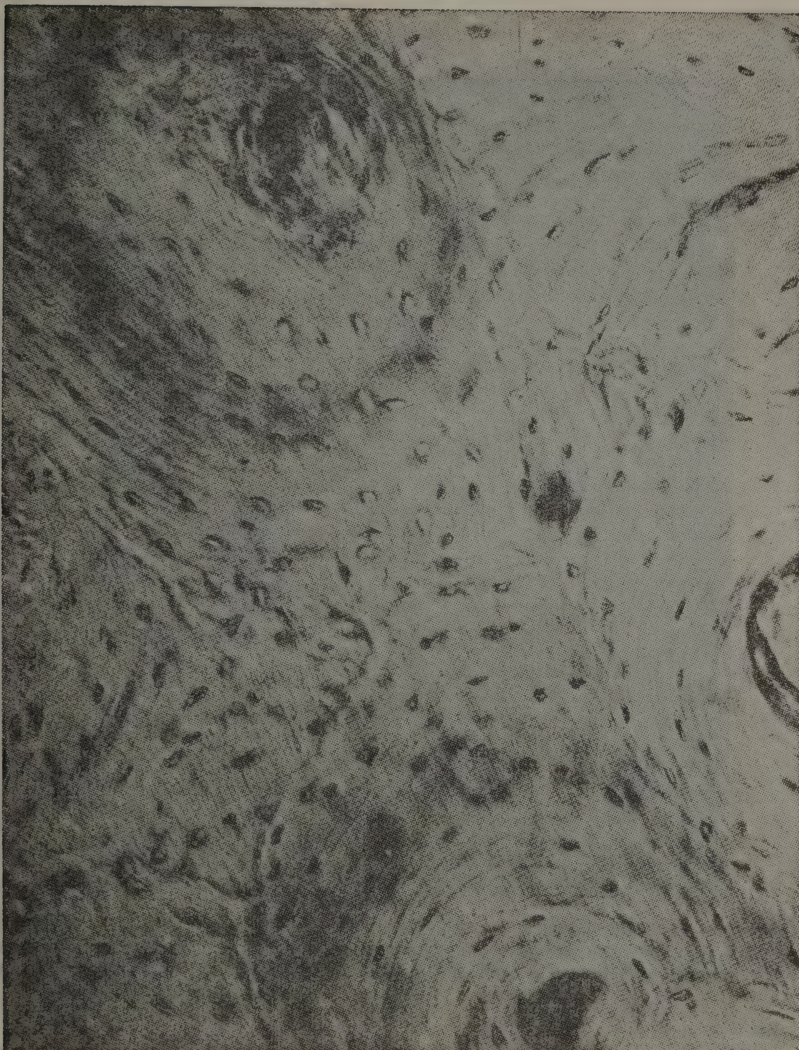


FIGURE 5.—Photomicrograph of bone underlying detailed autoradiogram shown in Figure 4. Note the dark concentric rings outlining lamellae with greater concentration of radium. Central part of Haversian system in upper left is undergoing destructive changes. This, together with figure 4, demonstrates how radium concentration and histopathological changes can be studied at the same time (X 218). (Reproduced courtesy of American Medical Association, Archives of Pathology.)

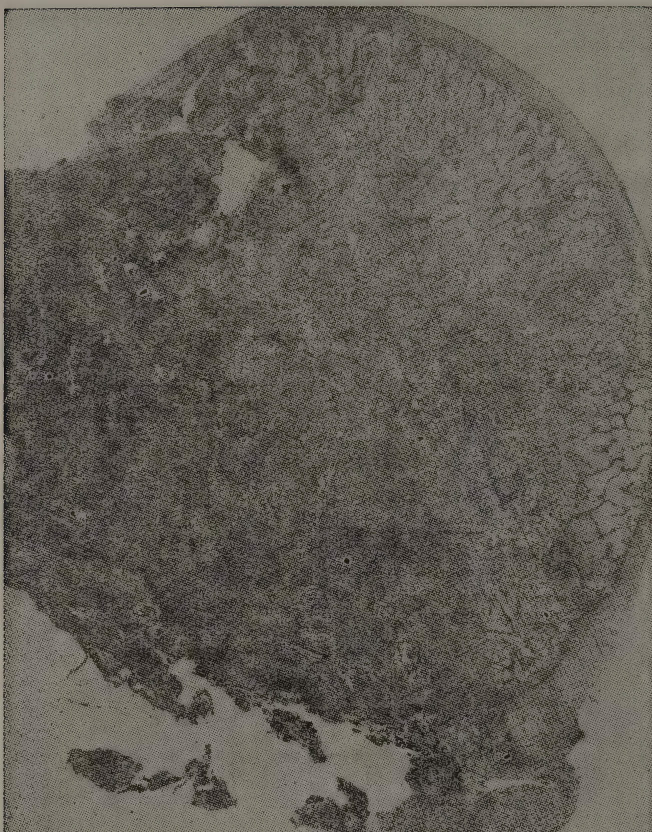


FIGURE 6.—Histological section of the head of a right humerus (fig. 2). Note the areas of atypical osseous tissue throughout the head. The largest area of atypical osseous tissue is seen at the top of the photograph near the articular surface. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

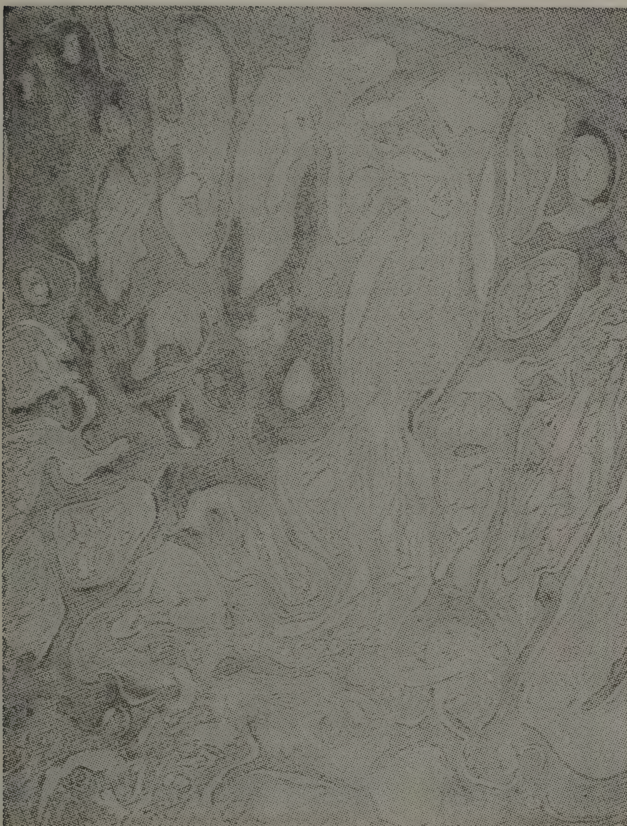


FIGURE 7.—Photomicrograph of one of the areas of atypical osseous tissue seen in the histological sections. The atypical osseous tissue is seen at the top of the photograph. The acellular fibrous tissue is seen throughout the trabecular spaces. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

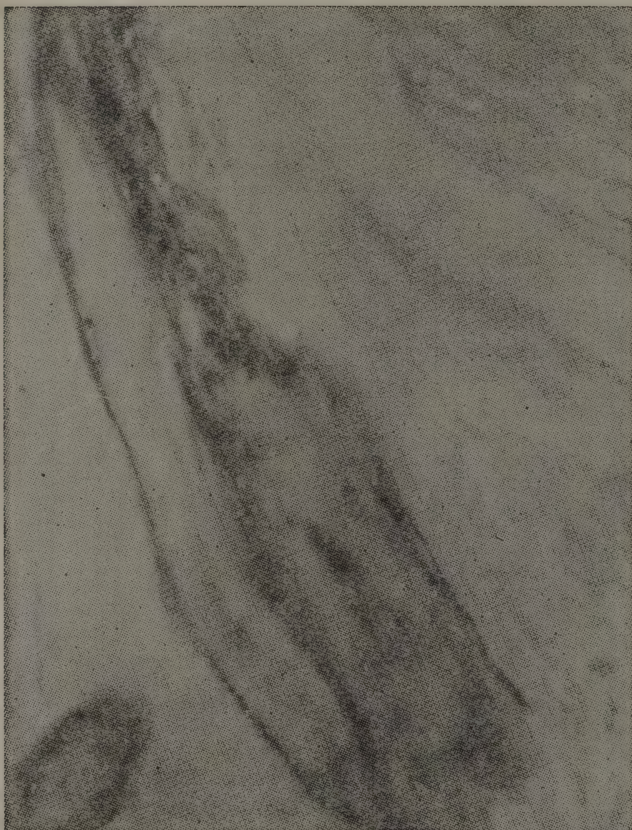


FIGURE 8.—Higher magnification of one of the areas seen in figure 4. Bone is seen in the lower left corner, with darker staining atypical osseous tissue adjacent to it. Fibrous connective tissue is seen in the remainder of the field. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

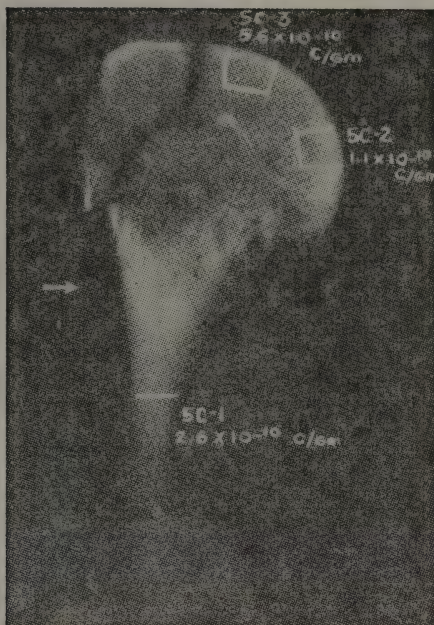


FIGURE 9.—Roentgenogram of a 2-millimeter section taken from the head of the right humerus. (See fig. 6.) Sections of bone, 5C–2 and 5C–3 were removed following the making of gross autoradiographs of this bone section. As will be noted in the figure, 5C–2 was taken from an area which seemed to have a large concentration of radium as shown by gross autoradiography; 5C–3 was taken from an area which seemed to have a small amount of radium as shown by gross autoradiography and a normal roentgenographic pattern. It should be noted that the radium content in RC–3 was five times the content in RC–2. Radium values are expressed as curies per gram of ashed bone. (Reproduced courtesy of the Journal of Bone and Joint Surgery.)

FIGURE 10.—Roentgenogram of the head of the femur of patient (R-34). The mottled appearance is the result of the atypical osseous tissue and hypertrophy of the trabeculae and is characteristic of the changes seen in cancellous bone following the deposition of radioactive elements (Review figs. 6, 7, 8, and 9). (Reproduced courtesy of Journal of Bone and Joint Surgery.)

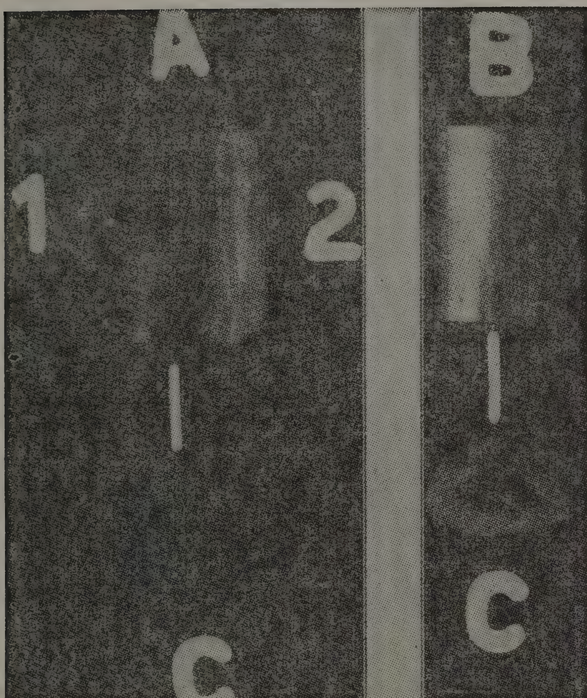


FIGURE 11.—Roentgenogram of a horizontal and vertical section of the fibula of patient (R–24). Note the "streaked" areas that are seen in the long bones as the result of well differentiated areas of destruction in the cortex. The pointers show the areas of destruction in the vertical and horizontal planes (Argonne National Laboratory Report, ANL 4666).

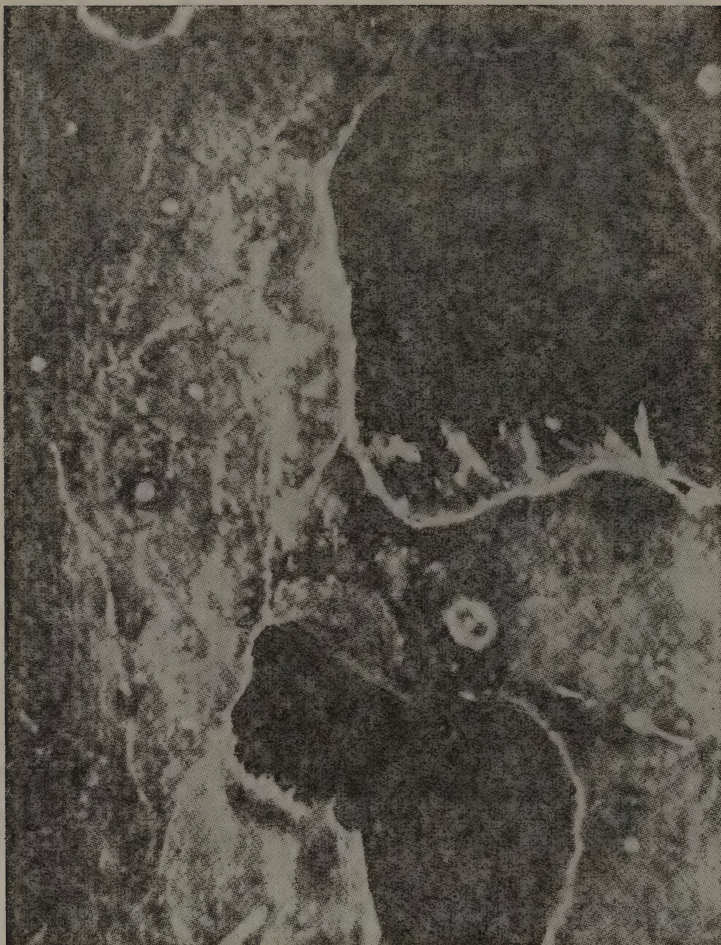


FIGURE 12.—Photograph of the areas of destruction seen in figure 11B. Note that the areas of destruction which give a "streaked" appearance to the long bones are about 3 to 6 times the diameter of the Haversian systems. The central canal in between the two macroscopic areas of destruction is about one-third the diameter of the Haversian system, while in the upper right corner there is a central canal that is enlarged to about two-thirds the diameter of the Haversian system (Argonne National Laboratory Report, ANL 4666).

FIGURE 13.—Roentgenogram of the lower arm and hand of patient M. L. (L–27). The small areas of decreased density in the radius and ulna and bones of the hand give a streaked appearance. These small well differentiated areas of decreased density are characteristic of the deposition of radioactive elements. (Reproduced courtesy of American Journal of Roentgenology, Radium Therapy and Nuclear Medicine, 72: 842, 1954.)

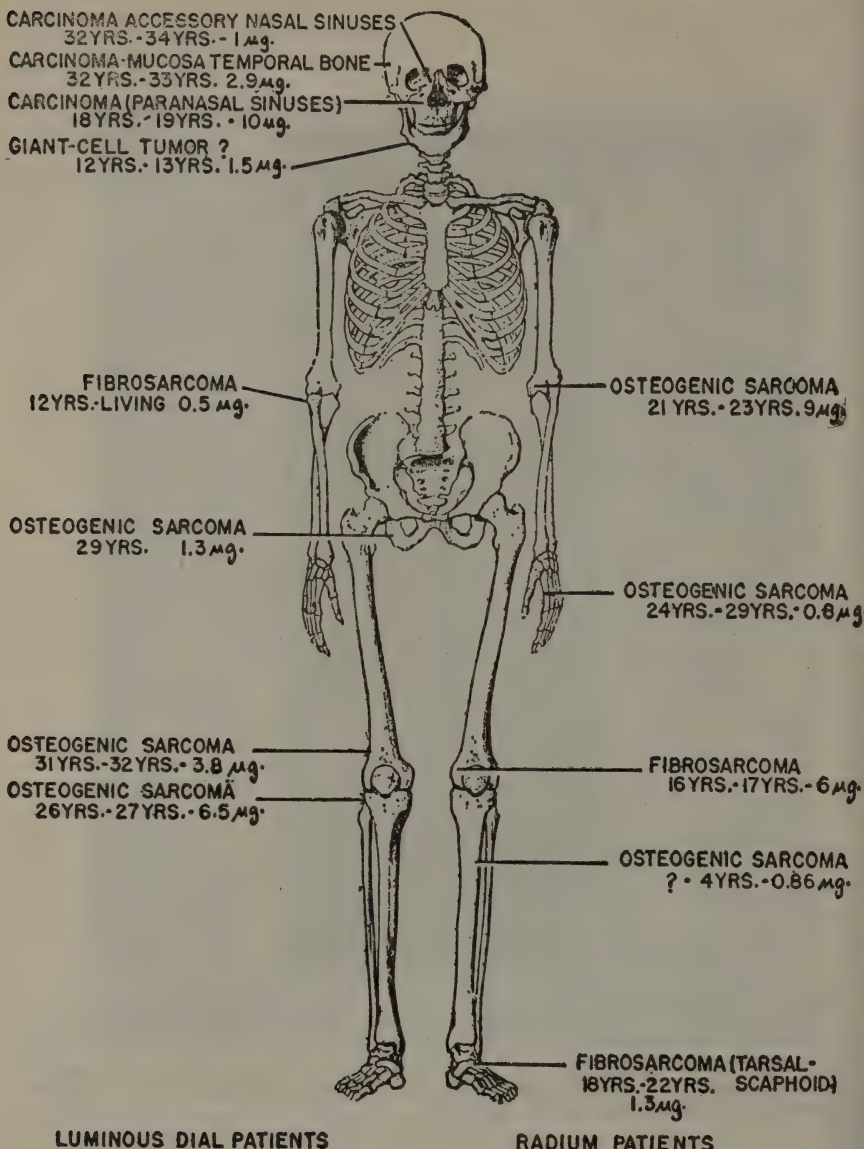


FIGURE 14.—Tumor formation in patients in Boston and Chicago investigations (1951). The five tumors which developed in the 50 patients who had received radium are shown on the right side of the skeleton and the 8 tumors which developed in 8 luminous-dial painters are shown on the left side of the skeleton. Two more tumors have developed in the patients who had received radium medically. These are not included. The type of tumor, the time from deposition to occurrence of symptoms, the time from deposition to death, and the amount of radium are given in each case. Reading from top to bottom, the numbers of the luminous-dial workers are 15, 20, 25, 16, 8, 14, 21, and 23; the numbers of the radium patients are 45, 17, 36, 18, and 24. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

REFERENCE BIBLIOGRAPHY

1. Arnold, J. S.: Progress Report: Radioautography, ANL 4625. Chicago, Argonne National Laboratory, 1951.

2. Aub, J. C.; Evans, R. D.; Gallagher, D. M.; and Tibbett, D. M.: Effects of Treatment on Radium and Calcium Metabolism in the Human Body. Ann. Int. Med., 11: 1443–1463, 1938.

3. Aub, J. C.; Evans, R. D.; Hempelmann, L. H.; and Maitland, H. S.: The Effects of Internally Deposited Radioactive Materials in Man. Medicine, 31: 221–239, 1952.

4. Bloom, W.: Histopathology of Irradiation from External and Internal Sources. New York, McGraw-Hill Book Co., 1948.

5. Brues, A. M.: Tyler, S.; Argonne National Laboratory Report 5518 (1956).

6. Brues, A. M.: Biological Hazards and Toxicity of Radioactive Isotopes J. Clin. Invest., 28: 1286–1296, 1949.

7. Brues, A. M.; Lisco, H.; and Finkel, M.: Carcinogenic Action of Some Substances Which May Be a Problem in Certain Future Industries. Atomic Energy Commission Document, MDDC–145, 1946.

8. Castle, W. G.; Drinker, K. R.; and Drinker, C. K.; Necrosis of the Jaw in Workers Employed in Applying a Luminous Paint Containing Radium. J. Indust. Hyg., 7: 371–382, 1925.

9. Curtiss, L. F.; Evans, R. D.; Failla, G.; Flinn, F. B.; Martland, H. S.; Paul, J. E.; Rogers, J.. S.; Stephenson, C. S.; and Taylor, G. T.: Safe Handling of Radioactive Luminous Compounds. National Bureau of Standards, Handbook H 27, 1941.

10. Cruz, M.; Coley, B. L.; Stewart, F. W.; Post Radiation Bone Sarcoma, Cancer 10, 72–88, 1957.

11. Cameron, W. H., and Viol, C. H.: Classification of the Various Methods Employer in the Internal Administration of Radium Emanation and Radium Salts. Radium, 4: 57–68, 1915.

12. Dudley, R. A.: The Toxicity of Skeletan Irradiation at Naturally Occurring Radiation Levels. 1957.

13. Evans, R. D.: Radium Poisoning. II. Quantitative Determination of the Radium Content and Radium Elimination Rate of Living Persons. Am. J. Roentgenol., 37: 368–378, 1937.

14. Evans, R. D.; Harris, R. S.; and Bunker, J. W. M.: Radium Metabolism in Rats, and the Production of Osteogenic Sarcomas by Experimental Radium Poisoning. Am. J. Roentgenol., 52: 353–373, 1944.

15. Finkel, M.: Argonne National Laboratory Report, 5518 (1956).

16. Finkel, M.: Argonne National Laboratory Report, 5597 (1956).

17. Hatcher, C. H. J.: Bone Surgery, 27, 179.

18. Johnson, L. C.: Personal communication.

19. Kulp, J. L.; Eckelmann, W. R.; Schulert, A. R.: Strontium 90 in Man, Science 125, 219–225, 1957.

20. Lisco, H.: The Standard Man. Quarterly Report, Biological and Medical Division, Argonne National Laboratory, ANL 4253, 96–101, Nov. 1948 to Feb. 1949.

21. Lisco, H.: Bone As a Critical Organ For the Deposition of Radioactive Materials, in the Ciba Foundation Symposium on Bone Structure and Metabolism. Little, Brown & Co., Boston, 1956.

22. Lisco, H.: Personal communication.

23. Looney, W. B.; and Woodruff, L. A.: Investigation of Radium Deposition in the Human Skeleton by Gross and Detailed Autoradiography. A. M. A. Arch. Pathol., 56: 1–12, 1953.

24. Looney, W. B.: The Initial Medical and Industrial Use of Radioactive Materials. Am. J. Roentgenol., 72: 838–848, 1954.

25. Looney, W. B.; Hasterlik, R. J.; Brues, A. M.; and Skirmont, E.: The Clinical Investigation of the Chronic Effects of Radium Salts Administered Therapeutically (1915–1930). Am. J. Roentgenol., 73: 1006–1037, 1955.

26. Looney, W. B., and Archer, V. E.: Radium Elimination Following a Radium Accident. (To be published.)

27. Looney, W. B.: Late Effects (25 to 40 Years) of the Early Medical and Industrial Use of Radioactive Materials. Journal Bone and Joint Surgery. Part I: 37–A: 1169–1187, 1955; part II: 38–A: 175–218, 1956; part III: 38–A: 392–406, 1956.

28. Marinelli, L. D.: The Evaluation of Maximum Permissible Radium Content. (Unpublished.)

29. Marinelli, L. D.: Miller, C. F.; Gustafson, P. F.; and Rowland, R. E.: The Quantitative Determination of Gamma-Ray Emitting Elements in Living Persons. Am. J. Roentgenol., 73: 661–671, 1955.

30. Marinelli, L. D.; Norris, W. P.; Gustafson, P. F; and Speckman, T. W.: Transport of Radium Sulfate from the Lung and Its Elimination from the Human Body Following Single Accidental Exposures. Radiology, 61: 903–915, 1953.

31. Martland, H. S.: The Occurrence of Malignancy in Radioactive Persons. Am. J. Cancer, 15: 2435–2516, 1931.

32. Martland, H. S.: Occupational Poisoning in Manufacture of Luminous Watch Dials. General Review of Hazard Caused by Ingestion of Luminous Paint with Special Reference to the New Jersey Cases. J. Am. Med. Assn., 92: 466–473, 1929.

33. Martland, H. S.: The Occurrence of Malignancy in Radioactive Persons. Am. J. Cancer, 15: 2435–2516, 1931.

34. Martland, H. S.; Conlon, P.; and Knef, J. D.: Some Unrecognized Dangers in the Use of and the Handling of Radioactive Substance. With Special Reference to the Storage of Insoluble Products of Radium and Mesothorium in the Reticulo-Endothelial System. J. Am. Med. Assn., 85: 1769–1776, 1925.

35. Norris, W. P.; Speckman, T. W.; and Gustafson, P. F.: Studies of the Metabolism of Radium in Man. Am. J. Roentgenol., 73: 785–802, 1955.

36. Phemister, D. G.: Personal communication.

37. Phemister, D. H.: Aseptic Necrosis of the Head of the Femur. Surg., Gynec., and Obstet., 59: 415–440, 1934.

38. Potter, R. M.: Personal communication.

39. Saubernann, S.: A Review of Radium Therapy. Seventy-ninth annual meeting of the British Medical Association. Lancet, 2: 447, 1911.

40. Schlundt, H., and Failla, G.: Detection and Estimation of Radium in Living Persons. III. The Normal Elimination of Radium. Am. J. Roentgenol., 26: 265–271, 1931.

41. Schwartz, L.; Makepeace, F. C.; and Dean, H. T.: Health Aspects of Radium Dial Painting. IV. Medical and Dental Phases. J. Indust. Hyg., 15: 447–455, 1933.

42. Seil, H. A.; Viol, C. H.; and Gordon, M. A. L: The Elimination of Soluble Radium Salts Taken Intravenously and Per Os. Radium, 5: 40–44, 1915; New York Med. J., 101: 896–898, 1915.

43. Silberstein, H. E.: Radium Poisoning. A Survey of the Literature Dealing with the Toxicity and Metabolism of Absorbed Radium. Rochester, University of Rochester, A. E. C. D–2122.

44. Van Middlesworth, L.: A Study of Plutonium Metabolism in Bone. Atomic Energy Commission Document, MDDC–1022, 1947.

45. Vaughn, J. M.: The Effects of Radiation on Bone. In the Biochemistry and Physiology of Bone, Academic Press. New York, 1956.

46. Voegtlin, C., and Hodge, H. C.: Pharmacology and Toxicology of Uranium Compounds. New York, McGraw-Hill Book Co., 1953.

47. Wintrobe, M. M.: Clinical Hematology. Philadelphia, Lea and Febiger, 1946.

ACKNOWLEDGMENTS

The author wishes to express his appreciation to Dr. William Rogers, editor of the Journal of Bone and Joint Surgery, Dr. Lawrence Reynolds, editor of the American Journal of Roentgenology, Radium Therapy and Nuclear Medicine, and to Dr. Paul Cannon, editor of the American Medical Association Archives of Pathology for granting permission to incorporate results of investigations published in their respective journals in this report.

From 1950 to 1952, the author was a postdoctorate Atomic Energy Commission fellow in the medical sciences of the National Research Council at the Argonne National Laboratory. He was assigned the responsibility for the clinical aspects of the investigation of individuals who had been given radium salts and people who had been employed as luminous-dial workers (1915 to 1930) and the responsibility for the correlation of the clinical aspects with the biophysical aspects of the investigation. This investigation was conducted by the following: Dr. A. M. Brues, Dr. L. D. Marinelli, Dr. W. P. Norris, Dr. R. J. Hasterlik, and Dr. A. H. Stehney, their associates and the author. The histopathological studies were made at the department of orthopedic surgery, University of Chicago, with Dr. C. Howard Hatcher. Detailed reports of the various phases of the investigation will be published.

The author was afforded the privilege of reviewing the clinical data of a similar investigation carried out at the Harvard Medical School and the Massachusetts Institute of Technology by Dr. J. C. Aub, Dr. R. D. Evans, Dr. L. H. Hempelmann, and Dr. M. S. Martland.

The Atomic Energy Commission fellowship, 1950–52, was under Dr. A. M. Brues, Director, Division of Biology and Medicine, Argonne National Laboratory.

Dr. L. D. Marinelli, Dr. W. P. Norris, Dr. A. H. Stehney, and their associates were responsible for the physical and radiochemical aspects of the investigation at the Argonne National Laboratory.

Dr. R. J. Hasterlik and Miss Ellen Shermont gave their assistance in the clinical and hematological aspects of the investigation at the Argonne National Laboratory.

Dr. Joseph Aub, Dr. Robley Evans, Dr. Louis Hempelmann, and Dr. Lawrence Robbins gave their assistance in the review of the clinical data of the investigation carried out at the Harvard Medical School at the Massachusetts Institute of Technology.

The late Dr. Dallas Phemister and Dr. C. Howard Hatcher of the University of Chicago gave their assistance in the orthopedic and histopathological aspects of the investigation.

Dr. Granville Bennett of the University of Illinois, Dr. Lent Johnson of the Armed Forces Institute of Pathology, and Dr. J. A. Turner of the Naval Medical School gave their assistance in the histopathological aspects of the investigation.

Dr. William Bloom and Dr. Franklin C. McClean of the University of Chicago gave their assistance in the histopathological aspects of the investigation.

Dr. James Arnold and Miss Lois Woodruff gave their assistance in the autoradiographic aspects of the investigation.

Dr. Paul C. Hodges and Dr. Russell Nichols of the University of Chicago and Dr. Robert Potter of Northwestern University gave their assistance in the radiological aspects of the investigation.

Dr. Edward Jerome of the Naval Medical Research Institute, and Mr. Sylvanus Tyler and Miss Joan Guerin of the Argonne National Laboratory gave their assistance in the statistical aspects of these investigations.

Dr. V. E. Archer of the National Institutes of Health gave his assistance in the preparation of the data on the elimination of radium.

Mr. Atlee Tracy of the Argonne National Laboratory and Mr. J. T. Stringer of the Naval Medical School were responsible for the photographs.

Mr. Melvin Runkel of the Naval Medical School and Miss Frances Fee of the Argonne National Laboratory prepared the illustrations.

A Study of the Dynamics of Strontium and Calcium Metabolism and Radioelement Removal [1]

PRELIMINARY REPORT, MAY 31, 1957

W. B. Looney,[2] C. J. Maletskos,[3] M. J. Helmick,[4] J. Reardon,[5] J. Cohen,[6] W. Guild,[7] and F. I. Visalli [4]

The classic studies of Aub, Evans et al.[1a] in 1938 in radioelement removal demonstrated that the renal clearance for radium was less than 1 percent in 24 hours. This finding suggested the possibility that direct radioelement removal from the blood might prove to be an effective way of eliminating radio-

---

[1] From the radioactivity center, Massachusetts Institute of Technology, the kidney laboratory, Peter Bent Brigham Hospital, and the John Collin Warren laboratories of the Huntington Memorial Hospital of Harvard University, Massachusetts General Hospital, Boston, Mass.

[2] Clinical research fellow in medicine, Huntington Memorial Laboratories, Massachusetts General Hospital, and Harvard Medical School. Visiting fellow in physics, Massachusetts Institute of Technology; special Public Health Service research fellow of the National Cancer Institute.

[3] Research physicist, Massachusetts Institute of Technology.

[4] D. S. R. staff member, Massachusetts Institute of Technology.

[5] Research fellow in medicine, Harvard Medical School; Public Health Service research fellow, kidney laboratory, Peter Bent Brigham Hospital.

[6] Visiting fellow in physics, Massachusetts Institute of Technology.

[7] Acting director, cardio-renal service, Peter Bent Brigham Hospital, instructor in medicine, Harvard Medical School.

[1a] Aub, J. C.; Evans, R. D.; Gallagher, D. M.; and Tibbetts, D. M.: Effects of Treatment on Radium and Calcium Metabolism in the Human Body. Ann. Int. Med. 11: 1443–1463, Feb. 1938.

elements. Both the artificial kidney[2] and ion-exchange resins[3] afford means for radioelement removal, as well as providing an opportunity for extension of the work by Hastings and Huggins[4] on the mobilization of calcium in the circulating body fluids.

Preliminary in vitro experiments were performed to determine some of the parameters necessary to evaluate the feasibility of using either approach.

The first experiments were carried out on a simulated artificial kidney. Stable calcium, calcium 45, and ethylenediamine-tetraacetic acid were readily dialyzable under conditions similar to those in the artificial kidney (calcium being omitted for the purpose of the present experiments).[5]

In later experiments solutions of strontium 85 and calcium 45 were passed directly through ion-exchange columns to determine the effectiveness of a synthetic cation exchange resin in removing radioelements.

A total of 20 dogs has been studied, each dog having been connected to either the artificial kidney or ion-exchange column following the administration of strontium 85, strontium 89, and calcium 45. The ion-exchange column has been used in preference to the artificial kidney because of its more effective removal of calcium, its simplicity and potential adoption for more extensive utilization.

The ion-exchange resin offers a wide range of potential application to biological investigation. Appropriate adjustment of the cation concentrations in the column permits preferential removal of a particular cation of interest. The ability to place electrolytes of a biological system out of equilibrium affords an excellent tool for the study of the dynamics of both the stable and the radioactive electrolytes of the biological system.

The efficiency of radioelement removal as a function of time after administration has been determined by both single- and multiple-isotope methods. Between 30–40 percent of the radio-isotopes injected intravenously 1 hour prior to connecting the dog to the resin column can be removed during a 4- to 6-hour period. At 12 hours after injection, total removal of the radioelements decreased to about 6 to 12 percent, and at 24 hours to about 3 to 6 percent. After 3 days about 2 percent is removed. When the experiment was repeated in 2 dogs 1 week later, less than 1 percent was removed.

Analysis of the removal of the isotopes in three dogs indicated the following: About 80 to 90 percent of the dose is in a hypothetical compartment of bone which has a half time of removal of 8 to 16 hours; the remainder of the dose is in a compartment approximating in size the extracellular space and the half time of removal is 15 to 30 minutes.

To test the influence of serum calcium concentration on isotope removal, calcium 40 was infused intravenously at the same time as the resin perfusion. No significant decrease in radiostrontium removal has been found in 2 dogs in which the serium calcium levels of 6 to 8 milligrams percent were maintained. This result would be consistent with the hypothesis that the principal mechanism of radioelement removal by the ion-exchange column is cation exchange rather than enhanced physiological response from depressed calcium levels.

These studies demonstrate that the ion-exchange column and the artificial kidney are practical means for studying the dynamics of stable and radioactive electrolytes. No major contraindications have been found to prevent its adoption for clinical use.

Representative Cole. Mr. Chairman, if you do not mind, I would like to ask again, Dr. Looney, if you will interpret your expression that the assumption that the incidence of the effect of strontium 90 is proportionate to the magnitude of the dose. You have indicated that experience and studies led you to the conclusion that this is an overcautious—did you say overcautious?

Dr. Looney. Yes.

---

[2] Merrill, J. P.: Medical Progress: The Artificial Kidney. New England J. Med. 246: 17–27, Jan. 3, 1952.

[3] Kessler, B. J.; Liebler, J. B.; Abrahams, J. I.; and Sass. M.: Reduction of Hyperkalemia by Circulation Blood Through a Cation Exchange Resin. Proc. Soc. Exper. Biol. & Med. 84: 508–510, Nov. 1953.

[4] Hastings, A. B.: Studies on the Effect of Alteration in the Concentration of Calcium Circulating Fluids on the Mobilization of Calcium. Metabolic Interrelations, Transactions of the Third Conference, 1951, pp. 38–50.

[5] Looney, W. B., Maletskos, C. J., and Helmick, M. J.: Removal of Radiocalcium in Dogs, Progress Report, Radioactivity Center, M. I. T., Department of Physics, May 1956.

Representative COLE. Assumption?

Dr. LOONEY. Yes.

Representative COLE. What do you mean by that?

Dr. LOONEY. I mean that the present clinical information that we have would not substantiate the concept of a linear relationship, neither does it disprove it. If I had to weigh and balance these two factors, I would say that most of the information indicates that a certain amount of irradiation is necessary to produce bone tumors in man. However, this is based on the available information. It certainly is to be again emphasized that these tumors may be produced in man at a lower dose, and we have not been able to detect this at present. There are some methods of obtaining more information from clinical studies. I think they will be done.

To say this might be an overcautious assumption is probably not a good term, but I hope I have given you the reason for making the statement.

Representative COLE. Since your clinical evidence indicates that the incidence may be less in proportion to the magnitude of the dose, would that indicate the possibility of a threshold for the effects of strontium 90?

Dr. LOONEY. I would say that the present clinical information in man would not substantiate either conclusion.

Representative COLE. Based on clinical evidence?

Dr. LOONEY. Based on the evidence in man; yes, sir.

Representative COLE. You are not able to determine yes or no with respect to threshold?

Dr. LOONEY. No, sir; I am not.

Representative HOLIFIELD. Dr. Looney, how were you able to determine the amount of exposure these people had, in view of the fact it was years later before you were aware of their illnesses?

Dr. LOONEY. The patients in Chicago were found by reviewing the records of a mental hospital in which the patients were given radium. The files of the United States Radium Corp. were also made available to the Argonne National Laboratory, and we were able to get names and to locate these people by following names.

Representative HOLIFIELD. This did not obtain to those employed as radium painters?

Dr. LOONEY. Yes; it did.

Representative HOLIFIELD. Were you able to measure the dose they received at the time they received it by the residual amount in their bones when it was called to your attention?

Dr. LOONEY. The physical estimates were made by the physicists at Massachusetts Institute of Technology, and at Argonne National Laboratory. That is a physical area, and I would prefer to leave it to the physicists.

Representative HOLIFIELD. I was just interested to find out if we had an accurate estimate of the original dose. I know in the case of the Hiroshima and Nagasaki people that is one of the missing elements in our evaluation of the dose, that we do not know exactly how much they have received.

Dr. LOONEY. Yes. In regard to the patients in the mental hospital, we do have a record of the amount given, and the estimates at 6 and 12 months. Estimates were also made after 20 years. This is the

best available evidence we have in man. Based on this information we can make estimates of the original dose of radium in other people in which we find the radium 20 to 30 years after administration. The physical data on the luminous dial workers is confused by the fact that mesothorium and radiothorium were present in the paint. This presents a very difficult problem in trying to establish reliably the relationship of the clinical changes to the physical estimates of the radiation dose.

Representative HOLIFIELD. Thank you.

Representative VAN ZANDT. Dr. Looney, is it possible to leach out selectively the poison that has gotten into the skeleton of the body?

Dr. LOONEY. You are talking about removal of these radio elements once they are deposited in bone?

Representative VAN ZANDT. Yes.

Dr. LOONEY. We have been working on that in Boston in the past 2 years, trying to remove strontium 85, strontium 89, and radio calcium from bone. We found in the first hour we can remove approximately 30 or 40 percent of the strontium. This efficiency of removal rapidly declined, until after 2 or 3 days, it was less than 1 percent.

Other methods using chemicals to remove bone-seeking radio element have been attempted. There has been some progress in this field, but once the radio element is deposited in the bone, the chances are very remote that we will be able to remove it.

Representative VAN ZANDT. Thank you.

Chairman DURHAM. Doctor, figure 14, where you have the skeleton——(See p. 1188.)

Dr. LOONEY. Yes, sir.

Chairman DURHAM. Was any conclusion made as to why the carcinoma was more prevalent in the head and nose and mouth than other parts of the body?

Dr. LOONEY. You will notice that these people who developed these tumors were luminous dial workers. There has been the hypothesis that because the material was ingested and inhaled, rather than injected intravenously or given orally, that it might be the result of the local effect. But, I might point out that one of the radium patients has also developed a similar type of tumor. I have no readily available conclusion as to why this developed.

Senator HICKENLOOPER. Mr. Chairman.

Representative HOLIFIELD. Senator Hickenlooper.

Senator HICKENLOOPER. Just from a visual examination of the chart, it would indicate that one might assume the tumors occurred in proximity to the point of contact or ingestion of this radium treatment, or the radium material. In other words, with the luminous dial patients, as I understand it, they ingested this as a result of either inhalation or putting the brushes in their mouths to wet them so they could point them up and paint the figures on the dials.

Dr. LOONEY. Yes, sir; that is correct.

Senator HICKENLOOPER. And the incidence seems to be much greater in the nasal and throat area in those. On this chart there is no incidence of a tumor above the elbow in the strictly radium patient as differentiated from the luminous dial patient.

Dr. LOONEY. Yes. However, you will note it is a different type of tumor in the radium patients. You will notice these tumors are carcinomas in the luminous dial workers, and you will notice that the

sacromas of bone have occurred both in the luminous dial workers and radium patients.

Senator HICKENLOOPER. I am speaking purely as a layman who knows nothing whatsoever about this thing. A layman might be led to the conclusion from the chart that radium treatments might have been given in other parts of the body, but in the luminous dial workers the repeated incidence is at the point of ingestion very frequently.

Dr. LOONEY. This is an interesting observation, sir. I know this has created considerable comment as to whether there is a casual relationship among the people associated with these investigations. I am trying to point out both arguments for and against this. It is a different type of tumor that has developed in his area, and most of the tumors have been sarcomas of the bone.

Chairman DURHAM. Did the same type rays produce the three different types of sarcomas and carcinoma? You have three different types. Did the same rays produce all three types?

Dr. LOONEY. I think that the present available evidence is that any radiation has a similar effect biologically. So that it would be a combination of these three effects.

As far as radium is concerned, the majority of the radiation comes from the alpha particles, probably 90 or 95 percent.

Chairman DURHAM. Then the conclusion would be that beta rays or gamma rays would produce all types of carcinomas?

Dr. LOONEY. In sufficient quantities we must be aware of the quantitative aspects of the effect of radiation. I think it was brought out previously that the very sensitivity of physical measurements may lead to overemphasizing the effects of minute amounts of radioactivity.

We have a range of thousands of times between the amount of strontium 90 that is present, and the amount of strontium 90 that we calculate to produce equivalent energy to cause tumors in man from the available information on radium.

Representative VAN ZANDT. Mr. Chairman?

Representative HOLIFIELD. Mr. Van Zandt.

Representative VAN ZANDT. Dr. Looney, you have been talking about bone tumors. What about the utilization of radiation to attack these bone tumors?

Dr. LOONEY. Well, sir, I am not a radiologist, and this is in the field of radiology. I am aware of this, but I think you would get a much more competent opinion on this from some radiologist who is actively engaged in treatment.

Representative VAN ZANDT. Would the same thing apply to arthritis?

Dr. LOONEY. Yes, sir.

Representative VAN ZANDT. One more question, Dr. Looney. You have had many years of experience now in studying the body that has absorbed radiation from radium. Have you developed any kind of a preventative to this type of radiation?

Dr. LOONEY. No, sir, we have not yet. So far as I know, there has been no effective means of treatment.

Representative VAN ZANDT. Have you developed any type of a program that would prepare the body to resist radiation stemming from radium?

Dr. LOONEY. I have no knowledge of such.

Representative HOLIFIELD. Thank you very much.

Our next witness will be Dr. W. F. Libby, Commissioner, Atomic Energy Commission, a distinguished chemist, and member of the Commission since 1954.

Dr. Libby, before you start your testimony, the Chair will insert, without objection, 2 letters of notification, 1 under date of May 3 to Mr. Strauss, which gave an outline of our hearings, and the witnesses that had been invited up to that time, and the second letter under date of May 21, which supplemented the first letter. I would like to read the pertinent part of that, in which I said to Mr. Strauss:

I would like to make clear that we would be happy to have Dr. Libby on hand to provide extra testimony and comment throughout the hearings after the introductory witnesses beginning on Monday afternoon, May 26. We understand that he is preparing written statements for certain portions of the outline. We will be glad to receive these statements for the record, and will be happy to have Dr. Libby comment on them orally as each topic comes up.

We would also appreciate hearing from Dr. Libby on topic 11, in which he could discuss the implication of our present knowledge of the fallout situation as it exists.

(The letters are as follows:)

CONGRESS OF THE UNITED STATES,
JOINT COMMITTEE ON ATOMIC ENERGY,
May 3, 1957.

Mr. LEWIS L. STRAUSS,
Chairman, United States Atomic Energy Commission,
Washington, D. C.

DEAR MR. STRAUSS: The Joint Committee on Atomic Energy plans to hold open hearings in Washington, D. C., on the subject "The Nature of Radioactive Fallout and Its Effects on Man," May 27–29 and June 3–7. This letter is to confirm arrangements made informally for representatives of the Commission and AEC laboratories to testify before the committee. It is our understanding that these arrangements have already been discussed at some length on an informal basis by our respective staffs.

We are attaching material covering the scope, approach, plan and outline of the hearings. You will note the planned division of the hearings into two parts: (I) an organized sequential presentation by expert witnesses, and (II) an open presentation by those working in the field or by interested members of the public who have asked for an opportunity to testify before the committee. You will also note that the outline gives guidance, by each topic I–XII, as to whether the presentation is planned as oral, written inserts for the record, or bibliography—or some combination of those. It is intended that witnesses use their own discretion as to the details of the presentation, and it is not necessary that the outline be rigidly followed. Points that the committee is particularly interested in may be developed by questioning of witnesses.

We are requesting the following representatives of the AEC to testify as expert witnesses for various parts of the organized sequential presentation:

Dr. Charles L. Dunham, Director, Division of Biology and Medicine, Topics I, XI, and XII.

Dr. Willard F. Libby, Topics IV, VI–C–4, IX (Sr–90), and XI.

Gen. A. D. Starbird, Director, Division of Military Application, topic V (General Starbird to be preceded by Dr. Bradbury, of LASL, and Dr. Shelton, of AFSWP).

Dr. Gordon M. Dunning, Division of Biology and Medicine, topic VII.

Dr. Merril Eisenbud, New York Operations Office, for topic VI–C–3 and VI–C–4 and topic III.

Dr. R. F. Reitemier, Division of Biology and Medicine, for a joint presentation of topic VIII, parts B, C, D, E, and G, with Dr. Lyle T. Alexander, of the Agriculture Department.

Dr. Forrest Western, Division of Biology and Medicine, for topic VIII, parts A and H, and perhaps to provide continuity for the other topics in

topic VIII. (The portions of VIII dealing with oceanography and marine life are planned for coverage by Dr. Roger Revelle, Scripps Institute.)

In addition, there is one topic, topic III, for which we would be glad to insert into the record any written statements the AEC would care to submit.

From the AEC laboratories, we are planning to invite the following persons to present oral or written testimony:

Dr. Mark Mills, UCRL, Livermore.
Dr. Wright Langham, LASL.
Dr. E. C. Anderson, LASL.
Dr. ——— Marinelli, ANL.
Dr. Austin Brues, ANL.
Dr. E. P. Cronkite, BNL.
Dr. Norris Bradbury, LASL.

These persons are being contacted directly.

It is possible that other persons in AEC or its laboratories will be needed or that changes in the planned presentation will develop. However, it is not expected that there will be major changes.

The Joint Committee would appreciate receiving as soon as possible short biographies of each person giving testimony, covering professional background, present work, home address and phone number, business address and phone number.

The Committee hopes that the forthcoming hearings will lead to a better understanding of a problem that has become the subject of serious concern to the Congress and the people of this country. Such understanding is essential, in the Committee's view, to the development of sound national policies and to the maintenance of good relations with our friends and allies throughout the world.

The cooperation of the AEC in contributing to the success of the hearings will be greatly appreciated.

Sincerely yours,

CHET HOLIFIELD,
Chairman, Special Subcommittee on Radiation.

CONGRESS OF THE UNITED STATES,
JOINT COMMITTEE ON ATOMIC ENERGY,
May 21, 1957.

Hon. LEWIS L. STRAUSS,
Chairman, Atomic Energy Commission,
Washington, D. C.

DEAR MR. STRAUSS: We are pleased to receive your May 17 letter offering to cooperate with us as we begin our radiation fallout hearings next Monday morning.

As you know from my May 3 letter, we plan that the first portion of the hearings, taking perhaps the days up to about Thursday, June 6, would be devoted to an organized sequential presentation by expert witnesses of the scientific subject matter related to fallout. This presentation would wind up with two topics concerned with the impact of the present state of affairs scientifically on national policy and on the research programs related to fallout.

The purpose of the presentation is primarily to educate the committee, the Congress, and the public on the scientific aspects of this important subject. The outline of this organized presentation, sent to you with my May 3 letter, was developed after consulting with various scientists to figure out the best way to present the subject matter fairly and impartially.

In line with the above arrangement, scientific witnesses were selected to present the subject matters. The list of witnesses and order of presentation was distributed last week. Dr. Dunham was selected to lead off with an objective presentation of the nature of the overall topic of the hearings: radiation and radioactivity, particularly fallout. He was chosen as a qualified represenative of AEC who happens not to have become publicly involved in controversy on the fallout question. It has been our aim to begin and carry on the hearings in a noncontroversial spirit.

My May 3 letter to you listed Dr. Libby as a desired expert witness for several topics, beginning with topic IV (natural background radioactivity) of the outline. We would be happy to have him come before the committee to present objective testimony on these topics. It has been our informal understanding

that Dr. Libby might prepare written statements for these topics but did not wish to appear personally as a witness except once, for a comprehensive statement. Accordingly, our latest timetable lists Dr. Libby only for topic XI (impact on policy), where he can cover any of the scientific facts and implications that he cares to.

Your cooperation and that of the AEC staff in assisting the committee with preparations for the fallout hearings is greatly appreciated.

Sincerely yours,

CHET HOLIFIELD,
Chairman, Special Subcommittee on Radiation.

Representative HOLLIFIELD. Dr. Libby, we are happy to have you before us, and we are ready for your statement.

STATEMENT OF DR. WILLARD F. LIBBY,[2] COMMISSIONER, ACCOMPANIED BY DR. CHARLES L. DUNHAM, DIRECTOR, DIVISION OF BIOLOGY AND MEDICINE, ATOMIC ENERGY COMMISSION

Dr. LIBBY. Mr. Chairman and members of the committee; it is a privilege to be present today because it gives me an opportunity to discuss with you this very important subject—one in which I have taken great personal interest, having spent a major part of my time in the last 4 years directly on this research—watching the progress, performing experiments, and making calculations. Excluding weapons, I consider Project Sunshine—the study of worldwide radioactive fallout and its effect on man—to be one of the most important projects the Commission has. The project is essential, if the Commission is to fulfill its responsibilities in protecting public health and safety. It is being conducted as a scientific study whose primary purpose is to discover the scientific truth and present the facts—publicly.

Senator ANDERSON. Before you go further, is this your own statement, or does this represent the views of the Atomic Energy Commission?

Dr. LIBBY. This is my own statement, Senator Anderson.

Senator ANDERSON. It has not been cleared?

Dr. LIBBY. I have asked for comment, but it is not an official statement of the Commission; it is my own statement.

Senator ANDERSON. Thank you. I thought that was important.

Dr. LIBBY. Yes.

Representative COLE. On that point, Mr. Chairman; you say you have asked for comments. I assume you meant you submitted your statement to other members of the Commission for their comments?

Dr. LIBBY. Yes.

Representative COLE. And have you received comments from any of the Commission?

Dr. LIBBY. There have been no substantial changes. I am not sure all of the Commissioners commented. I left it to my staff to collect the comments, and I am not just sure.

Representative COLE. Did you submit your statement to all of the Commissioners?

Dr. LIBBY. I believe so, sir; yes.

[2] Date and place of birth: December 17, 1908, Grand Valley, Colo. Education: Bachelor of science, University of California, 1931; doctor of philosophy (chemistry), University of California, 1933; Work history: Instructor of chemistry, University of California, 1933–38; assistant professor, 1939–43; associate professor, 1943–45; professor instructor nuclear studies, Chicago, 1945–54; member, General Advisory Commission, AEC, 1950–54; member AEC, 1954—. (Submitted by the Atomic Energy Commission.)

Representative COLE. Were there any of the Commissioners who did not respond or indicate a comment?

Dr. LIBBY. I will have to check with my staff on that.

Representative COLE. So far as you know?

Dr. LIBBY. I do not know of any.

Representative COLE. One more question at that point: How long previous to this morning did you submit the statement?

Dr. LIBBY. It was about a week ago, as I recall, Mr. Cole.

Representative COLE. Thank you.

Dr. LIBBY. This is my statement. It is not the Commission's statement, but it is my statement. But I know of no——

Representative VAN ZANDT. During the course of these hearings, this Project Sunshine has come up from time to time.

Dr. LIBBY. Yes.

Representative VAN ZANDT. Dr. Libby, just how did you arrive at naming this Project Sunshine.

Dr. LIBBY. Well, it happened in the summer of 1953 at the RAND Corp. conference at Santa Monica. I have been trying to think for the last several hours just how it happened. I do not remember, and I do not know, Mr. Van Zandt.

We recognized the need for some name, and one of the boys in the meeting invented this name, and we took it. I am sorry I have no better memory.

Senator HICKENLOOPER. Mr. Chairman, may I ask Dr. Libby—as I understand it, sunshine is stimulated by radiation, is it not?

Dr. LIBBY. Yes, sir; in the ultimate, sunshine is derived from radiation.

Senator HICKENLOOPER. And sunshine, as we know it, and as life exists, is completely vital to life?

Dr. LIBBY. Yes, sir.

Senator HICKENLOOPER. And the effect of the sun and radiation is vital to life. I am not trying to say how the term came up, but it seems to me there is quite a close correlation between sunshine and the effects on human life of radiation.

Dr. LIBBY. I am trying to find out how this name was invented. I am sorry I have not been more successful. I have given you the chronology of it.

Representative HOLIFIELD. There is this exception, however, that sunshine is beneficial to the growth of life, and radiation seems to be the other way. Is that not right?

Dr. LIBBY. At least radiation has many deleterious effects, Mr. Holifield. I think it has a few good ones.

Representative HOLIFIELD. As a Californian, I would like to say, the RAND Corp. being out of California, where we have a lot of sunshine, I would say, coming from California, this helps offset the reputation of smog we have out there.

Senator HICKENLOOPER. As long as we are in that field, Mr. Chairman, I might say, inasmuch as sunshine brings, and has over the milleniums, brought substantial amounts of radiation, perhaps it is that the more sunshine, the more danger there is to the skeletal structure of the human body. That might be argued in favor of those areas which are less blessed with sunshine. I do not know.

Representative COLE. Mr. Chairman, on this point of the name Project Sunshine, I fear a feeling may have developed that that name

was deliberately selected to mislead the public with respect to the importance of the subject under discussion.

Since you were connected to a rather direct degree with this project from its inception, can you assert unequivocally that the selection of the name "Sunshine" has no purpose or intent of misleading or minimizing the importance of the study?

Dr. LIBBY. Yes, Mr. Cole, I certainly can. It never had any purpose to mislead or be flippant about the whole matter. The name was selected—and I am afraid perhaps we did not pay too much attention to the name in selecting it. But there was never any intent to mislead or to minimize the importance of the hazards.

Senator ANDERSON. I thought you testified you did not know how it was selected. If you did not know how it was selected, how could you know the circumstances under which it was selected?

Dr. LIBBY. What I testified to, Senator Anderson, was I did not understand how the word "sunshine" rather than any other word was taken.

Senator ANDERSON. Then how could you answer Mr. Cole's question in the affirmative?

Dr. LIBBY. Well, we certainly did not select this word with any intent of misleading anyone about the seriousness of this subject. That is all.

Representative COLE. The witness may very properly testify he does not know how this was done, but he also may testify he does know why it was not done.

Senator ANDERSON. If you can prove a negative, you may go to it.

Dr. LIBBY. Fallout has been a subject of interest ever since the first atomic bomb was exploded. At the Alamagordo test, July 16, 1945, there were scientists present who were interested in fallout and studies were subsequently made of the sparse vegetation of that countryside.

The organization which is today known as the Health and Safety Laboratory came into being ith the establishment of the Commission on January 1, 1947. Scientists from the Health and Safety Laboratory made their first collection of fallout material on February 1, 1951, when radioactive snow was reported in Rochester, N. Y. At this time, assays of mixed fission products were made on snow and water from samples collected from throughout the Northeast. The source of this radioactivity as undoubtedly our very first Nevada tests conducted in the early spring of 1951—Operation Ranger. The first actual fallout collection network was established a few weeks later for Operation Greenhouse, an Eniwetok test series, and the collections were analyzed for mixed fission products; the network operated from April to June 1951, making collections in eight stations in the United States. The following year, 1952, the Weather Bureau and the AEC collected radioactive fallout at 120 Weather Bureau stations in connection with Operation Tumbler-Snapper.

Representative COLE. At that point, Mr. Chairman.

Dr. Libby, do you intend to indicate the present system of collection stations?

Dr. LIBBY. I would be very pleased to. It is not in my statement, though.

Representative COLE. Your statement says that in 1952 you did have these 120 stations. That was 5 years ago.

Dr. LIBBY. Right.

Representative COLE. I think the committee and the public would be interested to know what the present is.

Dr. LIBBY. I believe Mr. Eisenbud, who is in charge of this, did describe this system to you in earlier testimony, Mr. Cole. Did he not, Mr. Chairman?

I would be pleased to recount it.

He has a system now which is essentially worldwide, 2 sorts, I believe 3 sorts, really. He collects the gummed papers, which he described to you. He puts out buckets, or washtubs at various places to get a total collection. Then, in addition, we have a system of collecting soil samples on a worldwide basis. So we have a much more thorough coverage, not only of the United States, but of the whole of the Western World, at least, and through the U. N. Radiation Committee, I can say of the whole world to a certain degree than existed in this early time of 1952.

Representative COLE. Instead of 120 collecting stations located in the continental United States, can you indicate the approximate number of collecting stations throughout the world?

Dr. LIBBY. I would be hard put to give you even an approximate number. I am sure it is larger, but I can ask for that.

Representative COLE. Dr. Eisenbud is in the audience, and since this is a matter of general interest, let us have it in the record.

Dr. LIBBY. I am sorry. It comprises now only 94 stations in the United States. These are the gummed paper stations. We make a distinction between the gummed paper and the washtubs and the soil.

Ninety-four stations of gummed paper in the United States, with 75 now in foreign countries.

What I call the washtubs are located at seven stations in the United States, and in the following countries: Hawaii, Chile, French West Africa. Austria, Union of South Africa, Thailand, South Rhodesia, Pakistan, Kenya, Japan, Colombia, and Brazil. And this network is being extended.

Senator ANDERSON. Since we are considering the Hawaii statehood bill this morning, I think the Hawaiians would object to being put outside of the United States.

Dr. LIBBY. Well, we have had this network of gummed paper operating continuously since 1951 and 1952. The washtub or stainless steel pot system is just now beginning to operate. It was set out last fall, actually. And the soil collection data—well, I will describe those in my statement.

Representative VAN ZANDT. Dr. Libby, does this sampling go on around the clock, or just at a given time?

Dr. LIBBY. No, it goes around the clock, Mr. Van Zandt.

Representative VAN ZANDT. Around the clock?

Dr. LIBBY. Yes.

Representative VAN ZANDT. In other words, your gummed paper is in the open atmosphere around the clock, and picking up samples continuously?

Dr. LIBBY. Yes. The washtubs are, too. That is, you do not put the washtub out just when it starts to rain. You leave it out continuously. And after a rain you bring it in and swish it around to suspend the particulate matter and bring the rain and the sediment in for analysis. But you put the washtub right back out so that you get the total fallout in the area.

We have established rather definitely that the washtubs give good numbers that check with the total soil content of fallout. This has been done in 2 or 3 places quite carefully, and checks resulted.

Representative VAN ZANDT. Thank you.

Dr. LIBBY. Meanwhile, at the Oak Ridge National Laboratory, Dr. Nicholas M. Smith, starting in 1949, had made a theoretical analysis of the long-range aspects of fallout. Smith concluded that over a period of years strontium 90 would be the most hazardous component in fallout and that an accurate knowledge of the distribution of this substance over the world would be essential to any scientific estimate of the potential long-range health hazard due to fallout. In 1952, RAND Corp. was given a contract to make an independent study of fallout and this study culminated in the summer of 1953 in a conference of selected consultants who made an intensive overall review and evaluation of the fallout problem. The conference recommended that the study of mixed fission products, then current, be supplemented by a worldwide assay of the individual fission product, strontium 90, produced by nuclear detonations, and so Project Sunshine was born. Most of the information presented to you so far in these hearings, if we exclude genetics and the toxic effects of ionizing radiation, was developed in the Sunshine project.

Samples for assay have included soil, alfalfa, animals, dairy products, human bone, rain, well and spring water, snow, and many other similar materials from various parts of the United States and from countries all over the world. Some of the scientists were exceptionally eager to get started on the program and the first experiments had been performed before the 1953 conference adjourned. As a result, Sunshine was born with essentially three working laboratories—a group working at the Health and Safety Laboratory in New York City under the able direction of Merril Eisenbud and later Dr. John Harley; a group at Columbia University under the competent direction of Dr. Laurence Kulp of the Lamont Geological Observatory; and a group at the University of Chicago, which was initially under my direction and later under Dr. Edward Martell. A little later, Dr. Lyle Alexander, of the Department of Agriculture, became interested in Sunshine and has personally collected many of the extremely important soil samples which have been analyzed in the program. Subsequently, two commercial laboratories, Nuclear Science & Engineering Corp., in Pittsburgh, and Isotopes, Inc., in New York, were asked to assist in carrying the burden of isotopic analysis. In the last year or two, several other countries, notably the United Kingdom, have taken up the study, and the United Nations has organized, at the suggestion of the United States, the Scientific Committee on the Effects of Atomic Radiation with which all countries collaborate freely. Dr. Shields Warren, outstanding scientist and medical doctor, and the first director of our Division of Biology and Medicine, heads the United States delegation. This international committee has been very beneficial. The information furnished by the cooperating countries has been a substantial and significant addition to ours.

Originally, Project Sunshine was focused on the study of strontium 90 fallout and its effects, but with the passage of time, Sunshine has come to mean all of the Commission's activities directly connected with offsite fallout so that any summary of Sunshine activities must include the important work of Dr. Austin Brues and his collaborators

at the Argonne National Laboratory on the toxicity of strontium 90; of Dr. Cyril Comar at Oak Ridge on the metabolism of radiostrontium in humans and animals; Dr. William Neuman at the University of Rochester on the depoition of strontium 90 in bone; Dr. Kermit Larson, of the University of California at Los Angeles, on fallout materials in plants, and many others. At the present time, there are upwards of 50 contracts with outside laboratories bearing upon Sunshine and, in addition, work at the Commission laboratories.

Representative HOLIFIELD. Dr. Libby, this is a very impressive statement of the different projects which are engaged in in studying the effects of radiation. We asked Dr. Dunham for specific information in regard to numbers of people employed either directly or through contract. If that information is available, I believe this would be a good place to put it in.

Dr. LIBBY. All right, we will see that it is done, Mr. Chairman.

Representative VAN ZANDT. Mr. Chairman?

Representative HOLIFIELD. Mr. Van Zandt.

Representative VAN ZANDT. Dr. Libby, will you put in the record the 50 contracts, the names of the companies, as well as the amount of money involved, and the number of personnel employed?

Dr. LIBBY. We would be very pleased to do that.

Representative VAN ZANDT. And the nature of their studies?

Dr. LIBBY. Yes, sir.

Senator ANDERSON. Since there has been an interruption, maybe I can go back a bit.

You mentioned the work of the United Nations in organizating the Scientific Committee on the Effects of Atomic Radiation, and Dr. Shields Warren, whom we all recognize as a very fine person, heading the United States delegation. Is there a geneticist on the United States delegation?

Dr. LIBBY. The composition—I will have to ask Dr. Dunham to answer that.

Senator ANDERSON. I think we had some testimony from geneticists there was none.

Dr. DUNHAM. The official representative of the United States is Dr. Shields Warren. He has two alternates, Dr. Austin Brues, and Mr. Merril Eisenbud.

At the last meeting of the United Nations Scientific Committee, which was held in Geneva in April, he took with him as consultants, as did the representatives of the other member countries of this committee, two outstanding geneticists from this country—Dr. George B. Beadle of California Institute of Technology, and Dr. Sterling Emerson of the Division of Biology and Medicine.

Does that answer the question?

Senator ANDERSON. The answer to the question, then, would be, "No, there is no geneticist on the United States delegation." Is that not correct?

Dr. DUNHAM. That is absolutely correct.

Senator ANDERSON. It is a simple answer.

Dr. DUNHAM. There can be only one official delegate.

Representative COLE. How large is the official delegation?

Senator ANDERSON. And two people who accompanied him?

Dr. DUNHAM. Yes.

Senator ANDERSON. Who are these others that go along if they are not delegates?

Dr. DUNHAM. They are so-called alternates who may go along, and particularly act for the—officially act for the representative in his absence.

Representative COLE. Are either of the alternates specialists in any one field of biology?

Dr. DUNHAM. Dr. Austin Brues is a specialist in radiation pathology. He is an outstanding authority on the subject. Your committee asked him to testify as he did the other day.

Mr. Merril Eisenbud is not a biologist. He, however, is, as Dr. Libby has just indicated, the one who has been a prime figure in the development and accumulation of the information we have in this country on fallout. Therefore, it was felt appropriate that he be an alternate, because he lived this problem day in and day out.

Representative HOLIFIELD. Dr. Dunham, could you furnish now, early, for the benefit of the press and the audience, the numbers which we asked you for the other day? I think it would fit in at this point in the record.

There is a great deal of propaganda going around throughout the Nation that we are not concerned about this matter governmentwise, and I think this would help to show that there is concern on the part of the Congress and the Atomic Energy Commission, and that there is considerable effort both in terms of personnel and in money being devoted to a study of the problem.

Dr. DUNHAM. I have here a document I believe the committee already has copies of (p. 1393). I was planning to give it to you for tomorrow. I will read you the first page.

Scientic man-years for sampling and analysis of radioactive fallout, including fission products, toxicity and transport, 253.

The effects of radiation on humans, mammals, and other organisms, exclusive of genetic studies, 449.

Treatment and methods of ameliorating radiation effects, 60.

Genetic effects of radiation, studies on human genetics, 12.

Experimental studies on the genetic effects of radiation on species other than man, 71.

Biochemical and microbiologic studies of radiation effects, 110.

Environmental studies, 4.

Dosimetry research, development of improved methods of measuring fallout, 47.

A total of 1,006 scientific man-years.

Representative HOLIFIELD. Thank you very much.

Do you have a complete presentation for tomorrow?

Dr. DUNHAM. It is already in the hands of the committee, I believe.

Representative COLE. You confused me by that last expression, "scientific man-years." Get it down to the number of people the Commission has engaged, either direct by contact, in the study of biological aspects of radiation hazards.

Dr. DUNHAM. The figure, if we gave you numbers of people, would be much larger, but it would also be misleading, because many people do not spend full time on this work.

Representative COLE. Give me the total number, and allow me to decide how far I have been misled.

Dr. DUNHAM. I do not have that number, but I will get it for you, Mr. Cole. I will be happy to get it.

Representative COLE. Would it be in the order of three or four thousand?

Dr. DUNHAM. It would be in that range, yes; I am sure.

Representative VAN ZANDT. Mr. Chairman?

Representative HOLIFIELD. Mr. Van Zandt.

Representative VAN ZANDT. Dr. Dunham, I asked a question the other day, but I think it should be repeated at this point in the record.

Are there any independent studies being made of the radiation hazards, that is, independent of Government, independent of the Atomic Energy Commission?

Dr. DUNHAM. If you are talking about the actual research work and the collection of data, I believe the Atomic Energy Commission is responsible and supporting by far the large majority of that work which is done in this country. If it is a matter of evaluation of the data, as you know, the National Academy of Sciences has a continuing group of committees reviewing this problem. They have been meeting ever since their report a year ago, and will continue to do so, and review the subject.

Representative VAN ZANDT. Dr. Dunham, I imagine that the AEC has access to the studies of the independent groups?

Dr. DUNHAM. That is right. We are kept in touch with each other.

Dr. LIBBY. But these groups are entirely independent, Mr. Van Zandt, in their functioning.

But on the point of geneticists, Senator Anderson, I assure you we have no intention of slighting in any way the subject of genetics. We consider it to be very important.

Senator ANDERSON. I assume you have heard about the testimony of Dr. Glass.

Dr. LIBBY. I have not read it; I am sorry, but I read the morning papers.

Senator ANDERSON. I am sure your agency does not differ from any other. You have them reporting to you what is going on.

Here is a group that met 22 times in the last 3 years while Dr. Glass has been a member of it. He had been hopeful, I guess, sometime that the Atomic Energy Commission would ask him what they thought about the project, but nobody thus far has.

Dr. LIBBY. You see the committee reports to Dr. Dunham, and I would like him to comment upon that.

Dr. DUNHAM. The committee reports through me to the Commission.

Representative COLE. What committee are you talking about?

Dr. DUNHAM. I assume Senator Anderson is talking about the Advisory Committee for Biology and Medicine.

Senator ANDERSON. Exactly, which is the one Dr. Glass testified about.

Dr. DUNHAM. That is correct. I think the record will show that on a number of occasions Commissioner Libby has specifically requested that this subject be on the agenda. It has been discussed at practically every one of the meetings in the last 2 years.

Senator ANDERSON. Were any members of the committee there?

Dr. DUNHAM. Members of——

Senator ANDERSON. Any members of the Advisory Committee there, or in absentia?

Dr. DUNHAM. This is the Advisory Committee meetings, I am talking about. At their meetings.

Senator ANDERSON. You were not trying to imply it had been brought up by Dr. Libby at the meetings of the Atomic Energy Commission?

Dr. DUNHAM. Oh, no. That is something else.

Senator ANDERSON. You allowed the Advisory Committee to talk about what it is supposed to advise about?

Dr. DUNHAM. That is right.

Senator ANDERSON. But do not let it get to the Atomic Energy Commission.

Dr. DUNHAM. I do not——

Senator ANDERSON. You say that Dr. Libby has asked this be put on the agenda. The agenda of what?

Dr. DUNHAM. The Advistory Committee.

Senator ANDERSON. The very purpose for which it is created.

Dr. DUNHAM. That is correct.

Senator ANDERSON. He asks that the subject for which it is created be put on its own agenda?

Dr. DUNHAM. Oh, no. He asks that they specifically consider the status of project Sunshine. He has done that on several occasions. So has Commissioner Murray.

Senator ANDERSON. How many times has the report of the Advisory Committee been an item on the agenda of the Atomic Energy Commission that you recall?

Dr. LIBBY. The Committee makes a report to the Commission every meeting, does it not?

Dr. DUNHAM. Yes. After each meeting they write a letter summarizing their deliberations and any recommendations they might have.

Senator ANDERSON. I guess you misunderstood my question. My question was: How many times has a report of the Advisory Committee been on the agenda of the Atomic Energy Commission?

Dr. LIBBY. I do not believe that the reports, as such, ever are on the agenda, Senator Anderson, but that does not mean we do not give them the most serious consideration.

For example, the General Advisory Committee's report is never, as such, on the agenda. Items which they report on and recommendations which they make will be on the agenda, but the Commission is reported to through the mechanism of a letter to the Chairman of the Commission, which is circulated to all members of the Commission. Just a technicality. It does not go on the agenda. We do pay attention to it. We certainly are most interested in supporting genetic research, and furthering its investigations in every way.

Representative COLE. Dr. Libby, a point of clarification. This Advisory Committee covers all aspects of the biological consequences of radiation, does it not?

Dr. LIBBY. All aspects of biology and medicine.

Representative COLE. Of which genetics is one element?

Dr. LIBBY. Yes.

Representative COLE. And I understand from Dr. Dunham's statement that you, as a Commissioner sitting in with this Advisory Com-

mittee, have from time to time specifically asked the Advisory Committee to give consideration to the genetic effects of radiation?

Dr. LIBBY. I cannot recall——

Senator ANDERSON. He did so testify.

Dr. LIBBY. I cannot recall specifically emphasizing the genetic rather than the somatic hazards, but we have repeatedly made it clear to the Committee that any words of advice or conclusions that they have on any aspect of the effects of radiation, whatever its origin be, be it weapons tests or accidents in peaceful uses—or whatever its origin—we certainly want the information.

Representative VAN ZANDT. Dr. Libby, let me ask you this question: In your opinion, as a commissioner, do you think that the subject of genetics has been adequately covered?

Dr. LIBBY. I am not a geneticist, and I know so little about the subject of genetics, Mr. Van Zandt, that I hate to answer that question. It seems to me that I do not know the answer. I know we do want to encourage good, capable research men to work in this field in every way we can, and I am sure Dr. Dunham can testify more definitely to your question. I would like to ask him to.

Dr. DUNHAM. I think I can best answer it, Mr. Van Zandt, by calling your attention to the fact that the Advisory Committee for Biology and Medicine as always had a geneticist on it. This indicates the preoccupation of the Commission with the genetics problem.

The first Committee included Dr. George Beadle, of Cal-Tech. Following his 5-year stint, Dr. Kurt Sturn, of the University of California, took over, and currently it is Dr. Bentley Glass. Furthermore, since 1950, the Division of Biology and Medicine has always had a full-time geneticist of considerable statute on its staff, so as to be certain that this area would not be neglected.

Representative COLE. How large is the Advisory Committee?

Dr. DUNHAM. Seven people, I believe.

Representative COLE. And 1 out of the 7 has always been a geneticist?

Dr. DUNHAM. One has always been a geneticist.

Chairman DURHAM. That is just the Advisory Committee on Biology and Medicine?

Dr. DUNHAM. That is correct.

Chairman DURHAM. That does not include the Advisory Committee on the whole thing?

Dr. LIBBY. The law sets up the General Advisory Committee, and, because of the existence of the Advisory Committee on Biology and Medicine, there has been a tendency, I think, to not appoint biologists to the General Advisory Committee. So we have never had—I do not recall there ever has been a biologist or medical doctor on the General Advisory Committee. But it is for the reason that we have this special Committee for the area of biology and medicine.

Senator HICKENLOOPER. Mr. Chairman, may I verify this with Dr. Libby? As I understood the testimony of yesterday, first, the General Advisory Committee is an arm of the Commission; that is, it reports directly to the Commission?

Dr. LIBBY. Yes.

Senator HICKENLOOPER. And the Advisory Committee on Biology and Medicine is also an arm of the Commission and reports directly to the Commission. Is that right?

Dr. LIBBY. Well, it reports through the Director of Biology and Medicine, though I think the Committee certainly knows we are very glad to have them talk directly to the Commission at any time, and I try to attend the meetings when I can. I think your statement is correct, Senator Hickenlooper; yes. But there is a statutory connection which exists for the General Advisory Committee, which does not exist for the other Committee. You will not find the Advisory Committee on Biology and Medicine specifically mentioned in the Atomic Energy Act of 1954, I believe.

Dr. DUNHAM. May I amplify this?

Representative HOLIFIELD. Let's get to the main part of the testimony, now, and then we can have——

Senator HICKENLOOPER. If there is some amplification at this point, Mr. Chairman, I would like to hear from Dr. Dunham.

Representative HOLIFIELD. Go ahead, Doctor.

Dr. DUNHAM. I merely wanted to state that, when I said earlier the Committee reports through the Director of the Division, this is not actually in fact so. They report directly by letter, the letter that goes between the Chairman of the Committee and the Chairman of the Atomic Energy Commission.

Representative HOLIFIELD. And how does that differ from the General Advisory Committee? Do they report through the General Manager, or do they report by letter, or do they report directly to the Commission?

Dr. LIBBY. The Chairman of the General Advisory Committee reports by letter to the Chairman of the Commission.

Representative HOLIFIELD. Proceed.

Dr. LIBBY. At first it was believed that the work of Project Sunshine was so intimately concerned with weapons that it was necessarily secret, but it presently became apparent that more and more aspects were declassifiable; consequently, a year or so ago, virtually every aspect of the Sunshine project not already unclassified was declassified. It is also true, of course, that many of the problems which we think of today as part of Project Sunshine were not so considered in the beginning and had been in the unclassified areas since their inception. The information obtained has been issued publicly. With one exception, I do not know of any significant information on fallout in the possession of the Commission which is not available to the public.

Representative HOLIFIELD. Is that exception in the classified area?

Dr. LIBBY. Yes. That is in the next sentence. The exception is certain facts which would reveal information concerning intelligence and weapon design and, therefore, cannot be made public; however, your committee has access to these facts in executive session. The portion now classified is only a small segment of the large body of knowledge about worldwide fallout and, although important, is not vital in the understanding of fallout, its effects and its hazards.

There is another point I would like to make at this time about the study of worldwide fallout and that is that it is a scientific study. Thus it has as its objective just one thing, truth, the scientific truth. We have a consistent policy of encouraging all competent scientists, both here and abroad to contribute to the study. Many of these contributions can be comparatively indirect and can be carried out in most research laboratories as an adjunct to the regular work. In this con-

nection, I have frequently suggested particularly to those critics of the Commission's weapons testing program who are competent scientists that any contributions they might make in the way of scientific theories, data, or experiments would be most welcome.

Representative COLE. Mr. Chairman?

Representative HOLIFIELD. Mr. Cole.

Representative COLE. Would the chairman allow me to interrupt Dr. Libby again with reference to the studies of worldwide aspect of fallout?

Reverting to your system of sampling which you have indicated is substantially worldwide, as widely distributed as possible, do those studies support the assertions which have been made by some that the distribution of fallout is not widespread, is not general throughout the world, but rather is concentrated in the Northern Hemisphere?

Dr. LIBBY. Mr. Cole, the interpretation—I may say this: The only data that exists are the data Project Sunshine have collected and published. There are not any other data, except those few that have been collected in the last year or so by the other countries. So the majority of them are ours and whatever difference in interpretation there may be, or difference in statement, must be based on the same data.

Senator ANDERSON. I hope we talk about that, but I was going to wait for that portion of your statement, where you talked about the uniformity of stratospheric fallout.

Dr. LIBBY. Yes, sir; I would be very glad to discuss this question.

Representative COLE. All right.

Representative VAN ZANDT. Mr. Chairman?

Representative HOLIFIELD. Mr. Van Zandt.

Representative VAN ZANDT. Dr. Libby, this morning's Washington Post carried an article by Edwin Diamond, in which he refers to a scientific document entitled "Preliminary Data on the Effects of Atomic Bomb Explosions, and the Concentration of Radioactivity in Lower Atmosphere and Soil."

This is supposed to be a Russian document. Does the Commission have this document, or a copy of it in its possession?

Dr. LIBBY. Well, Mr. Van Zandt, I do not recognize it by the title, and I read every article that comes to the U. N. Radiation Committee from whatever source. That does not mean that I have not read it, and that we do not have it. I do not recognize the title.

I would say, from what Mr. Diamond said in the paper, that I have not read it. Maybe it is real new and we just have not seen it yet.

Representative VAN ZANDT. Thank you.

(A clarifying letter from Dr. Machta follows:)

JUNE 9, 1957.

To: For the record.
From: Chief, Special Projects Section, OMR, United States Weather Bureau.
Subject: U. S. S. R. fallout document.

Several questions by members of the Joint Committee on Atomic Energy at the fallout hearings dealt with a U. S. S. R. document entitled "Preliminary Data on the Effects of Atomic Bomb Explosions on the Concentration of Artificial Radioactivity in the Lower Atmosphere and in the Soil," edited by B. M. Isaev and D. L. Simonenko, Moscow, 1956, which was mentioned in an article by Mr. Diamond of INS in many newspapers last week.

Our office first showed this document to the reporter many weeks ago, as a result of inquiries about U. S. S. R. contributions to the International Geophysical Year in nuclear radiations. Mr. Diamond, however, neglected one fact in his

article, which the undersigned and Mr. R. J. List, of the United States Weather Bureau, gave him and repeated at least a half dozen times. This is the fact that at the recent meeting in Geneva of the United Nations Scientific Committee, the U. S. S. R. has withdrawn the data included in the report. In other words, there is no authentic fallout data from the U. S. S. R., insofar as we are aware. This is contrary to the facts of the newspaper article. My personal opinion is that the data in the report of the U. S. S. R. are wrong and withdrawal is justified.

LESTER MACHTA.

Dr. LIBBY. Our policy is to discover the truth about fallout and to make it public.

The Commission has provided its data to the United Nations Scientific Committee on the Effects of Atomic Radiation, which I mentioned a moment ago. Of course, the scientists employed by our contractors, and who are working in this field are encouraged to publish their findings in scientific journals—a practice which insures the availability of the information to all scientists everywhere. Any data or information which is not suitable for publication in scientific journals, but which has sufficient merit to warrant its distribution, can be and is published through the Technical Information Service Extension at Oak Ridge.

If one takes the summer conference at RAND in 1953 as the real beginning of the worldwide fallout study, then we began by considering all known aspects of the problem, and by planning a careful experimental attack on each of them. A continuous review has been going on ever since, conducted principally by the scientists engaged in the program, but vitally assisted by the study, The Biological Effects of Atomic Radiation, made last year by the National Academy of Sciences, and the continuing advice of the Advisory Committee on Biology and Medicine and the General Advisory Committee. Whenever it appears that some facet has been overlooked or is not receiving enough attention, an immediate effort is made to get the additional work going. This method of continuous criticism and evaluation has enabled the program to be productive and effective.

It is fortunate for our national interest—and for that matter the national interest of other countries as well—that this has been so, because were it not for the efforts of the scientists of Project Sunshine—they are relatively very few in number—not much would be known about worldwide fallout. As it is, the broad aspects of worldwide fallout are understood. In this connection, Dr. Dunham has asked that you put in the record 11 papers and technical speeches with detailed data. (See p. 16.)

As regards the physical rather than the biological facts about fallout, many possibilities still exist for minor controversy over detail, but no scientist who takes the trouble to learn what the facts really are will fail to agree with the overall picture. I know of no scientist who has studied the data who does not agree on the general amount of fallout received, the amount of strontium 90 in the body, the rainfall effect, and other features such as the stratospheric reservoir and the storage there for a time of many years. We still are not certain that 10 years is the best figure, but everyone agrees that it is a matter of years and the uniformity of stratospheric fallout is still under study as Dr. Machta has made clear.

Senator ANDERSON. Now that is the point where I would like to ask you a question, because you say the scientists do agree on the overall picture.

Dr. LIBBY. Yes.

Senator ANDERSON. Dr. Machta testified in here, and it seemed to me he testified that the stratospheric fallout was not uniform. I will just quote from him.

> After 2 years debris in the atmosphere from our Castle tests is still not uniformly distributed in the stratosphere. The upper air program of the Atomic Energy Commission can check this thesis in the near future.
>
> Delayed fallout has not been deposited uniformly over the earth. On the average, there is more delayed fallout in the north temperate latitude, even though the main injection was in the tropics, that is, the Marshall Islands.

Incidentally, this deposition of worldwide fallout was confirmed, I believe, by both Dr. Kulp and Mr. Eisenbud, who showed that the Northern Hemisphere had from 2 to 3 times as much strontium 90 as the Southern Hemisphere.

How do these check with your statement made on the 26th of April on uniformity?

Dr. LIBBY. I think they check very well. But let me explain that apparently paradoxical reply.

Senator ANDERSON. To a layman uniformity and nonuniformity are quite different terms, and you have used the term "uniformity" here again, and he keeps using the term "nonuniformity," Doctor. How do you put those into the same church?

Dr. LIBBY. I would like to use a chart for the rainfall in the Chicago area in the year 1955. I thought by selecting this one narrow thing I could explain it better.

Senator ANDERSON. It may be very helpful. But do you believe that strontium 90 is deposited uniformly over the world?

Dr. LIBBY. No, sir; and I never said that, sir. What I said was different—that the stratospheric components of the fallout might very well be uniform, and for the time being until we know better, we would so assume.

So the argument, really, if there be one—and I do believe it to be minor—is whether the stratospheric components of the fallout is uniform, Senator Anderson.

Senator ANDERSON. Do you think that sort of distinction occurred to the average person who is groping for information in this field?

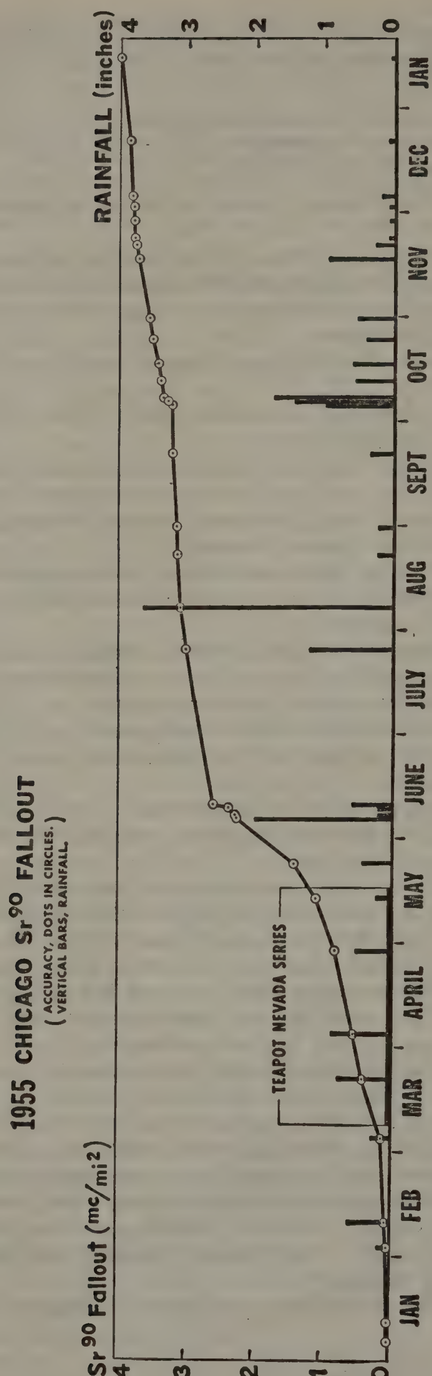
Dr. LIBBY. I am afraid not, and I am sorry to have to agree with you, Senator. I think it is not. We have tried hard to explain the nature of the fallout from what we call the lower atmosphere, the troposphere. This stuff has no time to mix worldwide and comes down in the same general latitude as the bomb was fired.

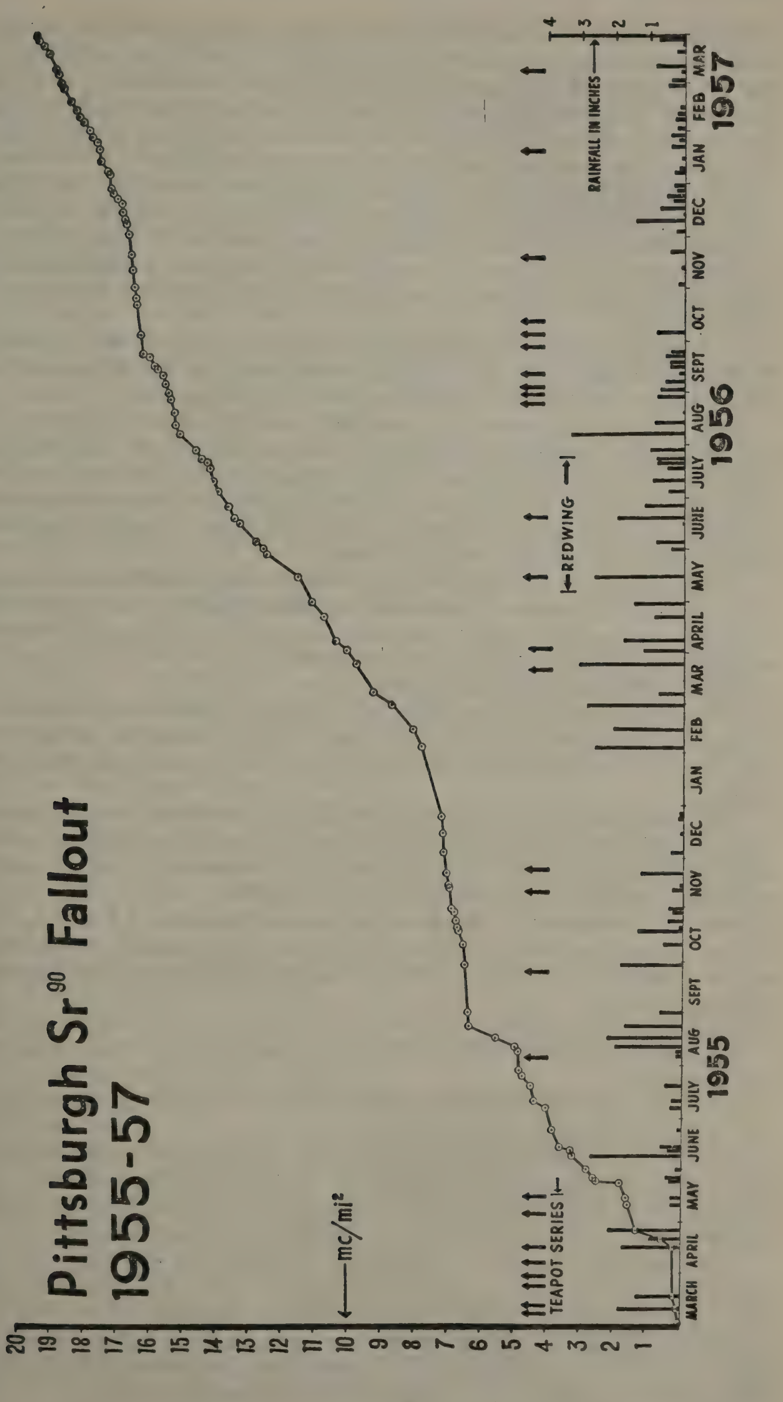
The stuff that goes upstairs into the stratosphere stays so long that it has time to mix pretty much all over the world.

Now, the real point of discussion here, Senator, is this stratospheric material, which comes from large-yield megaton-class bombs—though it could come from small bombs if they were fired in the stratosphere—but the material that gets upstairs into the stratosphere and stays there for a matter of years. And Dr. Machta agrees with this.

Now the question is: How thoroughly does this mix before it comes down in its slow fashion?

This figure I have over here perhaps will help me explain it to you.


1955 CHICAGO Sr^{90} FALLOUT
(ACCURACY, DOTS IN CIRCLES.)
(VERTICAL BARS, RAINFALL.)
Sr^{90} Fallout (mc/mi^2)
RAINFALL (inches)
TEAPOT NEVADA SERIES
4
3
2
1
0
JAN
FEB
MAR
APRIL
MAY
JUNE
JULY
AUG
SEPT
OCT
NOV
DEC
JAN


Pittsburgh Sr90 Fallout
1955-57
mc/mi²
TEAPOT SERIES
REDWING
RAINFALL IN INCHES
MARCH APRIL MAY JUNE JULY AUG SEPT OCT NOV DEC JAN FEB MAR APRIL MAY JUNE JULY AUG SEPT OCT NOV DEC JAN FEB MAR
1955
1956
1957

These are washtub data from the city of Chicago for the year 1955, where a washtub was placed on the roof of one of the buildings of the University of Chicago, and left out there continuously. It was only when it rained that we would empty the water and the solids that had accumulated and take those into the laboratory, and analyze them. The size of the dots indicates the error of the analysis.

I think you will notice at a glance that there is one big cliff in that curve of the strontium 90 fallout against time, which begins in January 1955, and extends throughout that year into January 1956.

There is a big cliff, and it is obviously correlated with the time at which we started firing small bombs in the Nevada test site, the so-called Teapot series.

So I say that of all of the fallout which occurred in the year 1955 in this particular area, on this particular roof, on the University of Chicago campus, most of it came from the Nevada series.

Now this is a difference of point of view which is not serious, in the sense that further information will straighten it out, and the difference for the prediction for the future is not measurable.

Dr. Machta and we agree about the occurrence of the stratospheric storage. We are not in disagreement. We have a range of opinion about the length of time it stays there.

The point about this band of stuff which lies in our latitude is that I say it is due to material that was deposited quickly, and I think Dr. Machta, to a certain extent, implies that part of that came from the stratospheric fallout which had not mixed.

Well, we do not know the answer to this.

I know that we have no place in the world where any rainfall at all occurs where we do not find fallout. I have measured snows from the South Pole, and every bit taken from the surface has detectable fallout.

We have had testimony from Dr. Kulp that bones from whatever part of the world, whatever part of the Southern Hemisphere, showed detectable strontium 90. The only places in the world where you do not find it are places where it has never rained.

Senator ANDERSON. I think we had one witness—and I will not try to recall his name—who testified that in the Latin American countries, or the Southern Hemisphere, it was half what it was in the Northern Hemisphere.

Dr. LIBBY. That is true.

Senator ANDERSON. Would that prove or disprove this theory of uniformity?

Dr. LIBBY. I think, Senator, that you and I understand; we must try to make this clear to everyone now.

You see that cliff there is not from the stratosphere, in my opinion. That cliff, that rapid rise in March, April, and May of 1955, was material that came from Nevada which had just come over. It did not go worldwide. That did not get in the Southern Hemisphere.

The flatter portion which you have there, in my opinion, was just about the same in the Southern Hemisphere. Now Dr. Machta says it was less. I do not think we know the answer, Senator.

Senator ANDERSON. I will read what you said on just the previous page. You said, "but no scientist who takes the trouble to learn what the facts really are will fail to agree with the overall picture."

Dr. LIBBY. I think that is right.

Senator ANDERSON. And you just got through saying you believe one thing and Dr. Machta something else.

Dr. LIBBY. No; he believes the same thing.

Senator ANDERSON. And naturally the rest of us get confused.

Representative PRICE. Will the gentleman yield?

Senator ANDERSON. Yes.

Representative PRICE. I think Dr. Machta said his findings—when questioned about your statement of April he said the disagreement came because of the fact that he was making his statement based on later findings.

Dr. LIBBY. I guess that is true, sir. I did not want to raise that point though. I mean it must be true; yes. That is, I am perfectly willing to change our mind if our stratospheric sampling—you see, we have a program now of going upstairs and actually finding out how much is there. This program is going and we will know this answer—and it is not very far away.

Representative HOLIFIELD. I think we are missing the point, if the Senator will yield. The point is not that there is a uniform distribution in the stratosphere. The point is, is the fallout from the stratosphere uniform or is it uneven, and I think Dr. Machta showed where the breaks in the stratosphere occurred and the downwinds came through those breaks that this was conducive to depositing the material unevenly on the earth's surface. Do you agree with that statement?

Dr. LIBBY. Certainly Dr. Machta is a most eminent meteorologist, and I am not a meteoroligist. I think your point is very well taken, Mr. Holifield, and may very well result that there is a band in both hemispheres, you see. That would follow from this analysis you have given us—that, if the mixing is at all uniform worldwide, there could be a band in both hemispheres.

Representative HOLIFIELD. It is also true, is it not, that the terrestial winds, the so-called jet winds, are more concentrated over the temperate zone, and as the material comes into a stream from other zones, why, it would tend to concentrate in the temperate zone; would it not?

Dr. LIBBY. Yes.

Representative HOLIFIELD. Your answer to that was "yes"?

Dr. LIBBY. Yes; I believe so.

Representative HOLIFIELD. On the chart over here, this really has very little, if anything, to do with stratospheric fallout. It is mostly the tropospheric fallout; is it not?

Dr. LIBBY. I think the stuff before and after the cliff is largely stratospheric.

Representative HOLIFIELD. You mean from January to March?

Dr. LIBBY. January to March, and then from——

Representative HOLIFIELD. That would be the stratospheric?

Dr. LIBBY. Yes. After the Teapot, Nev., series did you have a rise in the fallout, but that was tropospheric and not stratospheric. That was kiloton bombs.

Senator ANDERSON. Dr. Libby, in that speech in April you said:

> For air-fired megaton weapons, our present indication is that the fallout is almost worldwide; and for reasons of simplicity, and in the absence of better information at the present time, we work on the model that this is a uniform distribution over the entire world of the material that falls from the stratosphere.

If you had to assume something, why not assume what the results show—that it was nonuniform distribution?

Dr. LIBBY. You see, sir, my interpretation of the results would be that they are not conclusive in showing nonuniformity.

Senator ANDERSON. No, but 50 percent of this radioactive material fallout of fission products goes into the stratosphere. Now it is quite significant if you start to assume all of this is going to come down evenly, but if you start to assume it may not come down evenly, there may be a band where deposits are heavy, it might affect people in the Northern Hemisphere, it might really concern them, and you might have to revise, it would seem to me, your estimate of the future fallout pattern in the United States.

I am only trying to say, why do you always say "uniformity," when the experience shows nonuniformity?

Dr. LIBBY. The experience does not show nonuniformity on the stratosphere, Senator, definitely.

Senator ANDERSON. I do not say that. You twisted it to mean something else. I said: "Why do we always have to assume uniformity in the fallout pattern of these materials, when all of the experience shows nonuniformity in fallout?" I am not talking about what is upstairs.

Dr. LIBBY. We certainly should not do that, and we have never done that, Senator; and I think it is important to make that clear.

Senator ANDERSON. You mean this speech does not assume that?

Dr. LIBBY. That speech is correct as far as I know.

Senator ANDERSON (reading):

We work on the model that this is a uniform distribution over the entire world.

Dr. LIBBY. Stratospheric, sir.

Representative COLE. Senator, read the rest of the statement.

Senator ANDERSON. I have read it with care.

Representative COLE. You will see it is not——

Senator ANDERSON (reading):

Further evidence and data on this are rapidly being collected which will undoubtedly settle the stratosphere horizontal mixing question.

That does not give me any more comfort than the first statement. You assume uniformity even here this morning. After Dr. Machta's very enlightening discussion, you start with the uniformity of stratospheric fallout. Why do you use the word "uniformity"? Why don't you use the word "nonuniformity"?

Dr. LIBBY. Because it is a question which is subject to some—like all scientific conclusions, you take the data and you draw conclusions from the data. The fact that banding of fallout occurs around our latitude, Senator, nobody argues about. Nobody argues about that. We do have this heavy banding in our latitude. And in my opinion it is due very largely to young fallout which never has been up there for years. It has been up there for weeks or months, and has not had time to mix. This difference of opinion is going to be settled. I do not believe that it is a major controversy. It is a minor one and will not seriously affect the general conclusions of this hearing.

Senator ANDERSON. It affects tremendously the question of how much fallout is safe, how much testing is safe, because if you assume that the pattern is uniform around the world, when actually it is 2 times or 3 times heavier in a given place, then you have, by this as-

sumption, lowered the possibility of damage from fallout. And the question is: Is that the reason why we say fallout is uniform over the whole world, whereas if we calculated it directly according to the way it is coming down, we might get a different answer?

Dr. LIBBY. You are certainly right, Senator.

Senator ANDERSON. That is my only point.

Representative HOLIFIELD. Dr. Libby, may I clear up one technical point here?

Dr. LIBBY. Yes.

Representative HOLIFIELD. The figure has been used in testimony before this committee of 50 percent of the fission products of a weapons test going into the stratosphere. Let us clear up now, if we can, that we cannot rely upon the 50 percent, and I ask you this question: Does it not depend upon the size of the weapon as to how much goes into the stratosphere? And therefore the percentage would vary from a large weapon, let us say, from as much as 75 percent going into the stratosphere and a smaller weapon that would puncture the stratosphere might deposit 25 percent, and a still smaller one would not go into the stratosphere at all? Is that clear? And if it is not, will you please clarify it?

Dr. LIBBY. It is quite true, Mr. Holifield, all that you have said. The statement about 50 percent is sort of rough. I mean there is nothing magic about that number.

Representative HOLIFIELD. No.

Representative COLE. On that point, if I may, Mr. Chairman; the size of the weapon is not the only factor which determines the amount of material that goes into the stratosphere, is it?

Dr. LIBBY. Oh, no, no.

Representative COLE. What are the other factors?

Dr. LIBBY. There are many factors. For example, if you took a kiloton bomb upstairs and fired it, it would be in the stratosphere.

Representative COLE. Well, the altitude has a great deal to do with it?

Dr. LIBBY. Surely.

Representative COLE. Does the nature of the weapon have anything to do with it?

Dr. LIBBY. The yield certainly determines whether it is going to push up the yield, among other things. That is, if you fire two bombs at the same distance above the earth's surface, the megaton bomb will get into the stratosphere and the kiloton bomb will not. Of course that is a rough general statement, too. I mean a 1-megaton bomb would not all go into the stratosphere, and some of the 100-kiloton bomb would go into the stratosphere.

Representative HOLIFIELD. And it is true, as you said, a thousand-ton bomb, if projected in the stratosphere by missile, would release all radioactive material in the stratosphere.

Dr. LIBBY. It would all be in the stratosphere.

Representative HOLIFIELD. In my evaluation I was, of course, talking about ground explosions.

Dr. LIBBY. Yes, I understood that, Mr. Holifield. But I thought it might be interesting to make this point that even kiloton bombs can give stratospheric fallout in the southern hemisphere if they are fired high enough in the air.

Senator ANDERSON. I was about to ask you how many people live in the stratosphere, but let me get down to this: Since the worldwide average is not calculable if it did fall down in a uniform pattern, would it now appear desirable or necessary to raise the predictions for the Northern Hemisphere, including the United States?

Dr. LIBBY. We certainly, in considering this tolerable limit should consider the fallout pattern, Senator, not a theoretical or simplified one. One should take this into account, those bumps, like that [indicating] which are due to Nevada. We have to add those things in there.

Yes. The answer to your question is, "Yes," one must take these into account. To reach an overall simplified figure, it might be justified to say, "If we fired everything in the stratosphere, this would be the figure." Or to take another one. If we fired everything in the troposphere, this would be the figure. But in the actual determination, you must take the actual figures for the fallout as distributed rather than depending on some theory. This is certainly true.

To proceed with my statement:

These essential points are generally agreed and the questions under debate are really largely political and sociological. For example, we agree that the extra radiation from the test fallout is a small fraction of the natural dosage we receive from our own bodies, our surroundings, the cosmic rays and a very small fraction of X-ray doses taken by many individuals.

Representative COLE. Mr. Chairman.

When you say, "We agree," who are you talking of?

Dr. LIBBY. I am thinking of the scientists who are studying the physical part of the Sunshine project; that is, the fallout, worldwide fallout.

Representative COLE. You are referring to the scientists who are directly engaged in the Sunshine project?

Dr. LIBBY. Yes, sir. Dr. Machta, and me, for example.

Senator ANDERSON. I had not intended to interrupt, but in the previous sentence you say, "the questions under debate are really largely political and sociological."

These 2 or 3 weeks have been set aside for the examination of witnesses who are primarily scientific in their approach. Are we wasting our time on scientific testimony, and not getting the political and sociological?

Dr. LIBBY. Oh, no. I think it is very important. The service that the Congress has done in holding these hearings is tremendous, and I commend you on it. You have gotten the facts out in a way that is just fabulous. It would have taken years to get this information out in the normal course, it seems. And it is so wonderful. No, sir, it has not been a waste of time.

Representative HOLIFIELD. At this point, Dr. Libby, because of the comment you made, you studied our agenda, which was prepared with a great deal of care and with a great deal of scientific advice by our staff members and scientific consultants, and you have been to some of these hearings. Do you consider that these hearings are being held in a fair and impartial manner, and in a beneficial manner to the people?

Dr. LIBBY. I certainly do, Mr. Holifield. You could not have done a better job, in my opinion. It is possible you might have taken up

some other subjects, but I would not have been able to devise a better program or set of hearings than you have conducted. I certainly commend you for it. No criticism at all.

Representative HOLIFIELD. We asked for suggestions, as you know, from the AEC.

Dr. LIBBY. Yes.

Representative HOLIFIELD. On the preparation of the agenda. And of course, we have tried to limit it as much as possible to scientific testimony.

Dr. LIBBY. Yes.

Representative HOLIFIELD. Now we recognize that there are other fields, sociological, political, and moral fields, of interpreting the effects and the reasons for having these tests; but it is not the purpose of the committee to get into that in these hearings. We are trying to collate a great amount of scientific information, so that people can make their decisions on information and statistical data from reputable sources, rather than upon uninformed statements.

Dr. LIBBY. Yes. I certainly commend you for it.

Senator HICKENLOOPER. Mr. Chairman?

Representative HOLIFIELD. Senator Hickenlooper.

Senator HICKENLOOPER. May I ask Dr. Libby about my interpretation of his statement, which, of course, came to my attention when he read it, that—

> These essential points are generally agreed and the questions under debate are really largely political and sociological.

I interpret that to mean not that there were not scientific data involved here at all, but that the public has been debating the sociological and political implications of this, and that is one of the great interests in the minds of the public which might be clarified, or some scientific truth be developed.

Dr. LIBBY. Yes.

Senator HICKENLOOPER. Which would dispel a great many fears and doubts, and all of those things that have been created by hysterical journalism on occasion, and headlines which seek, or which do, in effect, create fears and minimize the actual facts. I took that to be your reference.

Dr. LIBBY. That is the sense of it.

Senator HICKENLOOPER. I do not mean to have you pass on the question of distorted understandings of these things, necessarily, but there is a social and political concern in the country about these things which scientific facts may give many answers to.

Dr. LIBBY. That is right.

Senator HICKENLOOPER. I will frankly say I have seen many news stories which attract the avid interest of the people, and I have had telegrams on them about the terrible effects of these tests, which in my opinion are not borne out by the facts in these hearings, but still they get into the social and political thinking of people as a result of what at least to me is a misunderstanding of the actual facts and data that are brought out in these hearings.

Representative HOLIFIELD. Referring again to your sentence—

> These essential points are generally agreed and the questions under debate are really largely political and sociological.

would you not also modify that statement to include a difference of interpretation of the meaning of certain scientific facts?

Dr. LIBBY. Yes, I certainly would.

Representative HOLIFIELD. As well as political and sociological?

Dr. LIBBY. Yes.

Representative HOLIFIELD. There is an honest difference of opinion among scientists as to what the facts which they are agreed upon mean, how they should be interpreted in relation to the effect upon human beings; is there not?

Dr. LIBBY. There certainly is, particularly in the biological side, and particularly—well, particularly in the effects of fallout on the human body. There is not such a range of disagreement about the amount of fallout, or how much is where or how much has been there. There is not so much disagreement about these.

Representative HOLIFIELD. Particularly in the field of genetics.

Dr. LIBBY. That is my impression, Mr. Holifield.

Representative HOLIFIELD. There seems to be a very great deal of unanimity in the opinion of geneticists that people in the area of science in which they are most interested are more deleteriously affected than in the biological, physiological sense.

Dr. LIBBY. I have been reading the testimony yesterday with great interest. As I say, I am not a geneticist in any way, but it certainly is interesting to hear what the gentleman had to say. My impression agrees with yours.

Representative HOLIFIELD. You may proceed.

Dr. LIBBY. We agree that the radiation given new bone—as in children—from strontium 90 amounts now from 1 to 2 percent of the natural dose and that the increase in dose from a wide variety of ordinary experiences such as living in the mountains, moving from one locality to another where the difference in uranium and thorium content of the ground may be very different, living in a brick instead of a wooden house—all these normal experiences give doses which far exceed those from fallout. This we all agree is so.

Senator ANDERSON. Dr. Libby, right there. Do we also not then worry about what this maximum permissible dose is going to be when some of this fallout descends from the stratosphere?

Dr. LIBBY. No, sir, not at all. But I am showing here the wide range of general agreement on the physical aspects of the fallout problem. Everybody agrees that the fallout dosage is very small compared to both the natural dosage and the variation in the natural dosage.

Senator ANDERSON. Dr. Kulp, in his testimony, indicated that if we proceed at the same rate of testing, in 30 to 50 years we would get up to 25 to 50 percent of the maximum permissible dosage.

Dr. LIBBY. I am talking about the present time, Senator. The present time. You see, when you get into the future, you get into theory.

Senator ANDERSON. It is like erosion in my State. If you go out and look at a field, you will say, "We do not have to worry about runoff at the present time. It is not doing anything but dipping a little bit." And then it dips a little bit more, and finally you have a gully. We are interested in what happens 50 years from now.

Dr. LIBBY. Absolutely, we certainly are.

Senator ANDERSON. That is the reason why we might recognize this. What do we do with the acceleration of tests? If we have new countries coming in with tests, and the tests pick up, we raise this figure of 1 to 2 percent very decidedly, do we not?

Dr. LIBBY. Where is the wide difference of opinion? Why do some say testing should be continued despite the fallout hazard which they regard as tolerable considering the advantages continuance of testing has, and others say essentially the opposite? The differences in opinion about the scientific facts are not the real issue. The differences exists, but they do not explain the wide divergence in final conclusions.

Representative HOLIFIELD. There was a challenge put out, Dr. Libby, in this hearing that the Commission should come forward with a clear-cut statement regarding the scientific reasons for the continuance of testing. The geneticists gave reasons which they thought should be considered in the elimination of testing.

Now we are facing, for instance, in Nevada, a series of tests. Do you intend in your statement, or could you at this time give some of the scientific reasons—I am not talking about moral or philosophic reasons—but some of the scientific reasons why we consider, if we do, that testing should be continued?

You might divide that up into two sections, into the military area and also into the area of increasing our knowledge from a protective standpoint.

Dr. LIBBY. I would like to ask you, Mr. Chairman, for permission to prepare a statement on this point for the record, because I think what I would say off the cuff, so to speak, would be less helpful than something I could say after careful consideration. I would be very pleased to do that.

Representative HOLIFIELD. This is the thing I think we ought to be alert to—an affirmative drive for the acceptance of democratic ideas, instead of always being on the defensive side in relation to Russian propaganda in tests.

I think if you will prepare a careful statement, we will have it appended to your presented statement today, so that we may have this subject covered also (see p. 1373).

Dr. LIBBY. I certainly will, Mr. Chairman.

Representative COLE. Mr. Chairman, on that point.

Representative HOLIFIELD. Mr. Cole.

Representative COLE. I am advised that this morning in the press conference at the White House, the President discussed the question of the need for continuing the weapons testing, the subject of the hazards of radioactive fallout; and I would like to ask at the conclusion of the meeting this morning, a synopsis of that press conference be inserted in the record.

Representative HOLIFIELD. The committee will consider accepting that for the record. Up to now we have received testimony from scientists. I do not consider the President of the United States a scientist. He may have the advice of scientists, but unless the committee directs the chairman to break our rules of presenting evidence from nonscientists, we will have to take specific action on that request in executive session.

Representative COLE. Do I construe the chairman's statement, then, as being an objection to my request that the President's press conference be inserted in the record following Dr. Libby's testimony?

Representative HOLIFIELD. It is not an objection; it is a matter of postponement for the committee to decide upon, as we have already agreed we will have no one but scientists to testify during this set of hearings. And we have turned down many people of outstanding character and belief throughout the Nation because of the fact they were not scientists because we want to hold this to a collection of scientific testimony.

Representative COLE. Mr. Chairman, I am going to at this time ask unanimous consent that the President's statement at the press conference this morning on this subject be inserted in the record at the point following Dr. Libby's testimony this morning.

Senator ANDERSON. I would hope that might be done, because the scientists are going to get a supplementary statement, and it may be well to have the official position of the Government on this. I think this might be all right, Mr. Chairman.

Representative HOLIFIELD, There is a unanimous consent request before the committee. If it is the will of the committee, the chairman will entertain the motion of Mr. Cole.

Those in favor of Mr. Cole's motion signify by saying "Aye."

Those against say "No."

(Representative Holifield was the only one voting no.)

Representative HOLIFIELD. Proceed.

Representative COLE. What was the conclusion?

Representative HOLIFIELD. The conclusion of the vote is a vote of 4 to 1 in favor of inserting it in the record. It will be inserted.

The Chair may ask to have other nonscientific statements put into the record at a later date, and if that request is made, we hope that the committee will also agree to the same principle.

Proceed, Dr. Libby.

Representative COLE. Well, Mr. Chairman, I do not want to belabor this point, but to make certain the extent to which the action we have just taken creates a precedent. It has been my understanding that in order for material to be inserted in the hearings requires a unanimous approval of the committee.

Representative HOLIFIELD. There has been no such action on that point by the committee.

Representative COLE. I know. Therefore, my request was a unanimous consent request. If there is a single objection, my request will not be accepted.

Senator ANDERSON. Let me plead with Mr. Cole not to worry about that. I think when we get to considering it we may come to the conclusion that these scientists are asked for an explanation of America's point of view, it is proper for the President of the United States to outline what the point of view is. I think we can solve it without any difficulty. I think we would like to insert it in the record, and I certainly would favor and not oppose it. I do not believe it is a precedent.

Representative HOLIFIELD. I have no personal feeling about the matter. I am merely trying to conform to the rules which were decided upon in executive session, and my vote is still "no" until the rules are changed.

Proceed, Dr. Libby.

Dr. LIBBY. Testing constitutes a small risk—very small compared to ordinary risks which can be tolerated. It is not contended that there is no risk. But all life, and every minute of our day and night, is

measured in terms of risk—40,000 highway deaths each year in this country, accidents in the home, et cetera. We make our choice: How much risk are we willing to take as payment for our pleasures—swimming at the seashore, for example—our comfort, or our material progress? Here our choice seems much clearer. Are we willing to take this very small and rigidly controlled risk, or would we prefer to run the risk of annihilation which might result if we surrendered the weapons which are so essential to our freedom and our actual survival?

Senator ANDERSON. Right there, Dr. Libby, we have much to talk about, I would think. In your words: "Are we willing to take this very small risk?"

Do you regard this a very small risk?

I am referring to the fact that Dr. Russell, who is the principal geneticist at Oak Ridge National Laboratory, was quoted in a Science Service news story as saying that:

> Offspring from a man exposed to such radiation will have their lives shortened on the average of 20 days for each unit of the radiation their father had received.

We figured that out to about 22 years when we attain this maximum period. Then Dr. Russell revised his figures to mean from 5 to 35 days, and at the lowest it would come to about 11 years.

Would you think the shortening of life in offspring to the amount of 11 years was a very small risk?

Dr. LIBBY. Did I understand you to say that the present fallout would shorten our lives 11 years?

Senator ANDERSON. No, you did not.

Dr. LIBBY. I am sorry, sir.

Senator ANDERSON. I quoted Dr. Russell. I thought it would be better if I stayed with the scientists, and I quoted Dr. Russell, who is the principal geneticist at the Oak Ridge National Laboratory, who should be well known to you.

Dr. LIBBY. Yes, sir.

Senator ANDERSON. I referred to a story put out by Science Service which Dr. Russell explained. I think it has to be taken in view of his explanation. I was very happy we had it, because it was most helpful.

The story said: "Offspring from man exposed to such radiation"—and his radiation was neutron radiation from an atomic bomb. I had better read the first paragraph, Dr. Libby.

Dr. LIBBY. I do not know of this.

Senator ANDERSON. I recognize that, and I am sorry. I will be happy to send you the clipping.

> Neutron radiation from atomic bombs can shorten the life of a man's children, Dr. W. L. Russell, the principal geneticist at the Oak Ridge National Laboratory here has found. Offspring from a man exposed to such radiation will have their lives shortened on the average of 20 days for each unit of radiation their father has received.

I do not know how much radiation he is going to have from an atomic bomb. I do not know how much he is going to have from atomic testing. I am not concerned about that, to try to measure it. But here is a scientist—and I am happy to have your assurance that all scientists are pretty well agreed on these essential facts—who believes it will shorten life. And when we discussed it the other day—I hope I do not misquote Dr. Russell; I do not mean to—he ex-

plained the 20 days, and said perhaps a 5 to 35-day figure might be a much better figure.

But do you regard that shortening of life in succeeding generations as a very small risk?

Senator HICKENLOOPER. Mr. Chairman, would the Senator yield to the Senator for a question before he answers the question?

Senator ANDERSON. I recall the old radio show where the Baron said, "The Baron always asks the questions," but I will yield.

Senator HICKENLOOPER. I understand, but I would like to have the Baron answer the full question.

It was my understanding of this testimony it was qualified by saying that a man who got the maximum dose would suffer, and the testimony seems to be replete with the fact that we are in no danger at the present time of coming anywhere near the maximum dose.

Senator ANDERSON. I will be happy to read it. I read then, and I will read now from the news story which does not talk about maximum doses, and says, "Neutron radiation from atomic bombs."

The question then came, If a man should get a maximum dose of, say, 400 roentgens, it would shorten life 10 or 20 years. But I tried to avoid that by pointing out he said it can shorten the life of a man's children.

If Dr. Libby questions that, then we can have Dr. Russell to defend himself at a later time.

Do you believe that neutron radiation from atomic bombs can shorten the life of a man's children?

Dr. LIBBY. I have nothing but respect for Dr. Russell. I do not know the details of his study. This is outside my field.

I would point out to the Senator, though, that there are no neutrons in fallout radiation. I am not saying this to beg the question. I do not know whether Dr. Russell has studied the effects of fallout radiation but neutrons are emitted only instantaneously when the bomb is fired, so you do not get neutrons from fallout.

Senator ANDERSON. Do I understand Dr. Russell was on the Sunshine Project?

Dr. LIBBY. Surely, but I hesitate to comment on Dr. Russell's statement, since I have not seen it, Senator.

Senator ANDERSON. Very well.

Dr. LIBBY. I will be very pleased to answer your question for the record after reading the statement.

Senator ANDERSON. I will skip the words "very small," and come to the next. "Are we willing to take this very rigidly controlled risk."

Dr. LIBBY. Yes.

Senator ANDERSON. How rigidly is this controlled? Do we have any control over Russia as of now?

Dr. LIBBY. Well, I was speaking more about our rigid controls, which are very rigid.

Senator ANDERSON. But we are not the only people that are depositing fission products in the atmosphere, are we, Doctor? Are we?

Dr. LIBBY. We certainly are not.

Senator ANDERSON. All right. If we are not, do we have any control over what Russia does?

Dr. LIBBY. Obviously no.

Senator ANDERSON. Do we have any control over the British?

Dr. LIBBY. Obviously no.

Senator ANDERSON. And there may be a fifth or a sixth or a seventh country coming along. Do we anticipate we will have any control over them?

Dr. LIBBY. I would not comment about them, because I would not know. But the point is this, Senator: We do have control over ourselves, and the debate is whether we stop testing, is it not?

Senator ANDERSON. No, I do not think so, at all.

Dr. LIBBY. Well, part of it.

Senator ANDERSON. I think the debate is whether or not the world tries to bring this under some sort of control.

Dr. LIBBY. Which we are all for.

Senator ANDERSON. I was particularly attracted, if you do not mind my saying so, to the testimony given by Dr. Langham, in which he suggested a level of 10,000 megatons of fission products put into the atmosphere as a possible goal toward which all countries——

Dr. LIBBY. You do not mean 10,000 megatons.

Senator ANDERSON. Ten megatons. Did I not say 10 megatons of fission products? I am sorry. I meant 10 megatons yield equivalent of fission products put in the atmosphere. We have been putting it into the atmosphere at about the rate of 10 megatons a year, and he thinks that is about the maximum, and certain other scientists agree with him, and some of us are interested in trying to bring this situation worldwide under control.

Now, the language of your statement says: "this very small and rigidly controlled risk." And I question the term "rigidly controlled risk," because I realize it is like the term "clean bomb," it makes good reading. But it is not rigidly controlled, is it?

Dr. LIBBY. I had reference to our controls, sir, and they are rigid.

Senator ANDERSON. Well, I go on—"or would we prefer to run the risk of annihilation which might result if we surrendered the weapons which are so essential to our freedom and our actual survival."

Is that a suggestion that if we had control we would have to disarm?

Dr. LIBBY. It is my opinion, Senator, that this testing is an integral part of armament and cessation of testing is a part of disarmament. I think most people agree with that.

Senator ANDERSON. You say you want testing continued?

Dr. LIBBY. I say, and what I did say, Senator, was that testing was an integral part of armament, and so stopping testing is a disarmament move, and I think most people agree with that.

Senator ANDERSON. And I understand you favor continuing testing?

Dr. LIBBY. I am in favor of disarmament under proper controls, very strongly, sir.

Senator ANDERSON. I again come back to your letter to Dr. Schweitzer, in which you say:

> Of course, a workable, safeguarded system of international disarmament is a paramount objective of the United States Government, and one which we must work for and hope and pray will be achieved.

Dr. LIBBY. Yes.

Senator ANDERSON. If you are going to work for it, and hope and pray it would be achieved, an initial step certainly would be to stop testing, a step to disarmament?

Dr. LIBBY. The cessation of testing would be a disarmament move.

Senator ANDERSON. And you are in favor of disarmament?

Dr. LIBBY. In general, yes; under proper controls.

Senator ANDERSON. Very well.

Senator HICKENLOOPER. Mr. Chairman?

Representative HOLIFIELD. Senator Hickenlooper.

Senator HICKENLOOPER. May I ask, Dr. Libby, I take it that what you are saying is that you do not believe it is in any interest of our own future security if we stop testing alone and permit Russia to go on testing, and other nations in the world to go on testing. Is that correct?

Dr. LIBBY. That is exactly correct, Senator.

Senator HICKENLOOPER. And you are thoroughly and completely in agreement with the disarmament which would include the stopping of testing if reliable controls were had, and reliable assurances by other nations that their testing would also be stopped, and the field of weapon experimentation would be eliminated?

Dr. LIBBY. That is right, sir.

Senator HICKENLOOPER. I just wanted to get that straight here.

Representative HOLIFIELD. Proceed, Dr. Libby.

Dr. LIBBY. The cause for real concern is not the deleterious effect of radiation resulting from weapons tests, but rather what would be the effect of the infinitely greater amount of radiation which would result from the massive use of nuclear weapons in warfare. Here we would be dealing with excessive radiation, not to all the people of the world as has been suggested, but quite probably to large numbers of people residing in areas of substantial contamination.

Senator ANDERSON. Right there, Doctor, did the geneticists not seem to indicate yesterday that radiation spread around the world? What about these areas of substantial contamination, Doctor? This is just narrowed down to a city or township near where it happened.

Dr. LIBBY. I would again have to study their statement, and I am not a geneticist. It would seem to me there is no doubt there would be damage to very large numbers of people. Whether there may be remote corners of the world which would escape is a question I think we do not know the answer to, really.

With regard to the people so overexposed——

Chairman DURHAM. Dr. Libby, yesterday Dr. Glass, I believe, testified that in some 2,000 skeletons strontium 90 showed up in all of those in all parts of the world.

Dr. LIBBY. Yes.

Chairman DURHAM. I mean, you do not doubt the fact that it falls out in some quantity around the world, do you?

Dr. LIBBY. No, sir. The question is what the fatalities would be in terms of an atomic war, nuclear war.

Chairman DURHAM. That is why I asked the question, because you are speaking to numbers here now, of course, in at least an act of war.

Dr. LIBBY. Right.

Chairman DURHAM. In your statement, "quite probably to large numbers of people residing in areas of substantial contamination"—what do you mean by large numbers?

Dr. LIBBY. Well, Mr. Durham, what I wanted to bring out is what we call the cigars, that is, the areas of local fallout, which Mr. Holifield spent so much time working on in connection with civilian defense. We have been in these hearings talking about the other side of the coin, the worldwide fallout. In the local fallout pattern which you get that

looks like a cigar, perhaps 7,000 square miles in area as from the March 1954 detonation, and so on.

We must not forget those things. On the one hand we have the subject of this hearing, which is worldwide fallout, and on the other hand we have the blanketing of the country with local fallout pattern. This is the distinction I had reference to.

There is absolutely no doubt that the people in those areas of heavy local fallout would be seriously damaged unless they took care of themselves, and looked to the problem ahead of them. There is absolutely no doubt.

Now the question of worldwide fallout and how serious it will be from the genetic point Senator Anderson brought out, well, I do not think we know; and I think the geneticists would be the first to agree they are not absolutely certain in the magnitude of effect. We know the effects are not good.

Chairman DURHAM. You know the life of strontium 90, though?

Dr. LIBBY. Yes, sir.

Chairman DURHAM. You know, of course, the first contamination is not the end of it.

Dr. LIBBY. Oh, no. There is no doubt there will be serious effects every place in the world if there were a nuclear war.

Representative HOLIFIELD. Proceed.

Dr. LIBBY. With regard to the people so overexposed there would be serious increases in the pathological effect of excess radiation, such as cancer and leukemia. There would also be the genetic effect which would manifest itself in the children and the children's children of such people.

Let us consider the present situation. We can take our normal experience with natural radiation as strictly limiting possible effects from the fallout—since natural radiation is so much larger. It is a queer fact of the present situation that we know far more about fallout dosage than about natural dosage.

The Sunshine project is only beginning measurements on natural radiation, though, as you have seen, its amassed data and its understanding of fallout makes it one of the most impressive scientific investigations ever made. Somehow, even those who have known for years about the wide variation in natural dosages have been more concerned with the study of test fallout than of building materials, homes, public buildings, schoolrooms, et cetera.

(Remarks prepared by Dr. Willard F. Libby, Commissioner, United States Atomic Energy Commission, follow:)

NATURAL OCCURRENCE OF RADIOACTIVITIES AND RADIATION

The radiation dosages that people receive from natural radioactivities and cosmic rays are of importance, for it is only from these dosages that limits on the deleterious effects of radiations received over long periods of time at low rates as in the case of radioactive fallout can be obtained. Studies of vital statistics for populations exposed to varying natural dosage levels offer hope for determination of human susceptibility to radiation effects both somatic and genetic. A brief summary report on this, published some time ago, is offered for the record. It was prepared with the thought of application to the fallout studies in mind: Dosages from Natural Radioactivity and Cosmic Rays, W. F. Libby, Science, 122, 57–58 (1955). (See p. 1459.)

Other lengthier and more complete studies are listed:

1. P. R. J. Burch and F. W. Spiers, Science 120, 719 (1954).
2. P. R. J. Burch, Proc. Phys. Soc. Lond. A67, 421 (1954).

3. Bengt Hultqvist, Kungl. Svenska Vetenskapsakademiens Handlinger. Fjarde Serien. 6, No. 3 (1956).
4. Radiological Data in Japan, Government of Japan, Yoshio Hiyama. U. S. Scientific Committee on the Effects of Atomic Radiation, A/C.82/G/R.70, March 27, 1957.
5. The Toxicity of Skeletal Irradiation at Naturally Occurring Radiation Levels, Robert A. Dudley, April 1, 1957.

I would call attention particularly to the work of Hultqvist on building materials in Sweden, particularly brick and light weight concrete containing alum shale. Measurements in this country indicate that our bricks may not differ appreciably.

Natural dosage in middle of rooms in Swedish homes (mr/yr) (Hultqvist)

Wood	83± 9
Brick	143±22
Concrete (light weight with alum shale)	215±65

We are beginning a survey on natural radiation dosages which should elucidate this and many other points. In the initial results we find that very appreciable variations from spot to spot occur just as would be expected on the basis of the occurrence of thorium, uranium, and potassium in various minerals. Inside a granite church in New York State the dosage was double that outside in the same vicinity.

The close reader of the reports cited above will note a discrepancy in the magnitude of the cosmic ray dosages reported. This has its ultimate origin in a real discrepancy in the physical measurements as between Professors Millikan and Neher in California and Dr. J. Clay and his school in the Netherlands. I have chosen the Millikan-Neher result which seems to have been confirmed in the Japanese report submitted to the U. N. Committee a few weeks ago, whereas others have selected the Clay measurements which give dosages about 70 percent of those of Millikan-Neher. The altitude variations are the same in all studies within the error of measurement.

We have only recently started measuring brick and arranging for widespread natural dosage studies, and most of what we know has come from studies abroad, part of which were through the United Nations Committee mentioned earlier.

For example, in Sweden, Hultqvist has shown that homes of wood give an average dose in the center of their rooms of 80 to 90 milliroentgens per year, while those of brick give 140, and those of lightweight concrete with alum shale, over 200. These numbers are to be compared with present United States fallout doses—and remember that the United States—I know there is an article in the morning paper that said something different—as far as we know is the highest in the world. The present United States dosage is 1 to 5 milliroentgen per year.

Certain areas of India occupied by many thousands of people have natural dose rates—due to the thorium in the sand—several times our average of about 150 milliroentgens per year. An important article on natural dosage appeared in one of our own technical magazines a few days ago. Written by Prof. H. V. Neher, a colleague of Robert A. Millikan's, it presents important early data gathered prior to any nuclear weapons tests. I would like to refer to the principal graph from Professor Neher's paper. It is upon the stand over there.

(The article including the graph referred to follows:)

[Reprinted from Science, v. 125, May 31, 1957, pp. 1088–1089]

Gamma Rays From Local Radioactive Sources

There is considerable interest at the present time concerning the possible effects of manmade radiations on man himself. Because one source of these radiations is of worldwide extent, the interest has also become worldwide. Although considerable literature now exists on the subject of manmade radioactive contamination, on the one hand, and on the biological effects of radiation, on the other, the actual importance of the first as far as the second is concerned has often been obscure. It is thought desirable at this time to present some independent experimental data that will allow individuals to reach their own conclusions.

As early as 1928, R. A. Millikan became interested in the gamma rays emitted by local radioactive materials in the soil and rock at various localities in order to determine the effect of these radiations on the cosmic-ray measurements in which he was primarily interested. These measurements extended from California into the Rocky Mountain area and on up to Churchill, Manitoba.[1] They probably represent a unique series of measurements, since they were made before manmade contamination became widespread.

Anionization chamber measures directly the quantity of interests as far as the biological effects of gamma rays are concerned, and this is the instrument here employed. One of the instruments Millikan made and calibrated is still in good condition after 26 years and is very convenient to use. A recent redetermination of the absolute value of the calibration[2] agrees with Millikan's value to 0.3 percent. In this survey, Millikan's instrument has been used for some of the measurements, and a more modern ionization chamber[3] for others. The two give essentially the same answer. Both were used unshielded in the measurements reported here.

---

[1] R. A. Millikan, Phys. Rev. 37, 242 (1931).
[2] A. B. Johnston, thesis, California Institute of Technology (1956).
[3] H. V. Neher, Rev. Sci. Instr. 24, 99 (1953).

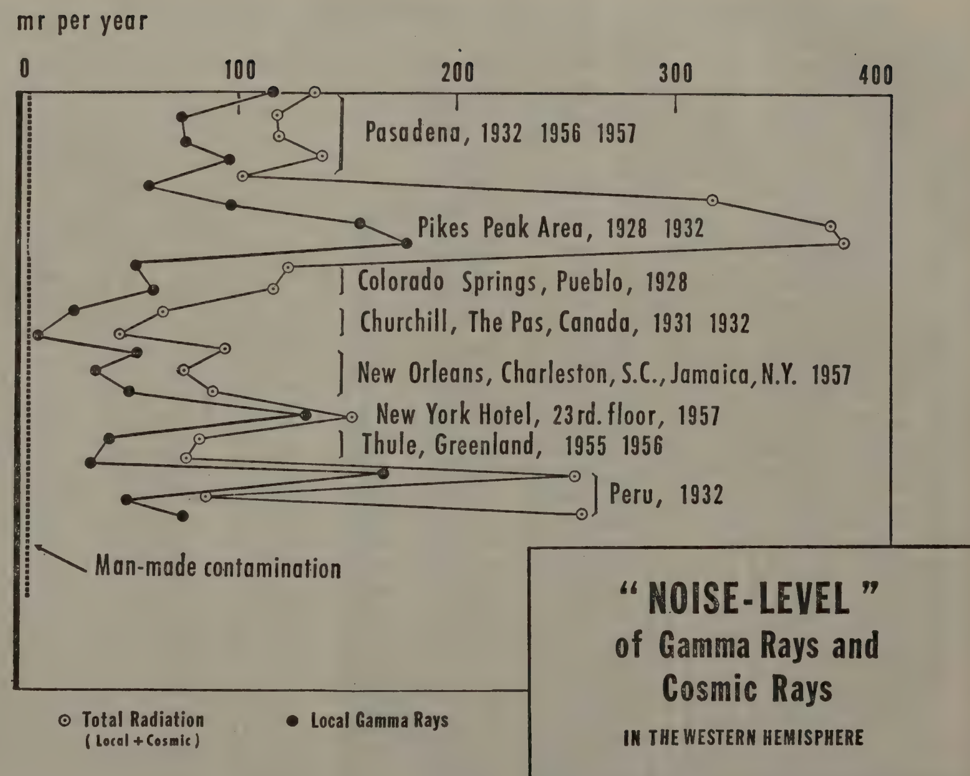


FIGURE 1.—"Noise level" of gamma rays and cosmic rays in the Western Hemisphere, Abscissas roughly increase with increase of distance from Pasadena. The amount of manmade contamination is taken from the National Academy of Sciences report, Biological Effects of Atomic Radiation (see footnote 7). As is stated in that report, "* * * United States residents have, on the average, been receiving from fallout over the past 5 years a dose which, if weapons testing were continued at the same rate, is estimated to produce a total 30-year dose of about 0.1 roentgen" (an average of 3 mr. per year).

In figure 1, most of the values taken during the years have been entered. The ordinates are in milliroentgens (mr.) per year. To convert into ion pairs per cubic centimeter, per second in 1 atmosphere of air, divide the ordinate by 15. The various stations are plotted as abscices with the same increment from one to the other. Roughly, the stations get farther from Pasadena with increase in abscience. The chief reason for plotting in this manner was to bring out the variability of radioactivity from one station and region to another.

Measurements were made of the total radiation at a given station; then the known contribution from cosmic rays[4] was subtracted to get the effect of the gamma rays from local radiation only.

In the Rocky Mountain region, the local radiation is high, presumably because of the granite, which is known to contain something like 4 g. of uranium and 15 g. of thorium per ton.[5] In Peru, the radioactivity of the coastal plain is much the same as that of the Mississippi region near New Orleans. The local radiation at an elevation of 15,000 feet in southern Peru is only slightly higher than that of the soils of the coastal plain. Most of the houses of Arequipa are built of a light rock called tuva which is of volcanic origin. This rock is 3 or 4 times as radioactive as the soil near Lima.

There is considerable variability of local radiation in some cases over small distances. According to Millikan,[6] the gamma rays on the Laurentian Shield near Churchill, Manitoba, give 0.8 ion cm^{-3} sec^{-1} atm^{-1} of air, or 12 mr yr^{-1}, while nearby the intensity on the glacial sand is 35 mr yr^{-1}. It may be of interest that the radioactivity on the icecap near Thule, Greenland, in August 1956 was less than 2 percent of cosmic rays.

A wooden building forms some shielding from local gamma rays. In my own house, the gamma rays on the first floor give 60 mr yr^{-1}, while in the back yard the intensity is 95 mr yr^{-1}. The rather high value of 130 mr yr^{-1} on the 23d floor of a major hotel in New York is presumably owing to the material from which the building is constructed.

The root mean square "noise" level of the total radiation given in figure 1 is about 160 mr yr^{-1}. To find the effect on the population, the local radiation must be weighted according to the population. This has not ben done. Perhaps it is fortunate that most of the population of the country lives where the radiations due to cosmic rays and local radiations are relatively low.

The dashed line near the bottom of figure 1 is taken from the summary reports on the Biological Effects of Atomic Radiation of the National Academy of Sciences.[7] Even though there is some error in the determination of this value, as well as considerable variation of fallout over the country, it is quite evident that manmade contamination is still small compared with the changes in radiation from one part of the country to another.

The data presented here are for gamma rays only, since the walls of the ionization chamber are too thick for beta rays to penetrate, either from naturally occurring or artificially produced radioactive materials.

H. V. NEHER,
Norman Bridge Laboratory of Physics,
California Institute of Technology, Pasadena.

The upper curve is the total dosage in milliroentgens per year. The first four points are various locations in Pasadena, Calif., in the years 1932, 1956, 1957. The lower curve is just that part of the dosage which is due to gamma rays. That is due to the radioactivity in the rocks. The difference between the two curves is the cosmic rays.

And as he goes to various places over the country, and over the world, you will notice that the very widest variation in these natural dosage rates.

In many cases the variation is due to the cosmic rays; in other cases it is due to change in the composition of the rocks.

---

[4] I. S. Bowen, R. A. Millikan, H. V. Naker, Phys. Rev. 46, 641 (1934).
[5] H. Paul, Ed., Nuclear Geology (Wiley, New York, 1954).
[6] R. A. Millikan, unpublished results.
[7] Summary Report of the Committee on the Genetic Effects of Atomic Radiation, in Biological Effects of Atomic Radiation (National Academy of Sciences, Washington, D. C., 1956).

This presents the total dosage due to cosmic radiation and radiation from the ground and general surroundings but does not include the 21 milliroentgens per year from our own bodies, so to calculate the total exposure under the various conditions described, we should add this in.

Of course, houses shield from the ground radiation although not appreciably from cosmic rays—so that they are a diminishing effect. Professor Neher remarks that in his own house in Pasadena, the gamma rays on the first floor give 60 milliroentgens per year while in the backyard the intensity is 95 milliroentgens per year—just to give you a feeling for the effect of being inside as compared to outside.

Well, these dosages are all very large as compared to the 1 to 5 milliroentgens per year which we receive here in the United States from fallout, and we must put our experience from fallout in this context.

Now certainly our experience with these much larger normal doses shows us that the effects due to fallout radiation are relatively small.

The fallout facts are pretty well known and agreed. On the other hand, small as the effects are, they most likely do exist and the critical and essential question is, Are they tolerable?

This question, however, calls for a political or sociological—rather than a scientific—judgment, and I think it is important that this distinction be made.

Chairman DURHAM. Mr. Chairman?

Representative HOLIFIELD. Chairman Durham.

Chairman DURHAM. In the previous years' study up until 1945, there was no such thing as strontium 90?

Dr. LIBBY. There certainly was not, Mr. Durham, at all. There was no strontium 90. These data are important because they are before any contamination from weapons at all, you see. This radiation is still there, but it may be a little more difficult to be sure about it.

I should point out on that graph—you see that line that is right down near zero, that is the test fallout line.

Chairman DURHAM. Of course, that was made by a higher authority than man, the natural radiation.

Dr. LIBBY. That is right.

Chairman DURHAM. And since then, of course, we are adding to it by putting strontium 90 into the air streams.

Representative HOLIFIELD. Dr. Libby——

Dr. LIBBY. I want to make sure, Mr. Holifield, this graph is clear to everyone. This top line is the total. If you take a counter and measure these various places, that is what is there. This part [indicating] of it is from the rocks. The difference is the cosmic rays, and this [indicating] is the way it varies.

If you go to Pikes Peak, Colorado Springs, and so on, this is the way it varies.

Here is the test fallout down here, this dotted line. The smallness does not mean that test fallout is not important, that it is not a risk and a hazard, but it does mean that the test fallout is a relatively small hazard.

The first point is, since we have been living on this earth so long, we have some practical experience where commonsense can take hold, and we can say there is some limit just by the fact that we have not noticed these big things in our normal experience—there is some limit

to the bad effects from test fallout, and that is the only point I wanted to make.

Representative HOLIFIELD. Dr. Libby, to make the picture complete, is it not true that there is a very important difference in the bomb fallout which is not contained in either the cosmic rays or in the natural radiation, and that is the factor of strontium 90?

Dr. LIBBY. Yes, sir.

Representative HOLIFIELD. Which gets into the bones of people?

Dr. LIBBY. Yes, sir.

Representative HOLIFIELD. Where cosmic rays and natural radiation do not have that particular residual effect in the bones of people. So we have got another factor and, therefore , your analogy there must take into consideration also this new factor of strontium 90 that we must consider.

Dr. LIBBY. There is a great deal of truth in what you have said, Mr. Chairman. To comment on your statement requires a small technical remark.

Biological——

Representative HOLIFIELD. Why did not you make that statement first, Dr. Libby——

Dr. LIBBY. I wanted to——

Representative HOLIFIELD (continuing). Instead of making a statement which was susceptible again to a benign interpretation?

Dr. LIBBY. I want to tell you that the statement I have just finished making is true.

Representative HOLIFIELD. I know it is. Most of your statements are true, sir. But the point is this: That when once you have made those true statements, they are susceptible of misinterpretation, and if you follow up with a complete truth, it might give a more balanced effect in the layman's mind.

Dr. LIBBY. Let me do that. I think it will only take a couple of minutes. I will try to make it short.

A milliroentgen is an amount of energy absorbed by a unit amount of tissue. Now, depending on the kind of radiation the biological effect varies. The biological effect from 1 milliroentgen will be different for 1 kind of radiation as compared to another. This is called the relative biological effectiveness.

While it is true that we have never had strontium 90 before, we have had radiations which have essentially the same relative biological effectiveness, and have always had them, and those are the cosmic rays.

The cosmic rays, milliroentgen per milliroentgen, are within a small error the same as strontium 90, milliroentgen per milliroentgen. This is not true, Mr. Holifield, for gamma rays from rocks, but for the cosmic rays which make up a good fraction of the total natural exposure, they are just as damaging to the bone milliroentgen per milliroentgen as strontium 90.

Representative HOLIFIELD. Do they reside in the bone and of themselves make emissions, such as strontium 90?

Dr. LIBBY. No, no; but that is not necessary, you see. It is not the presence of strontium 90 in the bone that is damaging. You ordinarily have ordinary strontium in the bone. The bad thing about strontium 90 in your bones is that it emits radiation.

Representative HOLIFIELD. That is right.

Dr. LIBBY. So the cosmic rays in passing through the bone, as they do through all of your body, deposit in the bones energy which is in excess of the energy deposited by the present burden of strontium 90.

What I am telling you, Mr. Chairman, is that you can compare these two kinds of milliroentgens. You can say that if the milliroentgens from cosmic rays are large as compared to those from strontium 90, then the bone cancers and leukemias, and all the other things that may be caused by strontium 90 should follow even more from the cosmic rays. That is what I am saying.

In other words, the gamma rays you cannot compare, but the cosmic rays you can.

So when I went to the chart and said that about test fallout, I was telling you the truth in the sense that a good part of that total radiation is cosmic rays.

Representative COLE. Mr. Chairman?

Representative HOLIFIELD. Mr. Cole.

Representative COLE. I was going to ask Dr. Libby to explain that point on the chart which indicates that the local gamma radiation is almost identical with the radiation resulting from the fallout from the tests.

Dr. LIBBY. It is described in Dr. Neher's article. It is a local condition, but he does describe it.

I am sorry. He does not give a detailed description. I can give you a general answer, Mr. Cole.

Representative COLE. That will be good enough.

Dr. LIBBY. I am sorry but these effects are complicated, the way thorium and uranium occur. You see that low point is due to the fact that the uranium and thorium are missing there. Uranium and thorium occur every place—in all granite rock, for example. That is why you seldom can get away from it, unless you are living on a sand, or a formation which has through the accidents of geology had the uranium and thorium washed out of it. So in order to tell you why that point is low I would have to consider the local geology very carefully.

Representative COLE. Do you know the locality of the point?

Dr. LIBBY. It is on the Laurentian Shield in Canada. But the exact locality is not specified. However, the reference to the article is given, and we could look it up for you.

Representative COLE. Thank you.

Representative HOLIFIELD. Are there further questions, now?

Senator ANDERSON. Doctor, I want to come back to this question of clean weapons which has concerned me quite a bit, and to the article in the U. S. News where they asked you that question.

One question was:

> Dr. Libby, at one time the Commission announced they were developing a clean bomb, and then many scientists argued with you and criticized and even ridiculed the statement of the Commission. How do you account for that?

Your answer was:

> Well, in most instances, they were not privileged to know what it was.

Then the question:

> They did not know what they were talking about, possibly?

You said:

> I did not quite say that, but they were not apparently mamiliar with all of the details of it.

In your speech you referred to a clean weapon. Do you still feel we have a clean weapon?

Dr. LIBBY. Well, sir, I think we all know what the facts are. It is a question of how you describe the situation. We certainly have succeeded in cleaning them up to a great degree.

If you call cleaner "clean," maybe you are telling a small fib. But we certainly have a cleaner weapon, as you know.

Now, the question of the adjective which is applied to that, all I can say is that perhaps we have used the wrong adjective, and perhaps we should say "cleaner."

Senator ANDERSON. It is very important, because headlines all around proclaimed this clean bomb.

You said:

> We have taken the problems of radioactive fallout into consideration—

and this is on fallout, and this is why I bring it up—

> in our weapons development program and have developed and tested clean weapons, that is, weapons in which the amount of radioactive fallout per megaton of explosive is very greatly reduced.

Dr. LIBBY. Yes.

Senator ANDERSON. If I took a little of this coffee I have here, and poured it in that glass, it is dirty (pouring coffee into glass). Then, if I pour a little bit over there (pouring coffee in another glass), that is clean because the amount of dirt, to use your language, is very greatly reduced.

Would that be a fair statement? You cannot get a better example of what happened.

Dr. LIBBY. I think we know what the situation is. We have very greatly reduced the fallout per megaton of yield, and that is imporant.

Senator ANDERSON. I am only trying to say that a lot of our people have been reassured on this question by this so-called clean weapon, and it is still a dirty weapon. We have also learned how to make our weapons even dirtier than they originally were.

I think it is dangerous to use terms like "uniformity" when we mean "nonuniformity," and "clean" when we still mean "dirty."

Dr. LIBBY. I think our responsibility, Senator, is to tell the truth as best we can. I think that we must stay as close to it as we can. We are all clumsy in the use of language, and I am one of the worst. We may have chosen not exactly the right adjective, but I think we have explained it enough now so people know what it means.

Senator ANDERSON. I hope so, because I have seen many headlines talking about clean weapons. And knowing you and respecting you as I do, I know you would be the last one to want to mislead. I just hope no one will think there is no fallout damage when it comes to the use of weapons in atomic warfare, because it will come.

That is all.

Representative HOLIFIELD. Are there any further questions?

Representative COLE. Mr. Chairman, before releasing Dr. Libby, I would like to express my own compliment to him for the very fine presentation he has given to the committee this morning.

So far as time will allow, they have been complete answers, unequivocal so far as possible, sound, and dispassionate, and I think he has rendered a great help to the committee, and his work on the problems of fallout hazards has been most beneficial.

Representative HOLIFIELD. Dr. Libby, we will excuse you at this time, but we hope that you are able to be present for the general conference this afternoon at the conclusion of the testimony.

Dr. LIBBY. Mr. Chairman, the Commission meets at a quarter to three, and I will try my best to get back here at 4 o'clock. I understand it is at 4 you are having your meeting.

Representative HOLIFIELD. We are reconvening at 2 o'clock in this room, and we will continue with our witnesses until they are through, and then we will have the conference. I am not sure just when it will be.

Dr. LIBBY. I will come back as soon as I can, Mr. Chairman.

Representative HOLIFIELD. Thank you, Dr. Libby.

The committee will be in recess until 2 o'clock.

(NOTE.—Several articles and statements by Dr. Libby have been printed as appendix 1, p. 1459. An article entitled "Radioactivity in Man and His Environment," by Dr. F. W. Spiers, appears in appendix 2, p. 1671.)

(A paper entitled "Radiation Dose to Man From Natural Sources," follows:)

RADIATION DOSE TO MAN FROM NATURAL SOURCES

Robert A. Dudley [1] and Robley D. Evans,[2] Department of Physics, Massachusetts Institute of Technology, Cambridge 39, Mass.

I. INTRODUCTION

Man has always been exposed to high-energy radiation from cosmic rays and naturally occurring radioactive substances in his body and in his environment. Twentieth century society has brought about an increase in his exposure to such types of radiation, from such sources as commercial radium preparations, medical X-ray machines, and fallout. This increase has focused attention on the deleterious medical effects of high-energy radiation. Since the effects of this additional radiation on man are very difficult to determine when the effects are small, several methods of predicting them have been attempted. One of these has been to compare the magnitude of the artificial radiation with the magnitude of the natural radiation to which man has always been exposed.

The types of radiation associated with cosmic rays and natural radioisotopes include α rays, β rays, mesons, and γ rays. The first two are important only when their source is deposited within the body; the second two are important only for sources external to the body. These natural radiations are very similar in their biological effects to the β and γ rays of artificial sources, although quantitative differences, considered later, do exist between α rays and the others.

The organs of the body receive different exposures, depending chiefly on the degree to which various radioisotopes concentrate in them through either physical or chemical processes. Thus the skeleton is exposed to radioisotopes chemically similar to calcium. The intestines are exposed to all radioisotopes which enter the stomach, even if the element is insoluble and is not absorbed into the bloodstream. The lungs are exposed to radioactive dust retained from inhaled air. All organs are exposed to cosmic rays and γ rays from the environment.

---

[1] Oberlin College, 1943–45; University of Pennsylvania, 1945–46 (B. A., physics); Massachusetts Institute of Technology, 1946–51 (Ph. D., physics); Fulbright student, British Medical Research Council, radiotherapeutic research unit, Hammersmith Hospital, London (1953–55); lieutenant USNR, attached to Division of Biology and Medicine, United States Atomic Energy Commission (1953–55); consultant to Egyptian Atomic Energy Commission, Cairo, Egypt (1955–56); research associate, department of physics, Massachusetts Institute of Technology (1956–). Professional research experience: Use of radioactivity as a tool in various applications, particularly biological research; evaluation of Sr^{90} fallout problem while attached to United States Atomic Energy Commission; study of radiation dose to humans from natural and artificial sources. (Submitted by witness.)

[2] B. S. in physics, 1928, California Institute of Technology, Pasadena, Calif.; M. S. in physics, 1929, California Institute of Technology, Pasadena, Calif.; Ph. D. in physics, 1932, California Institute of Technology, Pasadena, Calif.; National Research Council fellow in physics, 1932–34, University of California, Berkeley, Calif. 1934 to present, physics department, Massachusetts Institute of Technology, Cambridge, Mass.; Assistant professor, 1934–38; associate professor, 1938–45; professor, 1945 to present. (Submitted by witness.)

On account of the many types of radiation and the many processes for deposition of radioisotopes in different organs, the general subject of the natural irradiation of man has many facets. The present study is confined to the natural radiation dose received by the skeleton and the gonads, these being the two organs of greatest interest in the case of fallout.

Many previous investigations can be drawn upon for pertinent data. In particular, reference may be made to the review articles of Libby,[1] Lowder and Solon,[2] Hultqvist,[3] and Spiers.[4] Although past studies by no means close the subject, reliable quantitative estimates of dose from the several important sources may now be given.

II. NATURAL IRRADIATION OF THE SKELETON

A. *Radiation originating within the body*

There are four natural isotopes which with their descendants contribute skeletal doses of comparable importance: potassium 40 (K–40), radium 226 (Ra–226), mesothorium (MsTh, or Ra–228), and radium D (RaD or Pb–210). In addition to these, there are many others (e. g., uranium, thorium, carbon 14) which are of negligible importance by comparison. The four important isotopes will be considered in turn.

K–40 emits β rays of 0.6 Mev. average energy and at a rate equivalent to 8.4×10^{-4} microcuries per gram of element K. Its γ radiation is of negligible importance compared with its β radiation in the case of K deposited in the skeleton. The abundance of K in adult bone is about 0.09 percent on a wet-weight basis.[5] From these data the dose rate to the skeleton may be calculated as about 8 mrad/year, where by definition 1 mrad$=10^{-3}$ rad$=0.1$ erg absorbed per gram of bone. Since the element potassium has a definite physiological role, it is controlled by the homeostatic processes of the body and is therefore expected to be found in the skeletons of all individuals in nearly equal concentration.

TABLE 1

Investigator	Description of samples			Equivalent RA content in bone ash	
	Number	Age	Origin	Mean	Standard deviation
		Years		*Microcuries per gram*	*Microcuries per gram*
Hursh and Gates [1] [2]	21	33–85	Mostly New York State	5×10^{-14}	2×10^{-14}
	5	Stillborn	New York State	3.6	.7
Palmer and Queen [3]	50	32–93	United States, mostly Pacific Northwest.	1.6	1.0
Muth et al.[4]	14	27–74	Germany	13	7
Stehney and Lucas [5]	8	15–29	Chicago	1.2	1.2
	8	15–18	Lockport, Ill	12	5
	30	Adult	Joliet, Ill	6	(6)

[1] Hursh and Gates, Nucleonics 7, No. 1, 46 (1950).
[2] Hursh, Brit. J. Radiol., Suppl. No. 7, 45 (1957).
[3] Palmer and Queen, HW-31242 (July 6, 1956).
[4] Muth et al., Brit. J. Radiol., Suppl. No. 7, 54 (1957).
[5] Stehney and Lucas, International Conference on the Peaceful Uses of Atomic Energy, A/Conf. 8/1/852, U. S. A. (June 23, 1955).
[6] Variation depending on length of residence in Joliet.

Na–226, the familiar Ra isotope, decays to the noble gas Rn by alpha emission. Rn itself is radioactive, decaying with a 3.8-day half life by alpha emission. Subsequent descendants emit alpha, beta, and gamma rays, of which only the alpha rays are important dose contributors. Po–210, the last of the three Rn descendants which emit alpha rays, is of minor importance in this sequence, as its formation is long delayed by the intervening RaD isotope.

---

[1] Libby, Science 122, 57 (1955).
[2] Lowder and Solon, NYO–4710 (July 1956).
[3] Hultqvist, Kungl. Svenska Vetenskapsakademiens Handlingar. Fjarde Serien 6, No. 3 (1956).
[4] Spiers, Brit. J. Radiol. 29, 409 (1956).
[5] Tipton et al., ORNL CF 56–3–60 (March 12, 1956), and personal communication (March 1957).

Ra is found in the skeleton as an unessential trace element by virtue of its chemical similarity to Ca. Its most important source is solid food, although in some areas its abundance in drinking water exceeds its abundance in food. Many measurements of Ra concentration in human bone are available. The important published measurements are summarized in table 1. From these results a mean of about 4×10^{-14} microcuries per gram in chosen (the higher values being given less weight since they represent, in part, a study of a selected high region). Variations about this mean by a factor of 3 to 5 are found, depending on local conditions. The extremes of skeletal Ra concentration, as set by geography and other variables, cannot be accurately specified; however, it is unlikely that large population groups exist at concentrations differing from this mean by more than a factor of 10.

The magnitude of the radiation dose delivered to the skeleton by Ra and its descendants is influenced by the migration of the noble gas Rn out of the bone and its subsequent exhalation from the body. No data are available on percentage Rn retention from natural skeletal burdens of Ra, but at artificially elevated burdens the retention is about 25 to 50 percent. Presumably the natural situation is in this respect very similar. Taking the lower figure, one computes an average energy of about 11 Mev. dissipated within the body for each Ra disintegration. The mean skeletal dose rate is then found to be about 3 mrad/year attributable to Ra–226 (and its descendants produced within the body).

MsTh, a Ra isotope occurring in the radioactive series associated with Th, is itself a beta emitter of 6.7-year half life. However, it gives rise to several descendants which emit alpha as well as beta and gamma rays. While some of these descendants have such half lives or chemical properties that a possibility exists for migration out of the bone, it seems probable from animal experiments that, in fact, little such migration occurs.

MsTh has not yet been sought with sufficient thoroughness to provide good experimental data on its natural concentration in the skeleton. However, since it is an isotope of Ra–226, the dose attributable to it and its descendants may be reasonably accurately estimated from our knowledge of the Ra–226 situation. The activity of MsTh and Ra–226 in soil and water is on the average nearly equal. Therefore the MsTh/Ra ratio activity in newly formed bone will be nearly unity, while in older bone the ratio will be lower as a result of the shorter MsTh half life. Assuming nearly all the MsTh decay products remain in the bone, the energy ultimately delivered to the bone subsequent to each MsTh disintegration is about 30 Mev. Therefore MsTh gives a dose almost 3 times greater than an equal activity of Ra–226. In children the MsTh dose is expected to be about twice the Ra dose while in adults it will fall below the Ra dose. Averaged over the individual's life, MsTh (with its descendants) is expected to produce approximately the same skeletal dose as Ra–226, or about 3 mrad/year. This value is probably subject to approximately the same variations with geography as is Ra.

RaD is a member of the Ra series, decaying by beta emission with a 22-year half life. Its daughter, RaE, is a 5-day half life beta emitter, and its granddaughter, RaF (Po–210), is a 140-day half life alpha emitter. Only rough estimates of the RaDEF dose to normal bone can as yet be given. It is clear that RaD descended from Ra in the bone will accumulate with time, but in a quantity which is unimportant compared with the other members of the Ra series. In addition to the RaD generated from Ra in the bone, however, there is now good reason to expect a greater quantity deposited from food and water. These expectations are based on experimental measurements of stable Pb in the skeleton, from which calculations can be made for RaD. Preliminary unpublished measurements have been made of RaDEF in bones at the University of Rochester.[6] These measurements and calculations indicate that the dose from RaDEF is of the same order of magnitude as the dose from Ra, and it is tentatively assigned an average value of 3 mrad/year. More precise measurements will soon be available from several laboratories.

It is known that in the production of biological damage, the effectiveness of alpha rays, per unit of energy dissipated, is different from the effectiveness of beta rays, gamma rays, and mesons. This relative biological effectiveness (RBE) of alpha rays relative to the other radiations varies with the effect under consideration. In evaluating the toxicity of low dose rates to the skeleton, probably

[6] Black, Personal Communication (May 1957).

the most pertinent effect to consider is the production of bone tumors. Mouse experiments at Argonne National Laboratory on bone tumor production by beta rays (Sr–90) and alpha rays (Ra–226) suggest that the alpha ray RBE is about 4.[7] This number is here adopted, with recognition that at lower dose levels in humans the correct value could differ either way by as much as a factor of 2 to 4. A better estimate will be available from the dog experiments now in progress at several laboratories in the United States.

B. Radiation originating external to the body

Cosmic radiation at the earth's surface is composed mainly of mesons, gamma rays, and energetic electrons produced from the primary cosmic rays high in the atmosphere. The magnitude of the cosmic ray dose increases both with increasing altitude and with increasing geomagnetic latitude. The determination of this magnitude has been the subject of several series of observations. While the results are not all in close agreement, the discrepancy is not serious with respect to the accuracy here required. Taking the dose rate at sea level and high geomagnetic latitudes as given by Burch's review,[8] and the dependence of dose on altitude and latitude as given by A. H. Compton,[9] the skeletal dose rates of table 2 may be calculated. Using other data, slightly higher values are obtained.

TABLE 2

Altitude	Geomagnetic latitude		
	0°	30°	50°
0 feet	23 mrad per year	24 mrad per year	26 mrad per year.
6,600 feet	37 mrad per year	39 mrad per year	46 mrad per year.
14,300 feet	76 mrad per year	83 mrad per year	100 mrad per year.

There are three important sets of naturally occurring γ-emitting isotopes in man's environment: the uranium 238 (U–238) series, the thorium 232 (Th–232) series, and K–40. In the case of a man standing on flat ground, the contribution of skeletal dose from each series may be quite accurately calculated when its concentration in the ground is known. Furthermore, direct measurements have been made of the combined external γ dose and cosmic ray dose at several points.[3 4 10]

The concentration of the radioactive elements in ground is quite variable, tending to be highest in igneous rock (e. g., granite), and lowest in sedimentary rock (e. g., limestone) which is the more common at the earth's surface. Typical geochemical data [11] yield the skeletal dose rates attributable to each radioactive series as shown in table 3. (In this table, a factor of 0.7 has been applied to surface dose rate to give skeletal dose rate, thereby allowing approximately for shielding of one part of the body by another.) Considerable variation is found for the concentration of each series in such rock, but to some extent these variations in individual series cancel out. Some rocks (e. g., dunite) yield less than 1 mrad/year, while others (e. g., U or Th ores) give several thousand mrad/year. An example of the latter situation is found in the monazite sand region of south India, where about 100,000 people live in areas where the skeletal dose rate from Th and U in the ground is several hundred mrad/year.[12] However, these extreme conditions are surprisingly rare, and it may be assumed that only a very small fraction of the world's population is exposed from the ground to dose rates more than a factor of 2 outside the range shown in table 3.

---

[3] Hultqvist, Kungl. Svenska Vetenskapsakademiens Handlingar. Fjarde Serien 6, No. 3 (1956).
[4] Spiers, Brit. J. Radiol. 29, 409 (1956).
[7] Marinelli, Personal Communication (December 1956).
[8] Burch, Proc. Phys. Soc. London A67, 421 (1954).
[9] Compton, Phys. Rev. 43, 387 (1933).
[10] Evans and Raitt, Phys. Rev. 48, 171 (1935).
[11] Rankama and Sahama, Geochemistry (University of Chicago Press, Chicago, Ill., 1950).
[12] Bharatwal and Vaze, Report to United Nations environmental radiation committee numbered A/AC.82/G/R.33 (October 19, 1956).

TABLE 3.—*Skeletal dose rate in radioactive series*

Rock	U	Th	KK	Total
Igneous	17 mrad per year	22 mrad per year	27 mrad per year	66 mrad per year.
Sandstone	5 mrad per year	12 mrad per year	11 mrad per year	28 mrad per year.
Shale	5 mrad per year	20 mrad per year	28 mrad per year	53 mrad per year.
Limestone	6 mrad per year	21 mrad per year	3 mrad per year	30 mrad per year.

In buildings the dose rate from local gamma emitters will be somewhat altered from its value outdoors. In framehouses a slight reduction is expected, but by only a small factor since thin wood walls do not provide effective shielding. In houses built of brick or stone, shielding will be appreciable. However, the radioactivity in these materials (if of the same composition as the ground) will more than compensate for the shielding, and from purely geometrical considerations the dose rate inside will be approximately double that outside.

C. Summary of natural skeletal irradiation

The several components of skeletal dose rate previously listed are collected together in table 4. The unit now used is the mrem/year, where

$$\text{mrems} = \text{mrads} \times \text{R. B. E.}$$

Dose in mrems is thus equal to dose in mrads, except for α radiation where the R. B. E. is taken as 4. The values in table 4 are rough averages, and considerable variations in individual components are found for different population groups. However, these variations partially cancel, such that only a very small fraction of the world's population is expected to differ from the mean total exposure by as much as a factor of 3.

For comparative purposes, the following artificial dose rates may be considered: A fallout Sr–90 body burden of one one-thousandths the industrial maximum permissible concentration (roughly the present average in newly formed bone [13]) corresponds to about 3 mrem per year. The external gamma radiation from fallout is of the order of 5 mrem/year at present.[13] Medical X-ray dose rates to the skeleton vary from zero in backward areas to roughly the same magnitude as total natural dose rate in medically advanced societies.[14]

TABLE 4

Source of radiation:	Skeletal dose rate (*mrem/year*)
K^{40} (internal)	8
Ra–226 (internal)	12
MsTh (internal)	12
RaD (internal)	12
Cosmic rays (external)	30
Local gamma rays (external)	60
Total	134

III. NATURAL IRRADIATION OF THE GONADS

The only important internally deposited natural radioisotope in the gonad is believed to be K–40. Since it is an essential element having a definite physiological function, potassium in the gonads will be found at closely similar concentrations in all individuals. The mean value is approximately 0.2 percent,[4] giving a dose rate of about 18 mrads (or mrem) per year.

The gonads are exposed to the same external irradiation from cosmic rays and environmental gamma rays as is the skeleton, and the dose rates will be essentially identical.

The total mean natural dose rate to the gonads is therefore about 110 mrem/year, with variations from 1 region to another as associated with variations in cosmic rays and environmental gamma rays. These natural dose rates may be compared with the dose rates from fallout gamma radiation and medical X-rays as given above for the skeleton.

[13] Libby, W. F., speech to American Physical Society, Washington, D. C. (April 26, 1957).
[14] National Academy of Sciences, The Biological Effects of Atomic Radiation, a report to the public, Washington, D. C. (1956).

(Whereupon, at 1:10 p. m., the committee recessed, to reconvene at 2 p. m., of the same day.)

AFTERNOON SESSION

Representative HOLIFIELD. The committee will come to order.

Our first witness this afternoon is Dr. Ralph Lapp, a well-known physicist and author of books on radiation and related subjects. Dr. Lapp, we are pleased to have you before us this afternoon, and you may proceed.

STATEMENT OF DR. RALPH E. LAPP, WASHINGTON, D. C.[1]

Dr. LAPP. Thank you. Mr. Holifield, I received your invitation to testify before this committee while I was in Japan. I cut short my trip in order to attend the hearings. May I say that I appreciate very much this opportunity to appear here. I would like to add that I am very gratified that your investigations to date have thrown so much light on the problem of radioactive fallout.

I regret that the rate of accumulation of data in my files is so great that I have trouble in digesting it. I hope that during the next month I will have the time to analyze properly the amount of data that has been presented and that I hope I may be able to give you a later statement. I have been out of Washington for 3 months, and I have had trouble keeping up with things. Nonetheless, I am delighted to be here and testify before your committee.

Representative HOLIFIELD. We have permitted other witnesses to present additional data to substantiate their statements, and that same permission will be granted to you.

Dr. LAPP. I believe that these hearings will stand as a landmark in the history of our knowledge about this relatively new phenomenon.

Now I would like to add a comment about Dr. Libby. Appearing as I do after Dr. Libby, I would like to comment on his contributions to fallout. Dr. Libby has not only stimulated extensive research in fallout investigations such as Project Sunshine, but he has also taken the initiative in publication of his findings. I feel very strongly that he deserves a great deal of credit for his work on fallout. Were it not for Dr. Libby, we might well be confronted with a considerably smaller body of knowledge about fallout than we have today.

Mr. Chairman, I do not intend to read this entire document because it is too long, and I will skip over certain sections, with your permission, I might just mention that my interest in fallout extends over quite a period of years. I do not claim to be a great prophet with regard to the ultimate effects of radioactive fallout, since I think any examination of my publications about the Bikini shot of 1946 will

---

[1] Nuclear Science Service, Washington 7, D. C. Physics, Buffalo, N. Y., August 24, 1917. Strong Foundation fellow, Chicago, bachelor of science, 1940, doctor of philosophy (physics), 1946. Instructor, Chicago, 1940–41; research associate metallurgical laboratory, Manhattan project, 1943–44; division director, 1944; assistant to laboratory director, 1945; assistant laboratory director, 1946; Argonne National Laboratory, 1946; science adviser, General Staff Research and Development Division, U. S. War Department, 1946–47; Deputy Executive Director, Atomic Energy Commission, Joint Research and Development Board, 1947–48; Executive Director, 1948–49; head, Nuclear Physics Branch, Office, National Research, Department of the Navy, 1949–52; Director, Nuclear Science Service, 1953–; industrial consultant and lecturer, 1953–; assistant, Chicago, 1940–41. A. A., physical sociologist, cosmic radiation; meson; bursts; showers; mass spectroscopy; radiological safety; nuclear radiation physics. (From American Men of Science, 1955.)

show that I was not terribly impressed with the phenomena of radioactive fallout at that time. I had to learn the hard way.

I might say that my interest in radioactive fallout was really stimulated by the 1954 Bravo test at Bikini. This was the test which resulted in radioactive contamination of the *Lucky Dragon No. 5*, a Japanese tuna trawler.

Based upon results which were made available largely by the Japanese, I began the publication of a series of articles on fallout at the invitation of the editor of the Bulletin of Atomic Scientists, and with your permission I will put these into the record, if you so desire.

Representative HOLIFIELD. They will be received and filed with the committee.

(The series referred to follows:)

November 1954, Civil Defense Faces New Peril
February 1955, Radioactive Fallout
June 1955, Radioactive Fallout III
November 1955, Global Fallout
September 1956, The "Humanitarian" H-Bomb
October 1956, Strontium Limits in Peace and War

Dr. LAPP. I am dividing my testimony into four parts: general remarks, local fallout, remote fallout, and I am so bold as to add some constructive proposals.

Under "general remarks," I would say I believe public confusion about fallout will continue to increase unless scientists can provide a quantitative or semiquantitative evaluation of the various hazards associated with fallout. Precision is probably not possible due to the nature of the hazards, and we may have to be content with numbers which vary by a factor of 2, 3, or even 10. This committee, I believe, has already performed a valuable service in narrowing the range of estimates made by individual witnesses.

Regarding disagreement among the scientists, I think that the public may well conclude that if scientists cannot agree upon hazard, then all is confusion. It would be nice if the scientists could all agree upon a quantitative estimate of the hazard, which could then be given out to the public. I maintain two unusual circumstances have combined to produce the current confusion on radioactive fallout.

First, the urgency of our times has focused attention upon problems for which science did not have textbook answers. Available knowledge was inadequate and research such as Project Sunshine had to be initiated to provide answers.

Second, the ordinary process by which scientists argue out their answers was interdicted by the complexity of the problem and also by secrecy. Scientists outside the Atomic Energy Commission have full-time jobs, as do those inside, and could scarcely be expected to tunnel into the complexities of the problem in a few leisure hours.

With regard to the responsibility of the Atomic Energy Commission, I think that if we consider these factors, the Atomic Energy Commission has responsibility for providing the outside world with the facts about fallout as promptly as they become available.

Representative COLE. Mr. Chairman, I wonder if Dr. Lapp would mind interruption while his statement proceeds?

Dr. LAPP. I would welcome interruption.

Representative COLE. We all agree with your statement that the Commission should make revelation of its information about fallout as promptly as it becomes available. Do you have any feeling that the Commission has failed in that responsibility, except insofar as fallout material may have a direct hearing on weapons information?

Dr. LAPP. Mr. Cole, I believe that your qualification there makes it difficult for me to answer the question. When you say "except insofar as it may have bearing upon weapons information," you, of course, bring into play the difficulty. May I answer it as specifically as I can with regard to the question of radioactive contamination of large areas.

I believe that the Atomic Energy Commission could have made available to the public far earlier than it did its report of February 15, 1955.

This is the report on the effects of high yield nuclear explosions. It took almost a year from the time that explosion occurred until this, I would call it nonquantitative report, appeared. It has taken additional time until the weapons-effects handbook—I believe called The Effects of Nuclear Weapons—will appear, presumably this month.

It is my personal opinion, Mr. Cole, that could have been done more expeditiously.

Representative COLE. But if it had been published earlier than 10 months after the test, would you not agree that any report would have been based on conjecture rather than on facts?

Dr. LAPP. No, Mr. Cole, I think the facts about this particular incident were fairly well known as of May 1954, and I believe if one looks at the record of the investigation of this committee—I am referring to the investigation of March 24, 1955, called The Atomic Energy Commission-Federal Civil Defense Administration Relationship—in this report published by your committee, the Atomic Energy Commission has stated that it had a summary report of the radioactive effects of the Castle series of tests available, I believe, in May of 1954. So based upon that testimony by the Atomic Energy Commission, I would say that they had the facts.

Representative COLE. The Commission, as you know—either the President or the Chairman of the Commission—did shortly after that test give a generalized report on the results of the test.

Dr. LAPP. Yes. It occurred, I believe, in my memory serves me correctly, on March 29 or 30, 1954.

Representative COLE. Some time in March.

Dr. LAPP. Yes. I personally do not feel that report was an adequate or quantitative evaluation of the hazard.

Representative COLE. Of course, it was not. It was not intended to be. But it was a general report to the public as quickly as general facts could be made known.

Dr. LAPP. It was a general report.

Representative COLE. As to the detailed lessons based on facts as they were observed might necessarily involve a period of time. It would seem that a period of 11 months, considering the importance of the subject under study, was not inordinate.

Dr. LAPP. I think we may disagree how long would have been required for this. I might say, Mr. Cole, if I am permitted a moment to look into my own notes here—that so far as I personally am concerned,

based upon data made available from the Japanese, I was able to publish in November 1954 in what I would call semiquantitative form an analysis of the hazard, and again in 1955 I was able to publish in the Bulletin of Atomic Scientists in February—I am sure I have it here, but it just escapes me—let me quote from memory.

Senator ANDERSON. I think I have a full file of the bulletins.

Dr. LAPP. It is the February 1955 issue.

Senator ANDERSON. That is probably the one I do not have.

Dr. LAPP. In this issue I believe I presented a quantitative estimate of the hazard which was more detailed in numbers than was the Atomic Energy Commission's release of February 1955. The situation, I realize, is difficult if one wishes to make a precise statement. But I do not think it was a precise statement that was needed. I believe something which would indicate the general magnitude of the hazard which civil defense faced would have been important.

By the way, I will come to this point in connection with later testimony.

May I continue?

Representative HOLIFIELD. You may proceed.

Dr. LAPP. I believe, then, that scientists, technicians, and officials of the Atomic Energy Commission must present reasoned and careful estimates of the hazards based upon factual knowledge. Reckless or nonsubstantiated statements do a disservice to the Atomic Energy Commission and to the Nation.

Representative VAN ZANDT. Dr. Lapp, you say reckless or nonsubstantiated statements do a disservice to the AEC and to the Nation. You make this statement under the title "The Responsibility of the Atomic Energy Commission." Are you charging that the AEC spokesmen made reckless or nonsubstantiated statements?

Dr. LAPP. I believe that the examples I have given here, particularly when we have a man like Dr. Richard Doan coming to Tokyo only last month stating that the bomb test did not have the slightest possible effect—I would be happy to place the entire quotation in the record—upon humans, that is nonsubstantiated. I believe it constitutes what may be regarded in the light of the great world importance of atomic energy as amounting to a reckless statement.

I have not charged that Dr. Libby made a reckless statement here when he said, "However, as far as immediate or somatic damage to health is concerned, the fallout dosage rate as of January 1 of this year in the United States could be increased 15,000 times without hazard."

Representative VAN ZANDT. Dr. Lapp, do you have all available information in this field?

Dr. LAPP. I have such information as is available to the public.

Representative VAN ZANDT. But you do not have the reservoir of information available that Dr. Libby has, do you?

Dr. LAPP. If I am to believe from Dr. Libby's testimony this morning that it was all unclassified, I would say that I must have.

Senator ANDERSON. It is either classified or you have it?

Dr. LAPP. Yes. I am sorry you are asking me something that is difficult for me because I simply do not know whether or not there exist things which are buried in secrecy.

Representative VAN ZANDT. I am not worried about classified information. I regard Dr. Libby as a member of the Atomic Energy Commission and therefore having at his fingertips all possible informa-

tion with access to AEC files. Therefore, when he makes a statement he must certainly make it based on the most comprehensive information available.

Now, I am asking this question: Do you have comparable information?

Dr. LAPP. No, sir.

Representative VAN ZANDT. By making the critical statement regarding the AEC spokesmen, in effect you compare yourself with Dr. Libby from the standpoint of availability of information. Therefore, it amounts to this, does it not, that it is simply your own opinion?

Dr. LAPP. It is my opinion based upon the facts which Dr. Libby has presented.

Representative VAN ZANDT. That is all, Mr. Chairman.

Dr. LAPP. I might say I can only go on what Dr. Libby has stated plus my knowledge of science and physics.

Representative PRICE. Mr. Chairman, may I ask Dr. Lapp a question?

Representative HOLIFIELD. Yes.

Representative PRICE. Is it merely your personal opinion, or is it your opinion based not only on information made available to you, but on consultation and association with other scientists?

Dr. LAPP. I have consulted with many scientists. I may not say in the specific preparation of this document because I was working until the early hours this morning doing this. I have in the past number of years consulted with many scientists of considerable note. I do not pretend to speak for scientists. But I might say this, Mr. Price; I have been encouraged by scientists.

Representative PRICE. You do not feel that you are alone in these opinions?

Dr. LAPP. I am alone right now, sir.

Representative PRICE. I do not think you have been alone. I think others have testified pretty close to the testimony that you started out to give here. I do not think you are alone.

Dr. LAPP. I would like to make this statement, Mr. Price. I have come here today to try to present such observations as I can to be of help to this committee. I do not want to be put in the position of challenging every statement that Dr. Libby makes.

Representative PRICE. I did not assume that you were challenging everything. The point I wanted to draw out was whether or not these were just strong personal convictions on your part, or whether or not you had arrived at your conclusions not only through the examination of the material furnished to you but through your personal studies and association with other leading scientists not only in this country, but in other parts of the world.

Dr. LAPP. I have recently consulted with scientists in Japan. I might say that one of the useful things that I accomplished in discussing these problems with the Japanese was that I think I explained some of the strontium measurements to the Japanese and tried to reconcile Dr. Libby's data with their own. I think it was useful because many of their figures were quite high as compared with Dr. Libby's. Although I am not yet sure that they believed everything I have said, I have been in correspondence with them since and I have tried to bring about some agreement in this field.

Again, I state that I personally owe a great debt of gratitude to Dr. Libby as I think the country does in providing us with these very valuable data.

Representative PRICE. I think we all agree with you on that. I think you were interrupted before you were permitted to give the examples that you were going to give on statements you referred to as perhaps "reckless" statements.

Dr. LAPP. I think we must interpret this and perhaps qualify it somewhat. That is, we are dealing now with statements made to the public. This is an area in which I have had some experience, namely, in the translation from the world of megacurie and the megaton into the world of ordinary understanding. I think Congressman and Senators know how difficult this translation can be. Dr. Libby's speeches have not always been masterpieces of simplicity for the press to understand. I have in many cases attempted to translate these speeches so that they might be more useful.

Senator ANDERSON. Doctor, now that you have had a chance to refresh your memory as to your fallout data, do you find you reported in the February 1955 issue of the Bulletin of the Atomic Scientists?

Dr. LAPP. Thank you. It was in the February issue of 1955 that I presented a quantitative estimate of the radiation exposure. I believe this curve showing cumulative dosage from fallout agrees with the data which are now presented some 3 years later in The Effects of Nuclear Weapons. I am going to testify about this.

Representative HOLIFIELD. You made that chart without access to classified information?

Dr. LAPP. The basis of making this chart was, if I may be permitted—this is a complex technical matter—to give you a complete explanation.

Representative HOLIFIELD. Make it simple.

Dr. LAPP. I have here the reports of the Japanese scientists. These are called Research in Effects and Influences of Nuclear Bomb Test Explosions. I want to point out that this has been published only recently, but it contains many of the scientific papers of 1954 upon which I based my deductions.

These reports might just be illustrative to show how much other countries are publishing. These reports are the research in the effects and influences of nuclear bomb test explosions in two volumes compiled by the Committee on Compilation of the Report on Research and Effects of Radioactivity, published in Japan for the Society for the Promotion of Science. There are about 1,800 pages of data in these reports.

Representative HOLIFIELD. The chart that you gave in February 1955 has stood up, then, against challenge, has it?

Dr. LAPP. I believe so. I have discussed this with Dr. Eugene Wigner, who is a scientist of outstanding note. We were much concerned about the tail of the curve. This tail of the curve is quite important because it is upon the tail of the curve that the long term persistence of radioactivity will depend. At the time I talked with him, Dr. Wigner had his doubts about how this tail may be extrapolated.

My own feeling is the recent data of Dr. Dunning, contributed to this committee, support this curve.

Representative COLE. Dr. Lapp, you will concede that your report of January 1954—or is it February?

Dr. LAPP. I think it came out in February.

Representative COLE. That was the same month that the Commission's report came out.

Dr. LAPP. Yes, Mr. Cole.

Representative COLE. But your report was based on studies of exposure to the Japanese fishermen.

Dr. LAPP. Yes.

Representative COLE. Whereas, that was only a part of the persons who were exposed to radioactivity. Is that not correct?

Dr. LAPP. That is correct, Mr. Cole.

Representative COLE. There were other people, the Marshallese, who were also exposed.

Dr. LAPP. That is true, but the data which I am concerned with is not data on people, but rather the actual number of curies of radioactivity deposited on the surface of the Lucky Dragon.

I might say, by the way——

Senator ANDERSON. Your report came out in the February issue which reaches the subscribers ahead of the first of the month. The Commission report came out in March and followed by a considerable period of time the publication of the data that you have there, did it not?

Dr. LAPP. I believe there was a difference in time of about 8 days. I prepared this, by the way, in December of 1954. I again received a tremendous amount of help from Dr. Libby's first speech of December 1954. It was very useful to me. I really feel that Dr. Libby has contributed immensely to this whole phenomenon of fallout.

Representative COLE. Mr. Chairman, before Dr. Lapp leaves this question of the responsibility of the Commission to make factual publication of its information, Dr. Lapp has indicated three examples which in his opinion represent a failure to fulfill that responsibility of frank and honest statements. He has characterized them as reckless and nonsubstantiated by the facts.

Dr. LAPP. Not all the statements.

Representative COLE. I notice you made an exception with respect to Dr. Libby's statement that it was not reckless, which leaves the conclusion that his statement nevertheless was unsubstantiated by the facts.

Dr. LAPP. With respect to that, Mr. Cole, I would wonder whether or not in the light of the testimony that has been received by this committee that Dr. Libby would still wish to subscribe to the statement that the fallout dosage rate as of January 1, 1955, in the United States could be increased 15,000 times without hazard.

Representative COLE. I am not seeking to argue with you. I am simply trying to establish clarity in the record.

Another question is this. These three examples which you have enumerated, do they constitute the only instances which have come to your attention where employees or representatives of the Commission have made statements on this subject which you feel were perhaps reckless to some degree, or not substantiated by the facts?

Dr. LAPP. No, Mr. Cole. I would have to go through my files. I believe there are many more.

Representative COLE. But would you agree that these are the most glaring instances of recklessness on the part of such people?

Dr. LAPP. Until I really combed my files and did a thorough job I can't answer the question.

Representative COLE. It would seem from the fact that these impressed themselves on your memory, and that you have cited them would indicate that they were the most extreme instances.

Dr. LAPP. No, I would not believe that. I think I cite them because they were readily at hand when I prepared this testimony.

Representative COLE. On this point, Mr. Chairman, it would seem to me that in fairness to these individuals whose responsibility has been questioned in this regard—Dr. Eisenbud and Dr. Doan—they should be given an opportunity to give whatever explanation they may have by way of defense against the charge of having issued a reckless and unsubstantiated statement.

Representative HOLIFIELD. I will respond to that. They will be given this opportunity if they care to.

Representative VAN ZANDT. Dr. Lapp, I am not doubting your ability as a physicist nor am I challenging your right of opinion, but for the first time during the course of these hearings we have the integrity of a Government agency challenged. That is what it amounts to. It has changed the complete tone of these hearings as far as I am concerned. We are not sitting here as an investigatory committee. We are sitting here for the purpose of trying to find the answer to this radiation problem. I think you would make a great contribution to these hearings if you would delete from your statement that charge you have made against the AEC and certain physicists employed by them.

Senator ANDERSON. I thought we were going to hear their replies to the charge. I think if Dr. Eisenbud will come in and prove as they laid down these criteria of proof the other day, that fallout to date from all tests would have to be multiplied by a million to produce visible deleterious effects, except in areas close to the explosion, that it would be very interesting.

Dr. Libby says he does not know what will happen when this comes out of the stratosphere. If Dr. Eisenbud would give us a short statement proving that it will have to be increased a million times, I think that would be very interesting. It would contradict a great deal of testimony from the Atomic Energy Commission itself.

Representative COLE. Mr. Chairman, I would point out by way of important emphasis that this statement of Dr. Eisenbud was made on March 20, 1955.

Dr. LAPP. Yes.

Representative COLE. So whatever response he may have to make by way of justification or explanation will have to be predicated as of that day, and not as of today.

Dr. LAPP. I would like perhaps to amplify this one bit, that is, that when I talk of the use of the word "reckless," there, I am speaking in terms of how the noneducated public will interpret statements made to them. That was my only purpose. I am not challenging the integrity of those people.

Representative COLE. Dr. Lapp, would you assert that the Commission's spokesmen in the scientific field were the only scientists who have made reckless and unsubstantiated statements on this problem?

Dr. LAPP. I would not. Reckless statements have been made by scientists who are not in the Atomic Energy Commission.

Representative COLE. Would you also say unsubstantiated statements were made by scientists who are not in the Atomic Energy Commission employ?

Dr. LAPP. I would readily agree to that.

Senator HICKENLOOPER. Mr. Chairman, may I ask Dr. Lapp, have you examined the full statements of Dr. Eisenbud and Dr. Libby and Dr. Richard Doan that you referred to here?

Dr. LAPP. Yes, sir.

Senator HICKENLOOPER. So it is based on that examination of the full statement that you quote out of context here?

Dr. LAPP. Yes, sir.

Senator HICKENLOOPER. And draw your conclusions that these add up to what you call reckless statements?

Dr. LAPP. Yes.

Senator HICKENLOOPER. I think perhaps one might argue that there is a degree of recklessness in dogmatic statements even by yourself in drawing these conclusions, not having been closely associated with the investigation of the data involved, and I rather question the advisability of the use of the word "reckless" in this rather difficult and quite ramified and uncertain field.

Senator ANDERSON. Dr. Lapp, would you not agree with me that there is a possibility that Dr. Eisenbud might not have been correctly quoted in the newspapers? I say that because Dr. Eisenbud gave us what I regarded as extremely fine testimony in his appearance before the committee. I thought it was scientifically based and carefully put together, and you have spoken very highly of Dr. Libby. I share your high regard for him, and I would hope that you might express to me how you feel about Dr. Eisenbud. I think he is a very capable and fine man. I hope that would sort of find some response in your system also.

Dr. LAPP. I was much impressed with the statement he made before this committee. The statement he made was in the Sunday News for New York of March 20, 1955. I believe that this illustrates one of the problems, Mr. Anderson, of the scientists and the press. He is really responsible for being careful in issuing statements to the press. I am not in any away attempting to attack the integrity of any people. I merely point out the impact which this will have upon the public.

So far as world interest in fallout is concerned—I will try to read this quickly—the committee may be interested in my observation that fallout has become an acute weapon of propaganda. For example, I found the Japanese scientists are actively studying the radioactivity of their tea, because of the assertion from the Chinese mainland that Japanese tea is radioactive. Apparently fallout does not occur in China. Some people in Japan are so keenly aware of fallout——

Representative HOLIFIELD. I think you should clarify that facetious remark, because in the print your manner of delivery might create the impression that you have said that it does not.

Dr. LAPP. I am sorry. It seems to me that here is an example of how radioactive fallout can be used as a weapon of propaganda in which a country which might stand to gain from its sale of a product accuses another country of having radioactive tea, and forces that other

country to engage in fairly laborious research project to find out how radioactive their tea is.

I visited this laboratory where tea was analyzed and saw the vast quantities of tea tested for radioactivity. I cite it merely as an example of how this can be used.

Representative COLE. What were the results of those tests?

Dr. LAPP. The tests are still underway. I believe the results will be given out within the year. I did not find any unusual radioactivity from the contact I had with Dr. Shiokawa. Some people in Japan are so keenly aware of the fallout that they actually take showers after being out in the rain.

There was a great public outcry against the British Christmas Island tests, but there was no great demonstration against the Soviet tests. It seems to me that this is a great victory for psychological warfare experts when they can induce selective sensitivity to fallout.

Representative COLE. Dr. Lapp, since you were there in Japan at the time of these tests or immediately prior to the tests, can you account for the fact that there was such a striking demonstration against the Christmas Island tests and yet there was no equal or even slightly proportional demonstration against the Russian tests?

Dr. LAPP. I might point out in all fairness that so far as the Japanese Government was concerned, they did protest the Soviet test, too, but as far as the public demonstration was concerned, I know of no such demonstration against the Soviet test. I think, however, one can give a partial explanation for this. This is in the fact that the Japanese people were immensely affected by the radioactive fallout in the fishing areas of the Pacific. After the accident of March 1, 1954, the Japanese Government went to considerable effort and expense to monitor the fish supply, which is a great source of protein for the Japanese. I think therefore they associated with the Christmas Island tests an effect upon their food supply.

Representative COLE. If that is so, how do you account for an equally demonstrative representation against the tests here in Nevada?

Dr. LAPP. I was not aware that they had such an outcry.

Representative COLE. You must have been aware that some Japanese people stormed the gates of the American Embassy in Tokyo in protest against the Nevada tests.

Dr. LAPP. I am sorry. I was traveling about Japan a good deal and perhaps I missed this one, Mr. Cole.

Representative COLE. This only occurred within the past month.

Dr. LAPP. I see. I did not know about this.

Representative COLE. Yes.

Dr. LAPP. I cite my reason as one factor.

Representative COLE. It is understandable that the Japanese people should be unusually sensitive to the hazard of radioactive fallout. That is very understandable. But it is difficult for me to understand why they should distinguish between the hazard of the British or American fallout without apparent protest to the hazard from Russian test fallout. Do you have any explanation for it?

Dr. LAPP. I could not profess to be extremely competent in this regard. I believe, however, there is a Communist Party at work in Japan. I believe that they use radioactive fallout as a political weapon. In fact, I believe that in the case of the survivors of the

Lucky Dragon, that their families were visited by representatives of the Communist Party who promised them money by way of being helpful. To my knowledge they never showed up with the money, but they promised them money.

Representative COLE. Were you not told when you were in Japan, as I was, that the newspaper reports of the Russian tests indicated to the Japanese people that the Japanese scientists had detected some unusual turbulance in the atmosphere? It was characterized as such, with no direct reporting that this was radioactive material. It was simply tagged as an atmospheric disturbance, whereas the press described the British and American tests as radioactive contamination of the air.

Dr. LAPP. I think they are probably allergic to American bomb fallout. This is rather interesting, in view of the fact that the data given to me by the Japanese show that some 70 percent of the total gross activity of the fission debris falling upon Japan is of Soviet origin, 20 percent from the Pacific tests and 10 percent from the Nevada tests.

Representative COLE. That was given to you by Japanese scientists?

Dr. LAPP. Yes.

Representative COLE. Have you ever seen that conclusion published in the Japanese newspapers?

Dr. LAPP. I gave a number of interviews when I was in Japan, and I pointed this out, but I unfortunately do not read Japanese, and do not know whether they reported it. It may have been. I stated the fact that there is greater fallout on Japan from Russian tests than from United States tests or United Kingdom. I am unaware that it was published. It may have been.

Representative COLE. This is a rather remote and roundabout route by which to provide these conclusions for the information of the Japanese people. I can hope that the Japanese reporters who are present here today may report to their respective newspapers published in Japan the conclusion which was given to you while in Japan, a conclusion by Japanese scientists—that their tests of the contamination of the atmosphere over Japan was caused 70 percent from the Russian tests and the balance from American and British tests.

Dr. LAPP. I think, Mr. Cole, I hope that these facts are reported in the Japanese press. I have reason to believe they will be. I think it is entirely reasonable that the fallout upon Japan should be predominantly from Russian tests because of the greater tropospheric fallout which Dr. Libby explained this morning. The greater tropospheric fallout from these tests will occur from the Russian tests. Because of the fact that they are in the air mass trajectory from the Soviet test region, this fallout will occur upon Japan sooner than upon other parts of the world. Because of the freshness of the fallout, there will be a greater radioactivity.

Representative COLE. That being a scientifically provable fact, then, is it not appropriate that the Japanese should be far greater concerned over Russian tests and the hazard of Russian tests than the tests by the United States and United Kingdom?

Dr. LAPP. If I were a Japanese I certainly would be.

Representative HOLIFIELD. It is not strange, as far as I know that the Atomic Energy Commission has never revealed this very important fact, and that the first knowledge I have of it is as of today? If

they have revealed it, I am unaware of it. We are being subjected to propaganda as you have stated by the Communist Party seizing on the part of this technical information which is to their advantage, and using it in Japan. Why is not this a piece of scientific fact that could be used on our part and let the world know that 70 percent of the fallout on Japan comes from Russian origin, if that is a true fact.

Dr. LAPP. Mr. Chairman, I am trying to strain my memory to remember this, but I believe that the National Academy of Sciences report contains data on relative fallout. I am trying to remember if it was on Japan. I think that it does contain data on this, but it is probably buried in the scientific literature.

Representative HOLIFIELD. I have read the report fully, and if it is in there, I have either forgotten it or I could not understand it, the way it was stated.

Dr. LAPP. I do not think it was in the summary or general report, but was in the greater compilation of the appendix, the physical measurements.

Representative HOLIFIELD. But a report of the American Academy of Sciences is not a publicized report of the Atomic Energy Commission.

Dr. LAPP. I might say this. I cannot remember any Atomic Energy Commissioner making a point of this in his speeches. I read most of the speeches quite carefully. I do not remember this being made.

Representative COLE. You do not know whether this information has been made available or known to the Commission, do you?

Dr. LAPP. I could not state positively.

Representative COLE. Of course, if the Commission had wanted to or happened to it could have found the same information which was given to you. I do not question that. But there is no evidence, or is there, that the Commission had this information?

Dr. LAPP. I am sorry, I am not competent to discuss that.

Representative COLE. Mr. Holifield referred to this conclusion of yours, or this information which was given to you as having an element of propaganda. I am sure he did not intend to use that word in the strictest sense, because this information in my opinion is not propaganda. This is a statement of the scientific facts as resulting from the examination of competent Japanese scientists.

Representative HOLIFIELD. I will accept the gentleman's amendment, if he will allow me to substitute the word "publicity" in the place of "propaganda."

Representative COLE. Yes. It should be publicized generally, and widespread, and that is why I am talking about it as much as I am in the hope that the reporters who are present will make certain that it is publicized fully and in all of the newspapers in Japan.

Representative HOLIFIELD. I had assumed that Project Sunshine was all over Japan, too, and we knew about this.

Representative VAN ZANDT. Mr. Chairman, I would like to ask Dr. Lapp another question.

Dr. Lapp, the Japanese scientific documents you have at your elbow, have you gone through them as yet?

Dr. LAPP. I have gone through a great many articles, yes. Some of them did not interest me too much, so I skipped over them. I have gone through a great many.

Representative VAN ZANDT. Dr. Lapp, do you think all the information contained in those Japanese documents is original or has somebody borrowed the information from the American scientific family?

Dr. LAPP. I would say that much of it is original and of course scientists borrow wherever they get the information. That is the nature of science. It is an international community. There are a great many references in each paper to the United States reports, and to other nations as well. I think that the United States reports predominate.

Representative VAN ZANDT. In other words, the volumes contain much United States data?

Dr. LAPP. No, they reference United States data. For example, if we have a radiochemical technique for detecting a particular radio element, then they would use the technique and reference this. I want to point out, however, that the Japanese scientists, despite the fact that they are not many in number as compared with other nations, have quite a history of excellence. I had the great pleasure of talking with Professor Kimura only two Sundays, ago, and he was very kind to show me some of his original work, and I know it was of very high quality.

Representative VAN ZANDT. Dr. Lapp, have you had made available to you a copy of a Russian scientific document entitled, "Preliminary Data on the Effects of Atomic Bomb Explosions on the Concentration of Artificial Radioactivity in the Lower Atmosphere and Soil"?

Dr. LAPP. I have not. (See p. 1209.)

Representative HOLIFIELD. It was just called to my attention by the chairman that one of our outstanding scientists in California is an American Japanese.

Dr. LAPP. The Japanese scientists are particularly competent in the field of theoretical physics. One of the greatest theoretical physicists in the world is Professor Yukawa, famous for his discovery of the meson.

It is inherent in the very nature of the biological research into the effects of radioactivity upon humans that a high degree of accuracy is not attainable, especially on human experience basis. As Dr. Langham, of the Los Alamos Laboratory, has testified, human experience with retention of radium 226 is the basis for setting upon a maximum permissible concentration for radiostrontium. Yet our actual experience is confined to a small sample of acutely exposed individuals and a small sample of less acutely exposed people.

Actually, our concern should focus not upon acute effects in man, which are highly unlikely from peacetime bomb testing, but rather with the chronic, debilitating, long-term effects from irradiation of humans. We must be conscious of the need to appraise long-delayed effects, say 50 years after entry of radio elements in the body. Here our knowledge is quite limited.

SECTION F. RADIATION LIMITS FOR A GLOBAL POPULATION

I would like to stress the fact that consideration of safe limits for irradiation of the world's population is essentially a new problem. Prior to the awareness of global fallout, the International Commission on Radiological Protection made its recommendations for those who would be exposed to radiation in pursuit of their occupation.

Such groups initially were numbered in the hundreds and then in the thousands as atomic energy came of age. Individuals within such groups were healthy adults exposed to known and restricted hazards; they were subject to administrative controls and medical supervision.

In setting up limits for a total population, we must take into account the varying radiosensitivity of individuals, the complete spectrum of age, the persistence of the hazards, the lack of medical control, the varying degrees of health of people, and the variety of their diet. Yet it was not until last year that the Atomic Energy Commission introduced the difference between an occupational MPC and a global MPC into its releases on fallout.

May I explain that briefly? Dr. Libby, in his speeches referred always to the MPC. I am not accusing Dr. Libby of deliberately trying to mislead anybody, but, from the standpoint of the ordinary layman reading these things, there was no distinction between a maximum permissible concentration for occupational workers and for the world population. I think this is one of the things that is necessary when you are putting information out to the public—that you must distinguish between these different units.

Representative COLE. Why is that, Doctor? What is the difference whether the individual absorbs or is exposed to the maximum MPC in an atomic plant or as an employee or whether he is exposed to it outside. Why do you feel that a distinction should be made?

Dr. LAPP. I think the distinction should be made on the basis, first of all, of the difference in radiosensitivity of the individuals. You are dealing with the total population now when you are dealing with the global risk. You are dealing with people who have no medical supervision. You are dealing with people who are of different ages. I believe that the International Commission on Radiological Protection recommends a factor of 10 and others believe it should be more—a factor of safety—when dealing with the total population than when you are dealing with the small population, the occupationally exposed population. These are the recommendations of the international body on the subject.

In view of the nature of our knowledge and the totality of the sample with which we are dealing, I would urge a big factor of safety in setting limits to bomb testing. It would be tragic to find someday that we had erred in setting the limits.

Perhaps I might explain, in response to Mr. Cole's question, the probabilities involved here. Supposing that the probability of damage were only one in a thousand and you only had 10 people working in the laboratory; this would be a small risk for 10 people. But if you had one in a thousand and apply the same statistics to a total population of 2 billion people, obviously, you have a very different situation. It is part of the philosophy that goes into establishing such figures.

Representative COLE. When you say, in your opinion, that we should set a big factor of safety, don't you mean that we should set a very low factor of safety in order to be on the safe side?

Dr. LAPP. Perhaps my language is not clear there.

Representative COLE. You mean the same thing.

Dr. LAPP. Yes.

Representative COLE. We should be ultracautious in fixing a factor of safety.

Dr. LAPP. Yes.

The Soviet nuclear tests: I felt that the committee might be interested in learning some miscellaneous data I picked up in Japan. I learned that the Japanese scientists collected sufficiently active samples from the Russian tests to perform radiochemistry upon the bomb debris. I was informed that five Soviet tests produced a fallout on Japan from which scientists measured and identified the presence of radioisotope uranium 237. The Soviet explosions were characterized by such fallout that they were judged to be in the megaton range. These estimates are subject to considerable uncertainty, but one authority told me that he estimated that at least 2 bomb yields were in the range of 10 megatons.

Two Soviet nuclear tests were observed to originate in the Arctic region, whereas the remaining tests took place in a region estimated to be the Ozero Balkash, which is southeast of the new coal area of Karaganda. The air-mass trajectories from central Siberia frequently sweep across the islands of Japan, especially Hokkaido. They also produce tropospheric fallout over the United States, as well. Here in Washington you could swipe a Kleenex over a car top and cause a Geiger counter to respond. Perhaps that is a qualitative statement, because the normal counting rate of the counter is 20 counts a minute, and the counter may go up several times over that. But it was readily detectable by even such a simple analysis as this.

Representative VAN ZANDT. Dr. Lapp, that last statement you make; is that a fact or is it just hearsay?

Dr. LAPP. I was told this by one of my scientific colleagues, since I was not here at the time. I have no reason to doubt it.

Representative VAN ZANDT. Then we are getting it secondhand.

Dr. LAPP. Secondhand, but I can give you the source, if you wish. I have seen the measurements done myself. The presence of uranium 237 in the Soviet fallouts proves that the Soviets have achieved a compound fission-fusion or so-called multiple-stage weapon. According to my information, this was first accomplished in September 1954.

Representative COLE. First accomplished where?

Dr. LAPP. In the Soviet Union. The next statement has already been made.

I would like now to talk about the problem of local fallout. I am not going through all of section A, because what I am doing there is trying to explain a term which I coined some time ago in order to eliminate confusion in popular translation with the megacurie. The term I used is the "eternity roentgen" per square mile. It turns out that this particular compound unit is extremely easy to use in estimating the roentgen exposure of people in a bomb area. I will not go through all of this.

Could I have the chart of Dr. Shafer put up? (See p. 119.)

Last week Dr. Shafer testified before this committee about an attack of 2,500 megatons of bomb yield upon the United States, and specified that they were surface burst dirty weapons. By dirty it is meant that the ratio of fission to fusion is high. I assumed that 2,000 tons of fission products deposited locally. This I calculate as 12 billion roentgens per square mile.

Representative COLE. You have characterized these as dirty weapons, Dr. Lapp. How would you evaluate the content or force of a weapon which might be called a clean weapon? As you know, there

has been a considerable discussion of the maining of the words dirty versus clean, clean versus cleaner. You have used the words "a dirty weapon." Are there degrees of dirtiness?

Dr. Lapp. Yes. I would estimate the degree of dirtiness as the ratio of the fission to the fusion release in the bomb.

Representative Cole. So that in your opinion it is possible to fabricate a weapon which is clean.

Dr. Lapp. Relatively clean. To answer the question precisely, the question would be whether or not you could fabricate a weapon in which there were no fission products.

Representative Cole. You could not do that.

Dr. Lapp. This I do not believe is possible. So you can fabricate a cleaner weapon.

Representative Cole. Would you conclude that it is possible to fabricate a weapon which is so clean that dirtiness is not of great importance?

Dr. Lapp. I do not have the facts on which to answer that question.

Representative Holifield. No one else has come before us who had the facts to answer that question. Dr. Graves answered the exactly opposite. He said there is no such thing as a clean weapon. There are varying degrees of dirtiness.

Dr. Lapp. If I may muddy the water a little more, I would say that one must also include here in this argument about clean and dirty bombs the operational aspects of the weapon. You have first the problem of how much dirt is actually produced by the bomb, and then you have second the problem of how much dirt comes down. If you test the bomb at high altitude and set it off at high altitude, then you minimize the local fallout.

So we have two problems here. To get an index of dirtiness of an actual weapon tested you have to apply some formula here for the eternal dirtiness, and then the operational dirtiness. So it is a complex thing.

Senator Anderson. Doctor, if you had developed a type of bomb that would not explode high in the atmosphere but equipped it to explode close to the ground, would you add or subtract from its dirtiness? Would you not add to its dirtiness, so-called, to get down where it picked up particles of soil?

Dr. Lapp. When you pick up, I would call it the ballast, the soil debris, it tends to maximize the local fallout and make the weapon dirtier.

Senator Anderson. Therefore, if we were to prove whether our interest was clean or dirty weapons, we would need testimony from the military as to whether they had or had not developed weapons that would explode closer to the ground.

Dr. Lapp. I believe that the operational aspects, namely, the altitude of detonation would be very important. The testimony that Dr. Shafer gave is illustrated on this chart in which the varying degrees of contamination are illustrated by different colors. I am not going to use the exact figures. I merely wanted to illustrate the type of continental contamination that you get into if you have an attack with 2,500 megatons of dirty bomb.

I would take an example and then discuss the implications of dirtiness in a strategic attack upon a country. If you assume that 50 percent of this dirtiness falls out locally, you have then 1,000 megatons

of fission products concentrated upon Northeastern United States, the region which I use, and much of the region would then be subject to a fallout of 10,000 eternity roentgens.

May I define that? The eternity roentgen is a unit of the exposure from one hour, considering that the time of fallout to deternity, it is divided up on the following time schedule. May I jump to section C, which is the persistence of fallout. May I bypass the comments about this and jump directly to the data.

If you have 10,000 eternity roentgens, this amounts to a rate of 2,000 roentgens per hour at one hour. Here is the time table of the delivery of the roentgens to a person exposed in the open on a flat area. From the first hour, assuming the fallout occurred, then, to the end of the first day, there would be 4,700 roentgens of exposure. That would be about 1C times the lethal dose for an individual. So he had better be somewhere besides the top of the earth.

From the end of the first day to the end of the first week, there would be a dose of 1,730 roentgens, or about 3 times the lethal dose. From the end of the first week to the end of the first month there would be an additional dose of 920 roentgens which would probably also be lethal. From the end of the first month to the end of the first year, there would be slightly over 1,000 roentgens. From the end of the first year to 50 years would be an additional 840 roentgens, but I make the note that weather and terrain would make a significant difference in cutting down that dose.

This point, I think, is worthy of stressing, because of its great implication for civil defense and for analyzing what the ultimate consequences of an attack upon a country are. You have the problem here of confronting 920 roentgens from the first week to the end of the first month after the attack, and even after that is over, you have the problem of the 11-month dose of slightly more than 1,000 roentgens. After the first year, there would be a smaller dose of some 840 roentgens that would be the theoretical maximum.

Having listened to Dr. Crow's testimony yesterday, I proceeded to calculate late last night or early this morning—I forget which—just what this would mean because Dr. Pollard and the others pointed out the consequences of an attack upon the United States, and Dr. Libby this morning emphasized the great consequence of a nuclear attack itself.

Representative HOLIFIELD. I am glad you are going into this, because the chairman has received any number of telegrams—in fact, 1 was delivered at 6 o'clock this morning when my doorbell was rung by a messenger, and they got me out of bed to give it to me—condemning the committee for not going into the effects of a war and possible multimegatons. Apparently the people writing in are not aware of all the testimony, and are not aware of the fact that the extrapolation from this information can be applied to multimegaton exposure of the population. So I am glad you are bringing that point out.

Dr. LAPP. I just had the opportunity of discussing this at lunch with Dr. Crow. I have some adjustments to make with it, but nonetheless in view of the nature which he will agree is fairly approximate data, I will let these figures stand.

What I did here was the following: I assumed that by some means we were extremely fortunate in having the attack. I mean we were

fortunate after the attack in being able to hide our people from acute dosage during the first month.

I went on to calculate how much dosage these people would get if we took a generalized smear out of the radioactive fallout over the continental United States. Here is where the eternity roentgen square mile is an extremely useful concept, because I can simply make the calculations very quickly.

What I did was to arrive at through this mathematics adjusting for weathering at an average dose of 400 roentgens for the average exposure to every American who survived an attack of 2,500 megatons, with some 2,000 megatons of fission products released.

Senator ANDERSON. Doctor, it is hard to translate these things back and forth. The other day when Dr. Russell was testifying, we got into the question of the fact that if a person was exposed to so many units, it might shorten the lives of children by a certain number of days and so forth. Are these roentgens what you have called eternity roentgens the same thing he was talking about?

Dr. LAPP. I have adjusted. I used the eternity roentgen just for the simplicity of calculation. I have gone back to the pure roentgen.

Senator ANDERSON. 400 roentgens would be a very substantial dose for not only the person exposed, but would have very, very substantial effects upon children that generation and continuing, as he pointed out, for several generations to come. Geneticists did not stop when the individual was exposed. They went right along through the several generations.

Do I understand that this is a sufficiently large dose so that it would shorten the lives of those children depending upon whether you used the upper or lower limits of Dr. Russell's table from 10, 15, or 20 years or something of that general nature, if the father and mother got 400 roentgens.

Dr. LAPP. I am not familiar with Dr. Russell's data. I am sorry.

Senator ANDERSON. Are these the same roentgens he was thinking about?

Dr. LAPP. They are the same roentgens. The roentgen is the roentgen and if he was talking about the roentgen, this is the same roentgen. If he was talking about the neutron unit, this would be different.

Representative COLE. Was he talking about the neutron unit?

Dr. LAPP. As I judge from what I heard in this hearing, I thought he was testifying about the neutron unit. Is Dr. Russell here?

Representative PRICE. He definitely stated he was talking about the neutron.

Dr. LAPP. You all saw Dr. Crow's table, I believe, showing the expected genetic effect upon the first generation and upon the total succeeding generations. What I did was to calculate how this would scale up if you had an aftermath of a nuclear war under this very optimistic condition that the people who survived got no radiation for the first month, but then were exposed to a cumulative dose of 400 roentgens over the period of a generation—a reproduction generation. The first generation, according to this table, would have 1,600,000 physical and mental defects, a total of 16 million for all total generations. There would be stillbirths and childhood deaths of 4 million, a total of 120 million for all generations. Embryonic and neonatal deaths, 8 million, and 140 million total, and a much larger but unknown number of intangible defects. (See p. 1021.)

I think I would have to change this on the basis of what Dr. Crow told me, instead of 2 out of every 10 children in the United States in the first generation would be genetically defective, it would have to be 1 out of every 10. The sum total of all deferred deaths from the attack would be 272 million. I believe that should be scaled down because of what he told me to somewhat less than that. I believe about 150 million. I will have to check with Dr. Crow on this.

This, then, is the kind of genetic consequence from an attack which, according to Dr. Shafer, would have produced, I believe, of the order of 80 million deaths.

This kind of a calculation is to me a rather awesome one. When I was thinking it over and talking with Dr. Crow at lunch, I was thinking, supposing this happened and you tried to imagine what you could do about it in advance. Of course, one of the things you could do is to arrange for shelter of people, but shelter of people for the time periods we have in mind is going to be increasingly difficult even if we have the funds for it. I hope I can be forgiven for injecting in the testimony at this time a thought which I had. It is the nature of nuclear warfare which provokes this. There perhaps might be a national objective to have a stockpile of human sperm—the male sperm—which would be stockpiled at strategic locations in the United States for providing at least on the masculine side a pure line of nonirradiated sperm. I realize that this may seem like a bizarre suggestion. I understand according to biologists that you can keep human sperm viable for considerable periods of time. If you did that, then I believe you would cut your genetic consequences more than in half, because I understand that the female is less sensitive to radiation than is the male in terms of the sperm versus the ovum. This means you could cut in half or less than half—you could probably cut down between a half and a third—the consequences to future generations. It may even be, and here we would have to do a great deal of research, if you could continue the integrity and viability of the sperm through more than one generation, you then could continue nonirradiated nonmutated sperm through more than one generation.

I realize here I am getting a little fanciful. I am merely injecting this into our discussion, the kind of things you come up against when you consider the awesome consequences of nuclear warfare.

Representative Cole. Dr. Lapp, you have, of course, posed a most intriguing and bizarre as well as fanciful suggestion, but it occurs to me that is it not likely that if such an event occurred in which such a large proportion of our population were affected to the point where it would be advisable or helpful if we could have a reservoir of sperm, would not that concentration also affect other animal and plant life to a degree in which even though we were able to reproduce the human race, we nevertheless could not survive because of insufficient food, water and such?

Dr. Lapp. Mr. Cole, I believe that so far as the crops reproducing themselves are concerned, this would not be the fundamental problem, because I believe the mutation rates are quite different in crops and some of the other animals. I would like the specific question of the relative biological effect genetically to be addressed to one of the geneticists.

Representative Holifield. Of course, the suggestion you have made is an unusual suggestion, but we are dealing now with a world in

which the possibility of releasing these quantities of megatons of fission are either here or will be here very soon. It just accents the gravity of nuclear warfare and this is, of course, one of the things which mankind has to deal with for survival of the human race. If we are going to have this kind of warfare, these are the problems that are presented.

Dr. LAPP. Mr. Holifield, I personally believe that projections of the probable consequences of a nuclear warfare are in themselves the greatest deterrent to war. But this has to be absorbed on both sides of the Iron Curtain. From what Dr. Muller said yesterday about the state of genetics in the Soviet Union, I think it might be quite advisable to make sure that no one in the Soviet Union is in doubt as to the consequences of a nuclear war.

I am at a loss to say how to do this, but it might be accomplished through a good conference on genetics to which the Russians were invited.

Representative COLE. Dr. Lapp, you have indicated that since your luncheon visit with Dr. Crow you have revised your conclusions from your original script which estimated an effect on the first generation of 2 out of every 10—since your luncheon visit with Dr. Crow you have revised that downward to 1 out of 10, which is a very striking revision, a difference of a hundred percent; if that can be the consequence of a luncheon visit with Dr. Crow, might it not be conceivable that if you spent a dinner evening with Dr. Russell and other scientists, you might further revise your figures one way or another, or if you spent a week with them there might be even a greater revision?

Dr. LAPP. I am not sure that the degree of revision would be proportional to the time of contact with these individuals.

Senator ANDERSON. You would find out that at one time the Atomic Energy Commission had a figure of 50 which in a short time they brought down to 2. Maybe they should go to dinner also.

Dr. LAPP. I apologize to the committee for introducing this figure, but I did not understand from Dr. Crow's testimony yesterday that these two figures he gave were not mutually exclusive.

Representative HOLIFIELD. Mutually what?

Dr. LAPP. The point was that in the column of data he presented, he had two figures, one of which actually enveloped the other. I had thought they were separate. I was in error.

Representative COLE. I was impressed by the apparent fact that a casual luncheon conversation could result in such a striking revision of your conclusions.

Dr. LAPP. The same factor of two would have been produced from a single sentence he gave me when I showed him the results. This is entirely in the nature of how scientists iron out these differences. They talk with one another.

Representative HOLIFIELD. I might say that we have had many statistics given to us that range all the way from a factor of 2 to a factor of 10 or 15. So this variability in your figure is not unusual to other testimony we have had.

Dr. LAPP. I believe in presenting the data Dr. Crow mentioned that it probably was not exact by a factor of three. I suspect it could be even more. I am merely using this as an indication. I would claim no precision.

Representative COLE. But his admission that his calculations might be in error by a factor of 3 was based upon long periods of sober study and concentrated thought, and that even after such long period of study, he came to certain conclusions which he admits might nevertheless be in error by a factor of 3.

You have indicated that your statement today was composed in the wee hours of last night and the early hours of this morning, as well as the luncheon visit. Therefore, might it not be reasonable to conclude that your estimates might be in error by a factor of as great as 15 or 20?

Dr. LAPP. I think the physical calculation I have made I would be willing to stand on. I think it is correct within the method that I have estimated the figures are accurate. The uncertainty comes in when I apply it to the genetic data. Actually, I think, Mr. Cole, I have underestimated the situation deliberately. I have taken lower values, not upper ones.

One of the most important things that I wanted to discuss at least in my opinion before this committee, and I hope I can jump to it is the problem of the present test rate. I have indicated in the last page of this testimony my rough estimate—and I emphasize that it is a rough estimate upon all the data available to me of the rate of testing, the injection of fission products into the stratosphere over the past period of time since the first bomb was exploded in 1945.

This is a semilogarithmic plot. That is, the scale on the left in logarithmic. Starting at the bottom, the lowest value given in one megaton, and it runs up to 10 and 100 megatons of fission products injected into the stratosphere. To be perhaps redundant, may I explain that this is the number of fission products associated with the explosion of 100 million tons of TNT equivalent fission energy in a bomb. The reason why nothing appears up in 1951, up to the small value which I indicated as two-tenths of a megaton, is that prior to that time we were in the fission domain of weapons—the pure fission domain of weapons—in which the weapons had their energy released by a chain reaction in fairly expensive uranium 235 or plutonium 239, material which, as a rough estimate, we can say cost $10,000 a pound. The price is somewhat less now. Up until that time we were dealing with relatively small weapons and because of the fact that these weapons did not have great explosive power as compared with the megaton class weapons, their fission debris was restricted to the lower atmosphere or to the troposphere.

This meant that so far as the global aspects of radioactive contamination were concerned, and considering the method of transportation, the total amount of fission debris deposited was small and negligible. It was only when we entered into the era of the megaton in weaponry that we started to get into a situation where the injection of radioactive fission products into the stratosphere because of consequence and could be measured remotely all over the world.

If we look at that, we will find in the injection corresponding to the test in 1952—by the way, this includes the United States and U. S. S. R. I have not included any contribution from the British—and in 1953, relatively little testing so the curve goes down. In 1954 was when we had the Castle series of tests with a total estimated contributed fission yield of 30 megatons into the stratosphere.

The next year was cut back to a total of 3 megatons and then the next year, 1956, it went up. If you will permit me to go to another curve, I can give you the estimate. It is to about 14 megatons. I am not in a position to make an estimate of the 1957 contributions since I am not aware of what the British have contributed with the Christmas Island test, and am only partially informed of what the Russians have contributed with their spring series of tests. But I believe that this chart does illustrate some of the problems you have in discussing the present rate of testing.

One way—and this is the way Dr. Langham suggested—is that you simply average the testing over the past 5 years, which would mean we take these 5 bar graphs and add them up and divide by 5.

I believe that when this is done that this curve will come out to somewhat more than Dr. Langham estimated. I am not sure that in just a minute I can give you the answers to that.

Senator ANDERSON. Very close to it.

Dr. LAPP. It is close to it.

Representative HOLIFIELD. Of course, this excludes the Russian test.

Dr. LAPP. This includes the Russian test. I have been conservative with regard to the Russian test, probably overly conservative, because of the problem of estimating just what fission yield they had in the weapons, how much was injected into the atmosphere, and how much dumped out stratospherically.

Senator ANDERSON. Dr. Langham said it came to about 50 megatons in the past 5 years. If you add your figures, it comes out to about 50 and a fifth of that is 10 megatons per year.

Dr. LAPP. It is roughly 50, sir. I do not have the complete detail here as to how I arrived at all these figures. The reason for the Castle series of test figures being there is due to Dr. Libby. He presented the data which allows me to derive this value for the Castle series of tests. That is the principal contributor, and thus explains why my estimate should be so close to Dr. Langham's.

There is one thing which has puzzled me, that is, that if these data are in fact correct, then the Russians have not really tested a series comparable to our Castle series of tests.

Senator ANDERSON. It could be that we had one fairly sizable shot that has not yet been approximated by anybody else. That is a possible explanation.

Dr. LAPP. That is possible. I can only draw an inference.

Senator ANDERSON. Would you not be satisfied to take the total figure that Dr. Langham used in view of the fact that he is so closely associated with Los Alamos Laboratory which for many years of this period was doing the major part of the testing and has since been joined by Livermore—but the two laboratories work very closely together and their scientists are certainly knowledgeable of everything that has taken place in the tests thus far—they have the translations of what the Russian tests have been insofar as we are able to detect them, and could you not agree with him that 50 megatons would be about the total for 5 years and 10 for each year?

Dr. LAPP. I believe the agreement is very close, and I would certainly go along with Dr. Langham. The range of estimates for the test limit for the injection of radioactive material, assuming equilibrium, runs in the range of from 2 to 10 megatons, according to the discussion before this committee at the round table of Dr. Neuman and Dr.

Langham, Dr. Eisenbud, Dr. Kulp and one other person whom I have forgotten.

Because of the way I have made estimates before, I have used a value of 3 megatons per year, with a factor of 3 either way. In other words, it might be as low as 1 megaton per year or it might be as high as 9 megatons per year. One can see from this that our own test rate has exceeded this value. If we take three, it is exceeded about twice. If we take 10, it has exceeded once plus the Russian contribution, which would be a global passing limit of 2 times in the past decade.

This is assuming equilibrium. You are going to have to have more tests before you will load up the stratosphere. So from the standpoint of being reckless, the United States has not yet exceeded the limit.

I would like to make that very clear. I do not think my written statement adequately brings that out.

I realize I am taking a great deal of the committee's time, and I would like to go through some of my testimony relatively quickly. Perhaps the question of the future nuclear tests should be discussed briefly. I think in dealing with future commitments of radioactive debris to the earth's atmosphere we must deal with many unknowns. Had we attempted an estimate 5 years ago prior to Castle-like weapons we would have arrived at most misleading and optimistic projections. The end of weapon development is not in sight, and no one can say that unexpected developments may not occur. For example, may not smaller nations be stimulated by British success with thermonuclear type weapons and place maximum emphasis upon such development?

Additionally, can we be sure that a nation would restrain itself and not test a 100 megaton dirty weapon if military requirements and nuclear technology indicated that such a weapon was desirable in its own security interests.

Will not the requirements of adapting maximum megatonnage to a small warhead put emphasis on further development of dirty weapons?

I cannot answer these questions at this time, but I know that single weapons tests of very high fission yields can add a strontium burden to the atmosphere far beyond the limits we have been discussing here.

The United States has contributed the largest fraction of the radio strontium to the stratosphere, and I think it is distinctly encouraging that the fullest discussion of the strontium fallout should occur in this country. I am not aware of any large body of published information on this subject of Soviet origin. It is known, however, that the Soviets are engaged in strontium studies.

In concluding this section, I would again like to stress Dr. Libby's contributions to this subject. They are of very great value and I feel sure that we would be in a much poorer position today to evaluate the strontium problem were it not for Dr. Libby's personal interest in this field of investigation and the research which he has pushed so vigorously.

On the strontium problem itself, I would like to state briefly strontium 90 determinations in man must be expanded to assess the increase in strontium 90 burden which will occur in future time. Careful determinations of natural strontium in humans deserve increased

attention. We know that more strontium 90 will accumulate in humans as a result of bombs tested in the past and as a result of current tests. I believe that Dr. Selove is going to give a further discussion of this. The determinations as to how much of the radio element may be tolerated safely is a matter for the biologists to discuss.

This committee has heard a fairly wide range of opinion from its expert witnesses on the probable biological effects of strontium 90 in man. But it seems to me that even in this area some agreement was reached, especially when Dr. Shields Warren stated on June 3:

> I would be reluctant to see the average strontium 90 content of bones, particularly in children, go much above 10 times the present level.

Dr. Libby's speeches show that the strontium 90 fallout will continue, and the strontium 90 level in human bones will increase.

I believe that unless restraints are imposed upon commitments of fission products to the atmosphere, it is only a matter of time before the strontium 90 level of Dr. Warren will be reached.

I would not be able to extrapolate that curve very well into the future to determine this.

I would like to jump to constructive proposals—at least I believe they are constructive—if I may just insert one comment here.

A colleague, Dr. Jack Shubert, who is presently at the Laboratory for Inorganic Chemistry in Zurich, Switzerland, from the Argonne National Laboratory, and he has been in Britain recently and discussed data with the British on the question of the relative sensitivity, I would like to quote from a letter I received last night from Dr. Shubert:

> It used to be thought that at least 1,000 roentgens of absorbed radiation would induce cancer. Within the past few years, it has been found that as little as 200 roentgens delivered to children would induce cancer in later life. Now it has been found (this is by the British) that as little as 3 to 5 roentgens received by the unborn child in its last 2 months before birth has been responsible for cancer of all types appearing a few years later.

I believe that this statement from Dr. Shubert which represents the final conclusions of the data of Dr. Alice Stuart in England, is significant in that it does show that the incidence of cancer malignancy in children correlates with the X-ray of women prior to term. This is diagnostic use of X-rays which in the case of X-rays may involve of the order of a few roentgens, an amount which was thought harmless.

I believe also that this would have significant bearing upon the question of the threshold.

In order to allow time for my friend, Dr. Selove, may I jump quickly to my proposals and read them for you.

A. ATOMIC ENERGY COMMISSION INFORMATION POLICY

I suggest that this committee or its parent committee may wish to review the information policy of the Atomic Energy Commission, and I might add the Defense Department, with regard to nuclear weapon effects, with a view toward revising this policy so that information may be made available more promptly and completely. I am thinking particularly of the relations of the Atomic Energy Commission with the press. I believe that the national interest de-

mands a better relation, a freer flow of communication between the Atomic Energy Commission and the press.

My second proposal, I suggest that the Joint Committee on Atomic Energy might wish to recommend or to sponsor the preparation of an analysis of the probable biological effect of nuclear warfare. It would be useful to investigate probable lashback effects from various levels of nuclear bombardment. What I am thinking of under this category of lashback is the fallout which would occur upon the country which uses the nuclear weapon itself. That would be the remote tropospheric fallout.

The third point, data useful to civil defense. I believe that the committee's investigations have produced information of critical value to the civil defense planning. It might be useful to have a summary report of these data transmitted to the FCDA. I say that because I personally have not seen many representatives of the FCDA at these hearings.

Research in long-range estimation of nuclear explosives. It is known that considerable effort has focused on long-range detection of nuclear detonations. Attention should be given to the declassification of such data as would bear upon evaluation of the radio strontium problem. In particular I have in mind helping us to estimate how much the Russians are contributing, if this can be done, without jeopardizing sensitive data. Other data would be most useful in discussion of the feasibility of policing an agreed upon test limit, if one could get a multilateral agreement.

Annual fallout report. In view of the great public concern over fallout hazards, I would urge that the Atomic Energy Commission be required to issue an annual report on the degree of fallout and its uptake in biological systems. Perhaps the Atomic Energy Commission might wish to have that report prepared by a university task force.

Finally, I would urge that the Congress continue its investigations of radiation hazards, extending them into the broader area of peacetime uses of radiation. I believe that the ever-increasing uses of radiation must be subject to legislative controls. Radiation protection in the United States needs, in my opinion, uniform legal status.

Representative HOLIFIELD. Thank you very much, Dr. Lapp. Unless there are questions, we will hurry on to our next witness, Dr. Selove, in order that we might finish with him in time for the conference.

Representative COLE. Mr. Chairman, I should like to ask a few questions of Dr. Lapp, in order only to clear up what appears to be a conflict or some discrepancy in statements with respect to his experience and connection with the atomic energy program.

Dr. Lapp, have you seen the biography of yourself which has been prepared by the Joint Committee?

Dr. LAPP. I am sorry, I did not.

Representative COLE. I wish you would look at it if you would to see if that is a correct representation of your activities in the field of atomic energy.

Representative HOLIFIELD. I understand that the staff took that from the American Men of Science compilation.

Dr. LAPP. I believe it is correct from cursory examination.

Representative COLE. I would call your attention particularly to that part of the biography which states that you were Deputy Execu-

tive Director of the Atomic Energy Commission Joint Research and Development Board during 1947–48, and Executive Director during 1948–49. The information which has been given to me from reliable sources is the fact that you have never been an employee of the Atomic Energy Commission.

Dr. LAPP. That is correct.

Representative COLE. That is why I am giving you an opportunity to clarify this apparent discrepancy.

Dr. LAPP. May I read what it says here? It says, "Deputy Executive Director, Atomic Energy" and then there is an unusual abbreviation "Cmm Atomic Energy Committee of the Joint Research and Development Board," which was part of the Defense Department. It was Vannevar Bush's show. I am happy you brought this up. Frequently (I give lectures) I am introduced in a way which is embarrassing to me because it is incorrect. I have been introduced as everybody from the Chairman of the Atomic Energy Commission on down the line. This statement actually is correct, if you understand that is committee and not commission.

Representative COLE. How can you possibly get committee out of "Cmm"?

Dr. LAPP. I am not responsible for this. I am sorry.

Representative COLE. Then at any rate it is a fact that you have never been an employee of the Atomic Energy Commission or a consultant to the Commission.

Dr. LAPP. That is correct, Mr. Cole, I have never represented myself as such.

Representative COLE. I am not saying that you did. I am simply trying to give you an opportunity to clarify the facts of your experience in this field.

Dr. LAPP. I am glad to have this opportunity to state on this record that is correct.

Representative COLE. I further call your attention to the item "Head, Nuclear Physics Branch, Office of Naval Research, Department of Navy, 1949–52."

Dr. LAPP. That is incorrect.

Representative COLE. That is incorrect?

Dr. LAPP. That is incorrect.

Representative COLE. What is the fact?

Dr. LAPP. It was Acting Head, Nuclear Physics Branch, Office of Naval Research, Department of the Navy, 1949.

Representative COLE. 1949?

Dr. LAPP. I am sorry. Again I am not responsible for that.

Representative COLE. It is the fact that in 1949 you resigned from that post with the Office of Naval Research.

Dr. LAPP. That is correct.

Representative COLE. I do not press you on it, but would you care to give your reasons for your resignations?

Dr. LAPP. I stated on the record that I resigned of my own volition at that time.

Representative COLE. I am sure you did of your own volition. Were there peculiar circumstances surrounding the resignation?

Dr. LAPP. I had taken the job with the Atomic Energy Commission—pardon me, you have me mixed up—at the invitation of Dr. Widdel as a temporary appointment while he slid out from the posi-

tion of Head of the Nuclear Physics at that time. I took it for a short time and then resigned, I think, in June of 1949, Mr. Cole.

Representative COLE. Then there were no unusual circumstances that prompted your resignation?

Dr. LAPP. I think I would have taken this job only for a short time. I was lecturing or beginning to lecture. I was finding it difficult to lecture and also be employed by the Defense Department. But the job was temporary.

Representative COLE. That is all I have, Mr. Chairman.

Dr. LAPP. May I read the rest of this to make sure there are no other corrections? I believe it is correct as it stands now, as corrected.

Representative VAN ZANDT. Mr. Chairman, there still is in the record this statement attributed to Dr. Lapp, "reckless or nonsubstantiated statements do a disservice to the AEC and to the Nation." This statement was directed at Dr. Eisenbud, Dr. Libby and Dr. Doan. I would like to ask the Chair if we are going to give these three distinguished Americans an opportunity to answer the accusation?

Representative HOLIFIELD. Yes, sir. Would the committee like to have them come forward at this time or would you like to go forward with the next witness and have that take place in the conference?

Representative COLE. Mr. Chairman, if we are through with Dr. Lapp, I would strongly urge that we invite Dr. Eisenbud, who is in the audience, to come immediately to respond to Dr. Lapp's charge.

Senator HICKENLOOPER. Mr. Chairman, I would suggest that we finish with Dr. Lapp first. I have some questions I would like to ask.

Representative HOLIFIELD. Senator Hickenlooper has some questions, Dr. Lapp.

Senator HICKENLOOPER. Dr. Lapp, have you ever done what might be called extensive works in genetics under your own responsibility in connection with the effects of radioactivity on human cells?

Dr. LAPP. No, sir.

Senator HICKENLOOPER. Have you ever done any research in biology and medicine in connection with the effect of radioactivity on the genes or other parts of the human body?

Dr. LAPP. I have never done any research on genetic effects.

Senator HICKENLOOPER. Have you ever conducted any research yourself on the fallout or its intensity in this country? By that I mean any laboratory research of any extent.

Dr. LAPP. I have made some simple measurements myself, but my data here and my testimony is based upon the data of the Atomic Energy Commission.

Senator HICKENLOOPER. That was the next suggestion that I wanted to make. I have the impression here—I do not know whether I am correct or not—that you are appearing here more in the nature of a reporter or a correlator with some considerable educational background, I admit, of the scientific data as you personally interpret it, which has been compiled by a number of eminent scientific people who have actually done the work.

Dr. LAPP. I believe the invitation that was extended to me by the committee more or less put me on these lines of testimony.

Senator HICKENLOOPER. Yes. The only purpose of this suggestion is that you are not here, while you are a scientist in your own right on your education, giving firsthand evidence based upon data with which you have been acutely or intimately connected in connection with its

development. So you are reporting what you have read or what you have been told by others with varying degrees of accuracy and drawing your own conclusions.

Dr. LAPP. Mr. Hickenlooper, I think it is a fair statement to say that I have done considerable work based upon information made available by the Atomic Energy Commission and scientists in general. I have done active work in gathering data from people, especially as every scientist will do, when available.

Senator HICKENLOOPER. I know that. I make the point that there are several very eminent newspaper reporters in the scientific field who have also done a tremendous amount of work of gathering data and yet they have done no research of their own. They are reporters and in a very proper field they are reporting what they find. I merely have the impression that you are here today in the nature of a reporter or a compiler of information which you are interpreting as you see it. As differentiated, I might add, from the testimony of most of the rest or all of the rest, perhaps, of the witnesses we have had here who have been intimately working in this field and present the results in the main on their own experience, and the results of their own work.

Dr. LAPP. I believe, Mr. Hickenlooper, this is within the framework of the outline which requested me to testify.

Representative HOLIFIELD. I am very sorry. In order to correct the record, the Chair will have to state that Dr. Libby is a chemist, and the information he has given us in every field of science has been obtained from his association with other scientists and reading of other materials. So in order to keep the record straight, let us have the facts spread there that it is not only Dr. Lapp who had studied the other scientists' work and reported on them, but also Dr. Libby, one of the AEC Commissioners.

Dr. LAPP. May I say in general response, not specifically to Mr. Hickenlooper, that I admit to being critical of the Atomic Energy Commission. My criticisms of the Atomic Energy Commission, I always felt, have been directed toward trying to bring the facts out into the open, and the free play of public discussion. As Dr. Libby testified this morning, some of the problems involved here transcend the area of science. I have felt that this is important. I have tried to help in the somewhat new but I think important problem of education in science.

Senator HICKENLOOPER. Then where do you obtain your facts? Do you have access to restricted data?

Dr. LAPP. I have no access to restricted data, Mr. Hickenlooper. The facts that I have presented here are based upon information which is available freely in the scientific domain.

Senator HICKENLOOPER. Not only in the scientific domain, but from the Atomic Energy Commission also. Is that not true?

Dr. LAPP. Such data as the Atomic Energy Commission published. We were assured this morning by Dr. Libby that all of the data except for a very small fraction dealing with a long-range detection on Project Sunshine had been put in the public domain.

Senator HICKENLOOPER. What information do you have that the Atomic Energy Commission is not making available, if you have no access to restricted data, and if your information comes from data freely available from various sources including the Commission? Does not that indicate that data is being made available?

Dr. LAPP. If I may respond to the question, Senator Hickenlooper, my complaints in the past about the slowness with which the Atomic Energy Commission emitted data, which I thought were vital to civil defense, were, for example, that the data were not available. They had to be derived by me from other sources, other than the Atomic Energy Commission. As for example, the scientific data from Japan. Thus I think the fact that later on the Atomic Energy Commission actually confirmed through its pronouncements that these results were correct testified——

Senator HICKENLOOPER. Have you had an opportunity to go through the data in minute detail from Japan, examine the records and all the data that they have developed, or are you relying entirely upon the verbal statements of certain scientists made to you in the course of your visits with them?

Dr. LAPP. No. I have been in communication with Japanese scientists for some time. I have received letters and photostats and copies of their scientific papers, in some cases prior to publication.

Senator HICKENLOOPER. Thank you very much.

Representative COLE. Mr. Chairman, I had intended to interrogate Dr. Lapp just a little bit about his visit to Japan earlier when he referred to his visit in his statement. While I hate to keep him on the stand unduly, I am curious to have his observations on two points.

One is, Dr. Lapp, that you were registered as one of the scientists who would attend the conference.

Dr. LAPP. Yes.

Representative COLE. And yet you did not attend the conference.

Dr. LAPP. No; I did attend, Mr. Cole.

Representative COLE. It was reported to me, and the reason I raise the question is because that same question was raised by a number of persons who were there, as to the reason why you had not attended the conference.

Dr. LAPP. I would be glad to explain this.

Representative COLE. That is one point. Then the other, since you remained in Japan after the conference was concluded, I am interested to have your observations with respect to the accomplishments of the conference.

Dr. LAPP. On the first point, I arrived in Japan on, I believe, May 1, considerably in advance of the conference. It was my firm intention to attend every session of the conference, because I am very much interested in the work of the Atomic Industrial Conference. However, when I got to Japan, I found myself in a pretty mad race because I was digging up some data on the *Lucky Dragon* story, interviewing some of the fishermen who were in this unlucky boat; and I wanted to visit Hiroshima and visit some scientists in the various places. I found that when I had to deal with just the problem of hours spent in talking with the fishermen that by the time I got back to Japan the conference had already started. This was unavoidable, but I had to meet people at certain places.

Then the amount of commitments I had with the press and visiting the *Lucky Dragon* itself precluded my going to more than one session.

Representative COLE. Which session was that? It does not matter.

Dr. LAPP. I can give it to you. I did attend that. I wanted very much to attend the luncheon and dinner sessions. I had all the tickets, and I just could not use them. For example, the one night I wanted

to do that, I think I was invited to a banquet by one of the prominent newsmen. So I, unfortunately, did not have much time.

As for the second point, just what was my reaction, I did not have too much opportunity to discuss with the Japanese scientists their reaction to the conference, but I gathered from conversation with a few of them that they were greatly impressed with the conference. It was very well managed. It was well attended. I think they were very much impressed with the general conference.

Representative COLE. Then your conclusion is that it was a very worthwhile conference.

Dr. LAPP. I would say so.

Representative COLE. That is my own conclusion.

Dr. LAPP. Yes.

Representative COLE. I can also verify your statement that it was an extremely well-organized conference involving a considerable number of people. There were some meetings that were attended by as many as 4,000, 5,000 and 6,000 Japanese. It was an unusually well-organized conference, probably the first international conference of industrialists and scientists that has ever been held.

Dr. LAPP. I would in no way like to detract from the value of this conference. My only regret is that I was able to attend so few sessions. I cut short my visit in order to come back here. I would have liked to stay in Japan much longer.

Representative COLE. My only basis for concluding that you had not attended the sessions of the conference is that I inquired—because I looked forward to seeing you there—inquired at the registration desk at the end of the conference, and they said you had not, so far as they knew, attended the conference, and the papers in the box—there was a slot for each of the delegates—were still there.

Dr. LAPP. I have the papers in my office, Mr. Cole. Again I regret, and no one does more than I, that I could not attend these conferences. I was working. The press in Japan can at times be very aggressive. Perhaps you found that out yourself.

Representative COLE. Yes.

Dr. LAPP. I found myself going to a 15-minute interview and ending up with a 2-hour luncheon.

Representative COLE. Now, with respect to your sources of information, Dr. Lapp, you have indicated that your observations back through the years are based on reports and studies of unclassified data that have come to your attention. Are they not, also based on discussions with other newspapermen and analysts in this field, such as yourself?

Dr. LAPP. I am proud to say that I communicate with a great many members of the press.

Representative HOLIFIELD. Mr. Eisenbud, you are now given an opportunity to reply to the comment of your colleague in science, Dr. Lapp.

Senator ANDERSON. Before you do so, Mr. Eisenbud, may we sort of follow along in this same pattern of qualification? I look at your qualifications in here, and I see "EE, New York University." Does that mean "electrical engineer"?

Mr. EISENBUD. Yes.

Senator ANDERSON. What subsequent degrees have you acquired?

Mr. EISENBUD. I have no subsequent degrees.

Senator ANDERSON. Just the degree as electrical engineer?

Mr. EISENBUD. Many years ago; yes, sir.

Senator ANDERSON. I see you spent more time working for the Liberty Mutual Insurance Co. as a hygienist than anything you have done in your life and more than all the rest of your experience put together; is that right? Eleven years with them, and only 10 years since then.

Mr. EISENBUD. That is about right.

Senator ANDERSON. I am not trying to be critical, because I tried to speak appreciatively of what you had done a few moments ago. As a hygienist, did you ever have occasion to get into the question of deposit of strontium 90 in the atmosphere for the Liberty Mutual Insurance Co.?

Mr. EISENBUD. This was previous to 1947. Up until that time very little strontium 90 had been formed in this world. The radiaition problems of those days were X-ray and radium. We were only beginning to get interested in the kinds of things which we are talking about today.

Senator ANDERSON. The field that you were in had to do with the detailed discussion of the occurrence of strontium 90 and cesium 137 in the atmosphere, biosphere, and its uptake and behavior in man. What was there in your electrical engineering course that dealt with that?

Mr. EISENBUD. Very little, sir. But a great deal in some 21 years of professional experience, during which time I have attained the rank of adjunct professor of industrial medicine at New York University Medical School.

Senator ANDERSON. What was there in this work with the Liberty Mutual Insurance Co. as a hygienist that started you off lecturing on medicine?

Mr. EISENBUD. I got interested, about 22 years ago, in a legitimate subject for a young electrical engineer, namely, the electrical charges on dust, and went from there to the general physical properties of dust, and then into the physiology of dust and dust diseases, and spent a great deal of time from 1936 until 1947 studying the general behavior of dust, not only in the atmosphere, but in the lung and in the body.

Senator ANDERSON. Your paper was headed, "A Measurement of Strontium 90 in Geophysical and Biological Material." Are you a geophysicist?

Mr. EISENBUD. Sir, I do not know, really, how to answer that. I think anybody that has some interest or qualification in geography and geology and some in physics could at least write on the subject of geophysics, but I am not a geophysicist.

Senator ANDERSON. Are you a biological worker.

Mr. EISENBUD. I work in the biological field. I, myself, am not a biologist.

Senator ANDERSON. Have you conducted experiments in this measurement of strontium 90?

Mr. EISENBUD. I have directed the experiments, and have conducted some myself.

Senator ANDERSON. Have you conducted them? Have you done any experimental work yourself?

Mr. EISENBUD. The experimental work was performed under my immediate supervision. I have done some of the experimental work myself.

Senator ANDERSON. Most of it was done under your supervision?

Mr. EISENBUD. This has been a large program. This is much too large for one man. It was done by my immediate staff.

Senator ANDERSON. Then you are here as a reporter of what someone else has done.

(A letter from Merrill Eisenbud, setting forth a full record of his qualifications, follows:)

UNITED STATES ATOMIC ENERGY COMMISSION,
NEW YORK OPERATIONS OFFICE,
New York, N. Y., July 12, 1957.

Mr. HAL HOLLISTER,
Staff Member, Joint Committee on Atomic Energy,
Congress of the United States, Washington, D. C.

DEAR HAL: You will undoubtedly recall that during the proceedings of June 5, the question of my professional qualifications was raised by Senator Anderson. If this portion of the testimony is to be included in the published proceedings, it would be desirable, for the sake of completeness, that a full record of my qualifications be included as well. The attached curriculum vitae is somewhat more complete than the record to which Senator Anderson referred and which I believe was the Men of Science abstract.

I am also attaching a list of my publications. I do not wish that this list be included in the proceedings but I simply submit it as a matter of record for the files of the subcommittee.

I continue to hear favorable reports about the hearings. We all look forward to the final proceedings.

With best regards.

Sincerely,

MERRIL EISENBUD, *Manager.*

BIOGRAPHICAL DATA, MERRIL EISENBUD, JUNE 10, 1957

Date of birth: March 18, 1915, New York City
Education: New York University, College of Engineering (1932–36) B. S. in E. E. 1936
Positions held:
- Industrial hygienist, Liberty Mutual Insurance Co., 1936–47
- Chief, Industrial Hygiene Branch, Health and Safety Laboratory, United States Atomic Energy Commission, 1947–49
- Director, Health and Safety Laboratory, United States Atomic Energy Commission, 1949 to present
- Manager, New York Operations Office, United States Atomic Energy Commission, 1954 to present
- Senior scientific advisor, Preparatory Commission of the International Atomic Energy Agency, 1957

Lecturer, Columbia, School of Public Health, 1945–50
Adjunct associate professor, department of sanitary engineering, New York University, 1945–50
Associate professor, industrial medicine, New York University, department of industrial medicine, 1950–55
Adjunct professor, industrial medicine, Postgraduate School of Medicine, New York University, 1955–
Committees:
- National Research Council:
  - Toxicology Committee, 1952–
  - Committee on Atmospheric and Industrial Hygiene, 1952–
- American Standards Association:
  - Subcommittee on Radium, Dust, and Radon Gas, Z37, 1949–
  - Sectional Committee on the Use of X-Rays, Z-54, 1951–
- National Safety Council: Executive committee, Chemical Section, 1951–
- Radiological Advisory Committee: Office of Civil Defense, City of New York, 1950–
- Technical advisor, United States delegation, U. N. Conference on the Peaceful Uses of Atomic Energy, Geneva, 1955
- Alternate United States representative, U. N., Scientific Committee on the Effects of Atomic Radiation, 1956–.

Member, National Academy of Sciences Committee on the Meteorological Aspects of the Effecvts of Atomic Radiation 1956–.
Aspects of the Effects of Atomic Radiation, 1956–.
Scientific adviser, United States delegation, Conference on the Statute of the International Atomic Energy Agency, 1956–.
Member, World Health Organization, Expert Advisory Panel on Radiation, 1957–.

Memberships:

American Industrial Hygiene Association (board of directors, 1955–58), American Public Health Association, New York Academy of Science, Radiation Research Society, American Association for the Advancement of Science.

LIST OF PUBLISHED WORK OF MERRIL EISENBUD

Global Distribution of Strontium-90 from Nuclear Detonations, Scientific Monthly (May 1957), pp. 237–244.
Monitoring Network for Measuring Radioactive Fallout, J. Am. Water Works Association, Vol. 48, No. 6 (June 1956).
Radioactive Fallout Through September 1955, Science, Vol. 124, No. 3215, p. 251 (August 10, 1956) (with J. H. Harley).
Industrial Hygiene of Uranium Processing, A. M. A. Arch. Ind. Health, Vol. 14, pp. 12–22 (July 1956) (with J. A. Quigley).
Atmospheric Contamination, Chapter 11, Radiation Protection published by Thomas & Co. (in press).
Radioactive Fallout in the United States, Science, Vol. 121, No. 3150, pp. 677–680 (May 13, 1955) (with John H. Harley).
Health Hazards from Beryllium, Chapter 12, published in The Metal Beryllium by the American Society for Metals, 1955.
Industrial Hygiene and Medical Survey of a Thorium Refinery, Arch. of Industrial Health (March 1955) Vol. 11, pp. 234–242 (with Roy Albert, Paul Klevin, John Harley, J. Fresco and W. B. Harris).
How Important Is Surface Contamination?, Nucleonics (August 1954) Vol. 12, No. 8, pp. 12–15 (with Hanson Blatz and Eugene V. Barry).
Radioactive Dust From Nuclear Detonations, Science (Feb. 1953) Vol. 117, No. 3033, pp. 141–147 (with John H. Harley).
Dust Sampler Which Simulates Upper and Lower Lung Deposition, Arch. Ind. Hyg. and Occup. Med. (November 1953), Vol. 8, pp. 446–452 (with W. B. Harris).
Field Equipment for the Collection and Evaluation of Toxic and Radioactive Contaminants, Arch. Ind. Hyg. and Occup. Med. (June 1953) Vol. 7, pp. 490–502 (with W. B. Harris and H. D. LeVine).
Retention, Distribution and Elimination of Inhaled Particulates, with Particular Reference to the Evaluation of Radiologic Risk, Arch. Ind. Hyg. and Occup. Med., (September 1952) Vol. 6, pp. 214–225.
Epidemiology of Beryllium Intoxication, Arch. Ind. Hyg. and Occup. Med. (August 1951) Vol. 4, pp. 123–151 (with James H. Sterner).
A Method of Obtaining Reproducible Breath Radon Samples, Arch. Ind. Hyg. and Occup. Med. (July 1951), Vol. 4, pp. 1–9 (with John H. Harley and Evelyn Jetter).
Meterological Technics in Air Pollution Surveys, Arch. Ind. Hyg. and Occup. Med. (January 1951), Vol. 3, pp. 90–97 (with W. B. Harris).
Radiation Hazards in the Atomic Energy Program, Ind. Med. and Surgery, (January 1951), Vol. 20: 1, 7–11.
Fire Protection Precautions for Uranium Scrap and Powder, Nucleonics (May 1950), Vol. 6, No. 5, pp. 34–37 (with Edward J. Kehoe and Francis L. Brannigan).
Nonoccupational Berylliosis, J. Ind. Hyg. and Toxicol. (1949) Vol. 31: 5 (with R. C. Wanta, Cyril Dustan, L. T. Steadman, W. B. Harris and B. S. Wolf).
Environmental Studies In Plants and Laboratories Using Beryllium: The Acute Disease, J. Ind. Hyg. and Toxicol. (1948) Vol. 30: 5 (with C. F. Berghout and L. T. Steadman).
Health Hazards In Aircraft Manufacturing, Industrial Medicine (March 1949) Vol. 18: 3, pp. 99–102.
The Comparative Performance of Air-Supplied Welding Helmets, Welding Journal (April 1948) (with Leslie Silverman).

Mercury Exposures In Dry Battery Manufacture, J. Ind. Hyg. and Toxicol. (November 1947) Vol. 29, No. 6 (with Charles R. Williams and Stanley E. Pihl).

Control of the Lead Poisoning Hazard in Can Manufacturing, J. Ind. Hyg. and Toxicol. (December 1945), Vol. 27, No. 10 (with J. W. McEwan).

The Principal Health Hazards in Metal Finishing Departments and Their Control, Metal Finishing (1942).

Mr. EISENBUD. Thank you.

I am deeply gratified, Mr. Chairman, at the opportunity to correct the record with respect to the item which Dr. Lapp has included on page 2, paragraph C, in which he quotes a New York newspaper sentence and illustrates what, I think, needs not be illustrated; namely, the danger of taking something out of context. The date of that quotation is March 20, 1955. This was a very jittery period. This was immediately following the announcement within the AEC of the results of the March 1, 1954 detonation in the Pacific. It also coincided with the beginning of the Teapot series of detonations in Nevada, which, if my recollection is correct, began a week or two prior to this announcement, or perhaps shortly thereafter.

In any case, the fact that the first post-Castle detonations were about to take place in Nevada was very much in the minds of many of our citizens.

This reporter came to me to find out whether, in my opinion, the kind of accident which occurred in Bikini in 1954 could happen in this country as a result of the test being contemplated or already underway in Nevada. We were not talking about the long-range hazards of fallout. We were not talking about strontium 90. We were talking about the kinds of acute effects which one had, unfortunately, seen in both the Marshallese and Japanese fishermen in 1954.

My comment, which may or may not have been quoted accurately—I really don't know—had to do, primarily, with the relationship of acute effects to the kinds of radiation levels that are expected from the Nevada test, which are of the order of 1 milliroentgen or thereabouts, at least, in the United States.

Senator ANDERSON. Mr. Eisenbud, this statement was made on March 20, 1955. I understand you do not question the accuracy of the quotation.

Mr. EISENBUD. I do not question the accuracy of the quotation nor will I certify it nor will I certify the accuracy of Dr. Lapp's quotation of a quotation.

Senator ANDERSON. If it should prove he is accurate you are willing to stand by it.

Mr. EISENBUD. I am willing to accept this version of it.

Senator ANDERSON. The testimony introduced by Dr. Lapp and the chart introduced showed that we had the Castle series in 1954 in which we had put in the atmosphere more fission products perhaps in that year than we have put in all the rest of the tests together. Therefore we were at our highest peak when you made your comment. The figure was several times above the figure which Dr. Langham has said is a safe figure year by year.

It is your testimony that we could do a million times that and do no damage.

Mr. EISENBUD. No, sir. This was not the question which was asked of me. I was asked specifically whether there was any possibility in the eastern United States of an accident which would produce the

kinds of illness among the people that were seen in the Marshallese and Japanese fishermen.

This would require around 100 roentgen. The radiation doses which had been observed were something less than a milliroentgen. This is a ratio of about a million to one. When you put it into context it is perfectly accurate.

Senator ANDERSON. That is what I am trying to get to. We had just finished the Castle series. You would agree with that; would you not?

Mr. EISENBUD. That was a year before.

Senator ANDERSON. We had finished them in 1954 and this statement was made March 20, 1955. We had finished the Castle series, had we not?

Mr. EISENBUD. Yes, sir.

Senator ANDERSON. You said the total fallout to date from all tests would have to be multiplied by a million to produce visible deleterious effects except in areas close to the explosion itself. It is your testimony that having put that year into the stratosphere, or whatever fallout pattern there was, something in the neighborhood of 30 megatons of fissionable products, that we would have to put a million times that for it to have any ill effect except in the immediate vicinity of the test. Is that correct?

Mr. EISENBUD. That is incorrect.

Senator ANDERSON. You think that is a somewhat reckless statement?

Mr. EISENBUD. This is not what I said.

Senator ANDERSON. That is what I am trying to get to. The paper misquoted you.

Mr. EISENBUD. No, sir. I have not seen that quotation in 2 years. This sentence is out of context.

Senator ANDERSON. I am reading it to you. If it should prove to be an exact quotation, is it a reckless statement?

Mr. EISENBUD. Out of context; no.

Senator ANDERSON. In or out of context, is it a reckless statement to say that the Castle test which included an extremely large shot which turned loose in one series of tests as much fission products as probably all the rest of the tests by all the rest of the countries? If you can place reliance on the data gathered at Los Alamos, do I understand in context or out of context, you could have a million times that and have no visible deleterious effects except in the immediate area? Do you wonder that looking at that statement Dr. Lapp thought it might be a slight degree of recklessness?

Mr. EISENBUD. Sir, I would like Dr. Lapp to comment on what I have——

Senator ANDERSON. The point is that they wanted to give you an opportunity, and this is your day.

Mr. EISENBUD. Yes, sir. I think Dr. Lapp will probably understand what I am talking about. Let me say this, sir: I think it is a great misfortune——

Senator ANDERSON. Dr. Lapp was treated critically because he had used this language. They said in all fairness you would have an opportunity to reply. This is your hour for fairness. Why don't you go ahead and reply?

Representative COLE. I think he would if you let him.

Mr. EISENBUD. I think it is a great misfortune, sir, that to add to public confusion we find it necessary to discuss in the same session the long-range hazard and the close in hazard. For most of these sessions we have been talking about the long-range hazard from strontium 90 and from the gamma radiation.

In this interview I was talking about the close-range hazard, the kind of hazard that may develop within a few hours after detonation and the kind of hazard which produced illness in the Marshallese and Japanese in 1954.

Senator ANDERSON. Then from the close-range hazard—we will just confine it to that—since we put some 30 megatons of fission products in the atmosphere in the Castle test just passed, you believe that we could safely put a million times that in a single year in a test without doing any damage?

Mr. EISENBUD. Certainly not, sir.

Senator ANDERSON. What were you talking about? If it is not from the close range or long range, what other range is there?

Mr. EISENBUD. I was talking about the immediate gamma radiation from the fallout which occurs in the eastern United States within a matter of a day or so after a detonation in Nevada. This is not in the statement because the statement has been taken out of context.

Senator ANDERSON. Could that have been apparent if Dr. Lapp had read the whole interview?

Mr. EISENBUD. I can't vouch for the validity of the interview, sir. I do not recall this.

Dr. LAPP. I would like to make this statement in all fairness to Dr. Eisenbud, and I certainly do not mean to attack his integrity. The reason for quoting these and the use of the word "reckless" was to demonstrate the need for being quite exact when dealing with such a touchy subject as radioactivity. I am trying to interpret this recklessness not in terms of Dr. Eisenbud's personal recklessness but in terms of how it may appear to people who read these things and who do read single sentences.

Senator ANDERSON. I quite agree with you. I tried to say earlier that I think Mr. Eisenbud is a very fine public servant. I went up to him and told him that I appreciated very much the testimony he gave the other day. I did not regard your statement about him as a vicious attack upon him. But if it comes down to whether it was reckless, there are people who read that statement alone who would think it had just a slight tinge of recklessness in it, since the Castle test had just been finished.

Dr. LAPP. May I ask a question of Dr. Eisenbud? Is this permitted?

Representative COLE. Don't look at me.

Representative HOLIFIELD. I think——

Dr. LAPP. I would like to ask one single question.

Representative HOLIFIELD. You might ask the Chair a question.

Dr. LAPP. I would like to ask the Chair a question, as to what the roentgen dosage on the fallout on Troy, N. Y., was.

Representative HOLIFIELD. The Chair has been told, but the Chair has temporarily forgotten now. The Chair will ask Dr. Eisenbud if he recalls.

Mr. EISENBUD. Yes, sir. It has been variously estimated.

Representative HOLIFIELD. Is it an unclassified amount?

Mr. EISENBUD. It is unclassified. It was published 3 years ago. The upper limit of estimate is something under 100 milliroentgen. It loses about 1 milliroentgen. I would personally estimate it at about 10 milliroentgen.

Representative HOLIFIELD. The Chair thanks the gentleman for that information.

Representative COLE. Could I inquire, in order for the information to be helpful, if these samplings that occurred were related to what tests? When were the samplings taken which you say indicated an upper limit of 100 milliroentgen?

Mr. EISENBUD. I believe it was the third or fourth test of the series held in Nevada in the spring of 1953. There was a rain out over the Troy-Albany area which coincided with the passage of a cloud from Nevada. So that a very large percentage of this cloud was washed down.

Senator ANDERSON (presiding). Are there additional questions or statements?

Representative VAN ZANDT. Dr. Eisenbud, I understand your position is that the statement that Dr. Lapp attributes as being a reckless one was taken out of context.

Mr. EISENBUD. Yes, sir.

Dr. LAPP. Is it proper for me to respond? I have done a little arithmetic. Let us take 10 milliroentgens, as Dr. Eisenbud estimates, and we multiply 10 milliroentgens. That would be .01 roentgens by 10 to the sixth, which will give us 10 to the fourth, which is 10,000 roentgens.

Senator ANDERSON. 10,000 roentgens would kill everybody in sight.

Mr. EISENBUD. Yes.

Senator ANDERSON. So that would mean there would not be any immediate danger if you kill everybody in sight?

Representative PRICE. Mr. Chairman, one of the points that we do not want to overlook is that Dr. Lapp is trying to point out the responsibility of the Commission to release information as promptly as possible so that these types of statements would not be made. Is that not one of the reasons for you citing this statement?

Dr. LAPP. I really feel if we had better relations here between the press and the Atomic Energy Commission we could in a minute avoid much of this difficulty.

Representative PRICE. That is the reason I understood that you gave this example.

Dr. LAPP. Yes.

Senator ANDERSON. May I say, Mr. Eisenbud, that I am truly sorry that Dr. Lapp's quotation has caused any embarrassment. I want to repeat what I said before. I certainly regard you as a fine public servant doing a good job. I am very happy that you are here make your contribtuion today.

Mr. EISENBUD. Thank you, sir.

(The full statement of Dr. Ralph E. Lapp follows:)

STATEMENT OF RALPH E. LAPP ON RADIOACTIVE FALLOUT

EXPLANATORY NOTE

Mr. Holifield, I received your invitation to testify before this committee while I was in Japan. I cut short my trip in order to attend the hearings. May I

say that I appreciate very much this opportunity to appear here. I would like to add that I am very gratified that your investigations to date have thrown so much light on the problem of radioactive fallout. I believe that these hearings will stand as a landmark in the history of our knowledge about this relatively new phenomenon.

COMMENT ON DR. LIBBY

Appearing as I do after Dr. Libby, I would like to comment on his contributions to fallout. Dr. Libby has not only stimulated extensive research in fallout investigations such as Project Sunshine, but he has also taken the initiative in publication of his findings. I feel very strongly that he deserves a great deal of credit for his work on fallout. Were it not for Dr. Libby we might well be confronted with a considerably smaller body of knowledge about fallout than we have today.

MY INTEREST IN FALLOUT

I have had an active interest in atomic bomb phenomenology ever since I witnessed the Bikini Baker test in the summer of 1946. However, my interest in radioactive fallout was really stimulated by the 1954 Bravo test at Bikini. This was the test which resulted in radioactive contamination of the *Lucky Dragon No. 5*, a Japanese tuna trawler.

My initial interest in fallout centered upon civil defense. In this connection, I published a series of articles on fallout in the Bulletin of Atomic Scientists as follows:

November 1954: Civil Defense Faces New Peril
February 1955: Radioactive Fallout
June 1955: Radioactive Fallout III
November 1955: Global Fallout
September 1956: The "Humanitarian" H-Bomb
October 1956: Strontium Limits in Peace and War.

NATURE OF MY TESTIMONY

I am dividing my testimony into four parts:

I: General Remarks
II: Local Fallout
III: Remote Fallout
IV: Constructive Proposals

Because of the number and complexity of the topics covered, I am presenting my remarks in terse or fragmentary form. This will permit the committee to bypass topics of less importance and concentrate upon those of more concern.

PART I. GENERAL REMARKS

A. Necessity for numbers

Public confusion about fallout will continue to increase unless scientists can provide a quantitative or semiquantitive evaluation of the various hazards associated with fallout. Precision is probably not possible due to the nature of the hazards and we may have to be content with numbers which vary by a factor of 2, 3, or even 10. Th is committee has already performed a valuable service in narrowing the range of estimates made by individual witnesses.

B. Disagreement among scientists

The public is apt to conclude that if scientists cannot agree upon the hazard, then all is confusion. It would be nice if the scientists could all agree upon a quantitative estimate of the hazard, which could then be given to the public. Two unusual circumstances have combined to produce the current confusion on fallout.

First, the urgency of our times has focused attention upon problems for which science did not have textbook answers. Available knowledge was inadequate and research had to be initiated to provide answers.

Second, the ordinary process by which scientists argue out their answers was interdicted by the complexity of the problem and secrecy. Scientists outside the Atomic Energy Commission have full-time jobs and could scarcely be expected to tunnel into the complexities of the problem in a few leisure hours.

C. Responsibility of the Atomic Energy Commission

Considering these factors, I think that the AEC has the responsibility for providing the outside world with the facts about fallout as promptly as these become available. Scientists, technicians, and officials of the AEC must present only reasoned and careful estimates of the hazards based upon factual knowledge. Reckless or nonsubstantiated statements do a disservice to the AEC and to the Nation.

Example: Dr. Eisenbud is quoted in an article titled "Man Who Measures A-Fallout Belittles Danger" (Sunday News, New York, March 20, 1955) as follows: "The total fallout to date from all tests would have to be multiplied by a million to produce visible, deleterious effects except in areas close to the explosion, itself."

Example: Dr. Libby in a speech dated June 3, 1955, stated: "However, as far as immediate or somatic damage to the health is concerned, the fallout dosage rate as of January 1 of this year in the United States could be increased 15,000 times without hazard."

Example: Dr. Richard Doan while in Tokyo on May 13, 1957 stated that the bomb tests would not have "the slightest possible effect" on humans.

I do not label Dr. Libby's statement as reckless but interpose it to illustrate the spectrum of opinion being given to the public.

D. World interest in fallout

I am informed by a cable from Tokyo that the deliberations of this committee hearing are being "splashed across page 1" of the Japanese newspapers. This comes as no surprise to me for my trip through Japan alerted me to the overwhelming interest manifested there in atomic radiation.

The committee might be interested in my observation that fallout has become an acute weapon for propaganda. For example, I found that the Japanese scientists are actively studying the radioactivity of their tea because of the assertion from the Chinese mainland that Japanese tea is radioactive. Some people in Japan are so keenly aware of fallout that they take showers after being out in a rain. The great public outcry against the British Christmas Island tests, but there was no great demonstration against Soviet tests. It is a great victory for psychological warfare experts when they can induce selective sensitivity to fallout.

America puts itself in a bad light when it fails to present its case clearly to the world. Even casual analysis of the news reporting in this country will show that AEC pronouncements on fallout are not received with full credibility. This situation is obviously not in the full interests of national security.

E. The nature of biological data

It is inherent in the very nature of the biological research into the effects of radiation upon humans that a high degree of accuracy is not attainable, especially on a human experience basis. As Dr. Langham of the Los Alamos Laboratory has testified human experience with retention of radium 226 is the basis for setting upon a maximum permissible concentration (MPC) for radiostrontium (Sr–90). Yet our actual experience is confined to a small sample of acutely exposed individuals and a small sample of less acutely exposed people.

Actually, our concern should focus not upon acute effects in man which are highly unlikely from peacetime bomb testing, but rather with the chronic, debilitating long-term effects from irradiation of humans. We must be conscious of the need to appraise long-delayed effects, say, 50 years after entry of radioelements into the body. Here our knowledge is quite limited.

F. Radiation limits for a global population

I would like to stress the fact that consideration of safe limits for irradiation of the world's population is essentially a new problem. Prior to the awareness of global fallout, the International Commission on Radiological Protection made its recommendations for those who would be exposed to radiation in pursuit of their occupation. Such groups initially were numbered in the hundreds and then in the thousands as atomic energy came of age. Individuals within such groups were healthy adults exposed to known and restricted hazards; they were subject to administrative controls and medical supervision.

In setting up limits for a total population, we must take into account the varying radiosensitivity of individuals, the complete spectrum of age, the persistence of the hazards, the lack of medical control, the varying degrees of health of people and the variety of their diet. Yet it was not until last year that the

Atomic Energy Commission introduced the difference between an occupational MPC and a global MPC into its releases on fallout.

In view of the nature of our knowledge and the totality of the sample with which we are dealing, I would urge a big factor of safety in setting limits to bomb testing. It would be tragic to find some day that we had erred in setting the limits.

G. Soviet nuclear tests

On my recent trip to Japan, I learned that Japanese scientists collected sufficiently active samples from Russian tests to perform radiochemistry upon the bomb debris. I am informed that five Soviet tests produced a fallout on Japan from which scientists measured and identified the presence of uranium 237 (U–237). Soviet explosions characterized by such fallout were judged to be in the megaton range. These estimates are subject to considerable uncertainty but one authority told me that he estimated at least two bomb yields in the range of 10 megatons.

Two Soviet nuclear tests were observed to originate in the arctic region whereas the remaining tests took place in a region estimated to be Ozero Balkash (Lake Balkhash) which is southeast of the new coal area of Karaganda. The air mass trajectories from central Siberia frequently sweep across the islands of Japan, especially Hokkaido. They also produce tropospheric fallout over the United States as well. Here in Washington you could swipe a Kleenex over a car top and cause a Geiger counter to respond readily.

The presence of U–237 in the Soviet fallouts proves that the Soviets have achieved a compound fission-fusion or multiple-stage weapon. According to my information, this was first accomplished in September 1954.

I would like to add that I am informed by Japanese sources that Soviet tests produce 70 percent of the fallout observed on Japan. Pacific tests account for 20 percent and the Nevada shots add 10 percent.

PART II. LOCAL FALLOUT

Definition: By local fallout, I mean that which comes to earth within several hundred miles of the explosion site and is deposited within the first day or so. The following points are discussed with relation to the direct effects of external radiation. My interest centers upon the problem of civil defense in dealing with the radiation hazard in a contaminated area.

A. Areas of contamination

It is evident from the analyses such as Dr. Schafer presented to this committee that a nuclear attack upon the United States would involve an overlapping or smeared out pattern of bomb fallouts, especially over Northeastern United States. In making assessment of the radioactive power of bomb fallout, it is useful to introduce a new unit "the eternity roentgen square mile." This is a measure of the irradiating power of bomb fallout. By "eternity roentgen" I mean the total roentgens accumulated in dosage from 1 hour to eternity. This unit is then multiplied by square miles over which fallout occurs.

Example: To estimate the eternity roentgen square mile contamination from a 15 megaton explosion we proceed as follows. Assume that the ratio of fission to fusion energy release is 2:1. Then 10 megatons of fission energy will be involved. Assume a 50 percent local fallout. This yields 5 megatons of fission products in the fallout area. Simple calculation shows that 1 megaton of fission products could contaminate (if uniformly deposited at 1 hour) 1,000 square miles so that the 1 hour to infinity dose in open air would be 6,000 roentgens. Thus 5 mt. of fission products could raise this dosage to 30,000 r. Or if the area of fallout were greater, say, 5,000 square miles, the dosage would be 6,000 r.

An attack such as Dr. Schafer assumed involved 2,500 mt. of bomb yield and he specified "dirty weapons" surface burst. By "dirty" it is meant that the ratio of fission to fusion is fairly high. If we assume 2,000 mt. of fp (fission products) locally deposited this amounts to a total of 12 billion roentgen square miles of potential contamination. Obviously, this is a maximum since much fallout will occur after 1 hour; this will dissipate harmlessly in the air until it is deposited on the ground. Nontheless, the figure especially for surface burst bombs where local contamination will be maximized gives an indication of the magnitude of the fallout hazard. If any considerable fraction of this total figure is concentrated on a relatively small area, such as Northeastern United States industrial heartland, the corresponding radiation intensities will be severe.

Example: If a 1,000 mt. of fp concentrated upon Northeastern United States much of the region would be subject to a fallout of about 10,000 eternity roentgens. This would correspond to a fallout intensity of 2,000 r./hr. at 1 hr. I shall discuss the significance of such fallout in section C.

B. Clean and dirty bombs

The above discussion should make it obvious that the fallout from dirty weapons is of immense importance because of the area contaminable with a medium-weight attack. However, it may be useful to compare the damage areas of the two types of weapons.

A clean or relatively clean air burst bomb would have to depend upon blast and heat for its destructive effects. Consider, for example, the areas hit by the blast of a 20 mt. bomb.

Blast overpressure (pounds per square inch)	Distance in miles	Area in square miles
100	1.5	7
10	7.5	175
3	15.0	1,600

For purposes of comparison, one might select purely military targets such as air fields, missile sites and "hardened" targets which would require up to 100 pounds per square inch blast overpressure for destruction. Under such cases the aiming accuracy in delivery would have to be very great if you wished to "hit". A miss by as little as 2 miles with a clean bomb could be considered a complete miss. If one is concerned with population bombing and the criterion is the destruction of a framehouse, the 3 pounds per square inch blast would be appropriate. A greater aiming error would be allowable but one would not want to miss by more than 10 miles.

To complete the comparison, it is necessary to assess the persistence of the radioactive effect of fallout to discover whether such contamination would be effective in denying land to normal or to even emergency use.

I make this point, not to assert that there would be no military uses for clean bombs, but to emphasize that from the standpoint of civil defense, it might be very misleading to assume that an enemy would forego the use of dirty bombs.

C. Persistence of fallout

Witnesses before this committee have testified as to the rapid decay of fission products in fallout. It is true that a fresh mixture of bomb-produced fission products exhibits rapid decay. Half of the radioactivity, as measured from a time base at 1 hour, disappears in 32 hours. The AEC states in its report The Effects of High-Yield Nuclear Explosions (February 1955) "The main radioactivity of fallout decreases very rapidly with time—for the most part, within the first hours after the explosion."

Section 10.1 of the "Effects of Nuclear Weapons" (June 1957) states: "The radiation intensity decreases rapidly with time and except for areas of very high initial contamination, it ceases to be a serious hazard within a few weeks."

These statements, it seems to me, give the impression that civil defense has nothing to worry about after a few days, or a few weeks. I believe that the discussion in section A (part II) coupled with Dr. Schafer's estimates of the fallout intensities show that large areas of the United States could be contaminated to the extent of 2,000 roentgens per hour at 1 hour. The following schedule of roentgen dosages results:

From 1 hour [1] through end of 1st day	4,700 r.
From end of 1st day to end of 1st week	1,730 r.
From end of 1st week to end of 1st month	920 r.
From end of 1st month to end of 1st year	1,060 r.
From end of 1st year to 50 years [2]	840 r.

[1] The dosage of 4,700 r. depends upon fallout at 1 hour after detonation. Fallout at later times (i. e., farther downwind) would significantly reduce this first day dose.
[2] Terrain and weathering would play a significant role in reducing this dose.

If we look only at the decay rate, the decay seems rapid. Starting at 1 hour after the explosion, the rate would be 2,000 r./hr. At 7 hrs. it would decrease to 200 r./hr., at 1 day to 45 r./hr. At the end of 2 days it would be 20 r./hr. At

1 week it would be 4.2 r./hr. or 100 roentgens per day. At the end of 1 month it would be 17 r./day; this would decrease to 4 r./day at 100 days, and to 0.8 r./day at 1 year.

D. Significance of local fallout

I believe that the combination of the vast areas contaminable with high-yield thermonuclear weapons with the long term persistence of the fission products poses a problem for civil defense of great magnitude as different from that of the A-bomb as that was from the TNT bomb.

E. Genetic consequences of a 2,500 mt. attack upon United States

Assume, as in section II–A, that the United States is hit by a 2,500 megaton attack, in which some 2,000 mt. of fission products are deposited on the ground. As we have seen this can be expressed as a contamination equivalent to 12 billion roentgen square miles, where the roentgen as used here is the eternity roentgen. If we make the simplifying assumption that this contamination is spread uniformly over the continental United States, this will produce an eternity exposure of 4,000 roentgens (land area is 3 million square miles). In an actual situation this would be unevenly distributed but the probability is that exposures would be greatest nearest inhabited areas, so this calculation probably underestimates the effect.

Let us assume that people go into hiding and receive no significient radiation exposure in the first month after the attack. I grant that this is highly unlikely so the calculation is again underestimated. From 1 month to 30 years the exposure would average 20 percent of 4,000 roentgens or 800 r. Divide this in half to take account of weathering, so that we get 400 r. as the average exposure to every American who survives. We may now apply Dr. Crow's data of June 4 to this figure of 400 r. Instead of 2 billion children in the next generation we consider one-twentieth this figure. This means that we multiply Dr. Crow's values by 400 and divide by 0.1 (Dr. Crow's assumed 30-year exposure) times 20. Thus we multiply all his figures by a factor of 200. This yields:

Effect	1st generation	Total
a. Physical and mental defects	1,600,000	16,000,000
b. Stillbirths and childhood deaths	4,000,000	120,000,000
c. Embryonic and neonatal deaths	8,000,000	140,000,000
d. Intangible defects	(1)	-------------

1 A larger but unknown number.

In the first generation about 2 out of every 10 children would be genetically defective. The sum total of all deferred deaths from the attack would be 272 million or several times the number killed by the direct attack. In addition almost everyone would shoulder an increased genetic burden.

PART III. REMOTE FALLOUT

NOTE.—I believe that previous testimony and the roundtable discussions on the production, injection, transport, and fallout of radioactive debris from bomb explosions provide a more solid base for evaluating the hazard. It is obvious that in the area of the uptake of fission products there still remain some unknowns which future research and global survey will resolve. It is doubtful if the uncertainties inherent in estimating the biological effects of radiation will be resolved as readily. I wish to comment specifically upon several topics which I feel deserve amplification and emphasis.

A. The "present test rate"

This term has been used frequently before this committee as well as in the public domain during the past few years. Rarely has it been defined in a quantitative manner. To interpret the meaning of the present rate of testing one has to specify a precise number of megatons of fission products injected into the stratosphere.

I do not profess to have any inside information upon which to base an estimate of the present rate of testing. However, it is instructive to consider how the test rate has progressed since 1945. I am drawing upon data openly available and I apologize for the roughness of the data. Nonetheless, the progression of the annual increments to the curve may be worth considering.

The curve I shall plot covers the period from 1945 to 1957 with a projection to the future. Extrapolation of the curve will be considered. First, we plot the total fission yield of all bombs tested in each year. Up until 1952, and more generally until 1954, this total fission yield will correspond roughly to the total bomb yield. After that time corrections have been estimated for the fission fraction of the explosive yield. Second, we then estimate the fraction of the fission debris which is injected into the stratospheric reservoir and is globally dispersed. This fraction will depend upon bomb yield and firing conditions.

I claim no accuracy for the estimate and present the curve for qualitative illustration of the trend in the bomb test rate.

As long as weapons tests were confined to pure fission weapons of relatively low yield the global fallout of strontium 90 would be negligible. With higher yield fission weapons, more of the fission products began to be injected into the stratosphere and retained there for global distribution. However, because of the economy limits (cost of fissionable material) the global hazard was still quite small.

On November 1, 1952, the United States entered a new domain of weapons testing. The Soviet Union followed suit within a year. These tests were then followed by the Castle series of tests in the Pacific in the spring of 1954 when high-yield contaminating bombs were tested at Bikini. These tests added to the stratospheric reservoir the majority of the radiostrontium still present there. Dr. Langham of the Los Alamos Laboratory testified before this committee that one might assume an average rate of 10 megatons of fission products per year. This total of 50 Mt. for the past 5 years would check with the data that I have estimated.

In order to define the present test rate one has to wait until the end of the year, add up the Soviet, United States and United Kingdom contributions to the stratosphere and thus reach a reasonable figure. In the absence of international exchange of data, this is done through remote instrumentation.

B. A limit to bomb tests

Dr. Neuman in testifying before this committee arrived at a "safe" test rate of 2.2 megatons of stratospheric strontium 90 per year; i. e., the amount associated with the annual injection of strontium 90 produced in an explosion such that 2.2 megatons of fission energy inject their fission products into the stratosphere. Some time ago, I estimated on a similar line of reasoning that this "safe" limit would correspond to 3 megatons (plus or minus a factor of 3 times this value); i. e., as high as 9 or as low as 1 megaton per year. I believe this is in general agreement with the 2 to 10 megatons estimated by your roundtable discussion of May 29.

Superimposing these values upon the chart for the yearly test rates, it is seen that this "safe" limit was exceeded in 1954, 1956, and probably will be exceeded this year as well.

I believe the concept of a safe testing rate is of very great importance from a global health viewpoint. But the limit seems so low that it would appear that setting up a quota for each nation's annual testing would de doomed to the same fate as the attempt to control battleship construction in the 1920's.

But I believe that an internationally constituted monitoring system could keep systematic check on the level of fission products which fall out as a result of bomb tests. The publication of these measurements would have a profound effect on world opinion.

It would be of interest to learn from the Atomic Energy Commission the value for the safe annual limit which it assumed in its deliberations during the past 3 years.

C. A limit to war

There is obviously a great difference between the risks which a nation and its people take in time of war. During wartime the "safe" limit for the military application of thermonuclear weapons would be, at least in my opinion, 50 times greater than the peacetime safe limit. Whatever the value, the concept of a limit to the use of nuclear weapons in war is quite new. I have in mind here the inevitable lashback which an aggressor would suffer from the fallout of his own bombs. Dr. Libby, in his speech of January 19, 1956, estimated that between 330,000 and 440,000 megatons of bombs would have to be exploded before "the likelihood of untoward effects would be appreciable."

In view of the consequences to humanity, I wonder whether the Atomic Energy Commission or the Department of Defense has ever prepared a full study of the biological consequences of a nuclear war.

D. Evaluation of risk

Dr. Eisenbud's remark that he is not troubled by the hazard of giving milk containing traces of Sr–90 to his children illustrates, I believe, an extreme form in which a radiation risk may be assayed. The probability of injurious effect to a sample of three people is very small; from a personal viewpoint it is probably negligible. But if one applies the same probability of injury to 3 billion people, then even a very minute effect becomes significant to those whose lives are affected. Suppose we deal with a probability of one in a million. For a global population this would involve about 3,000 people. Such an effect would, from a personal viewpoint, constitute a very small risk. The same line of argument applies to Dr. Shields Warren's reference to the personal danger from the radiation emitted by the microgram of radium in his luminous wristwatch dial. The fact that he observes no visual change in the skin directly beneath the wristwatch proves nothing—certainly, it does not constitute a test of a radiation threshold.

If I may comment on the testimony of other expert witnesses who testified on Monday, June 3, I find Dr. H. L. Friedell's philosophy with regard to bomb tests rather whimsical. Apparently, Dr. Friedell believes we do not have enough data to evaluate the risk and we should proceed on a path of blissful optimism.

Testimony introduced on behalf of Dr. Jacob Furth by Dr. Shields Warren contains a recommendation that "the burden of decision rests not with biomedical investigations, but with military experts." In a matter so rooted in nuclear science and so veiled in conflicting opinions, I am reluctant to entrust the burden of the decision to military experts.

Dr. E. P. Cronkite cites three reports as authoritative in evaluation of strontium risks: (*a*) the United Nations report, (*b*) the British Medical Council report of June 1956, and (*c*) the National Academy of Sciences report of June 1956.

(*a*) So far as I know the United Nations report on strontium has not been concluded. I am informed it will not be published this year.

(*b*) With regard to the British report, I would call attention to the last sentence of that report. "Nevertheless, if the concentration in human bones showed signs of rising greatly beyond one-hundredth of that corresponding to the maximum permissible occupational level it would indicate the need for immediate consideration of the problem."

(*c*) With regard to the National Academy study, may I refer to page 60 of the general report, section 3: "However, if the testing programs of the several countries producing theromonuclear weapons were to intensify, strotospheric storage time may become a critical item in terms of the hazard to mankind."

Since the National Academy report was issued the United States, the U. S. S. R., and the United Kingdom have all tested theromonuclear weapons. I submit that the testing programs are intensifying.

E. Future nuclear tests

In assessing the future commitments of radioactive debris to the earth's atmosphere, we must deal with many unknowns. Had we attempted an estimate 5 years ago, prior to Castle-type weapons, we would have arrived at most misleading and optimistic projections. The end of weapon development is not in sight and no one can say that unexpected developments may not occur.

For example, may not smaller nations be stimulated by British success with thermonuclear type weapons and place maximum emphasis upon such development?

Additionally, can we be sure that a nation would restrain itself and not test a 100 megaton dirty weapon if military requirements and nuclear technology indicated that such a weapon was desirable?

Will the requirements of adapting maximum megatonnage to a small warhead put emphasis on further development of dirty type weapons?

We cannot answer these questions at this time, but we do know that a single weapons test of very high fission yield can add a strontium burden to the atmosphere far beyond the limits we have been discussing.

The United States has contributed the largest fraction of radiostrontium to the stratosphere and I think that it is most encouraging that the fullest discussion of the strontium fallout should occur in this country. I am not aware of any large body of published information on this subject of Soviet origin. It is known, however, that the Soviets are engaged in strontium studies.

In concluding this section, I would like again to stress Dr. Libby's contributions to this subject. They are of very great value and I feel sure that we would be in a much poorer position today to evaluate the strontium problem were it

not for Dr. Libby's personal interest in this field of investigation and the research which he has promoted so vigorously.

F. The strontium problem

It is clear from testimony given to this committee that data on the fallout of radiostrontium are becoming more firm as research results come in. I think that scientists can agree on the pattern of strontium fallout around the world. We are in a poorer position in our knowledge of strontium uptake in the biosphere. I am disturbed by fluctuations which have occurred in AEC statements on discrimination factors in the uptake of strontium into the food chain. Discrimination factors are high in the milk link of the food chain, but are much lower in foods consumed directly by humans. A discrimination factor of 7 is estimated by Dr. N. S. MacDonald of UCLA and Dr. W. Neuman estimates an average discrimination factor of 8.

Strontium 90 determinations in man must be expanded to assess the increase in strontium 90 burden with time. Careful determinations of natural strontium in humans deserve increased attention. We know that more strontium 90 will accumulate in humans as a result of bombs tested in the past and as a result of current tests. The determination as to how much of this radioelement may be tolerated safely is a matter for the biologists to discuss. This committee has heard a fairly wide range of opinion from its expert witnesses on the probable biological effects of Sr–90 levels in man. But it seems to me that even in this area some agreement was reached, especially when Dr. Shields Warren stated on June 3:

"I would be reluctant to see the average strontium 90 content of bones, particularly in children, go much above 10 times the present level."

Dr. Libby's speeches show that Sr–90 fallout will continue and the strontium 90 level in human bones will increase.

Unless restraints are imposed upon commitments of fission products to the atmosphere, it is only a matter of time before the strontium 90 level of Dr. Warren is reached.

PART IV. CONSTRUCTIVE PROPOSALS

A. Atomic Energy Commission information policy

I suggest that this committee or its parent committee may wish to review the information policy of the AEC with regard to nuclear weapon effects, with a view toward revising this policy so that information may be made available more promptly and completely. I believe that the national interest demands a much better relation between the press and the Atomic Energy Commission.

B. Report on the probable biological consequences of a nuclear war

I suggest that the Joint Committee on Atomic Energy might wish to recommend or sponsor the preparation of an analysis of the probable biological effect of nuclear warfare. It would be useful to investigate probable lashback effects from various levels of nuclear bombardment.

C. Data useful to civil defense

I believe that the committee's investigations have produced information of critical value to civil defense planning. It might be useful to have a summary report of these data transmitted to the Federal Civil Defense Administration. I have not seen many representatives of the Federal Civil Defense Administration at these hearings.

D. Research in long-range estimation of nuclear explosives

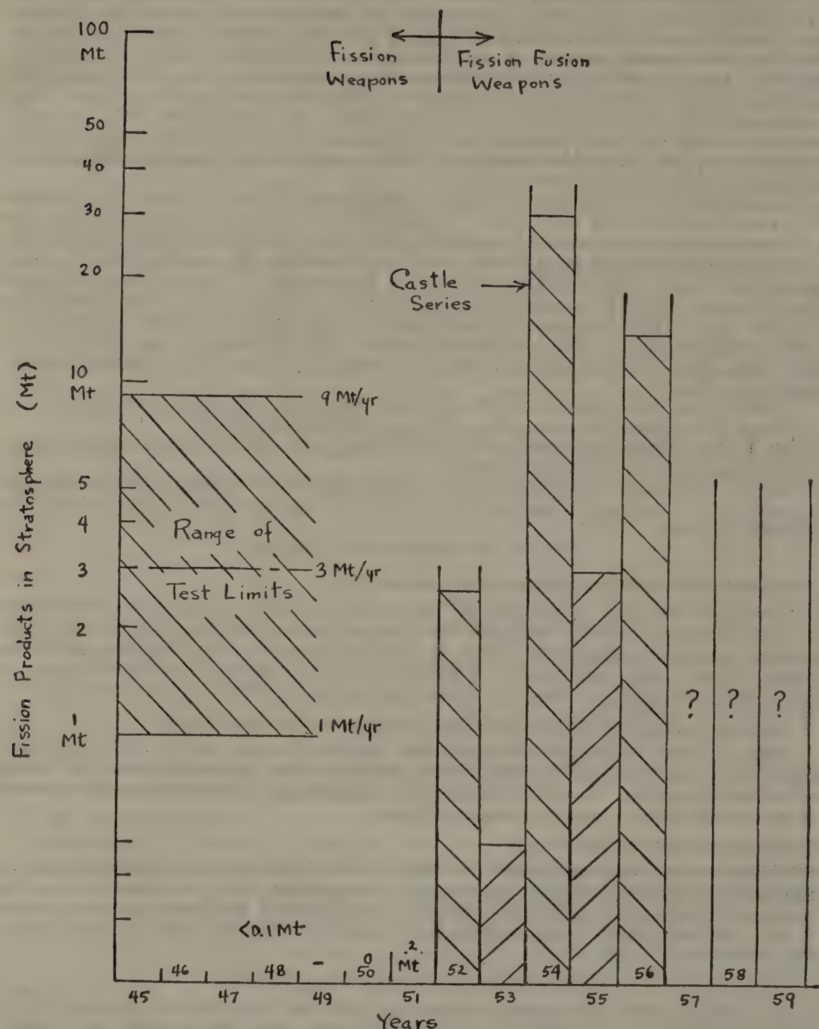
It is known that considerable effort has focused on long-range detection of nuclear detonations. Attention should be given to the declassification of such data as would bear upon evaluation of the radiostrontium problem. Other data would be most useful in discussion of the feasibility of policing an agreed upon test limit.

E. An annual fallout report

In view of the great public concern over fallout hazards, I would urge that the Atomic Energy Commission be required to issue an annual report on the degree of fallout and its uptake in biological systems. Perhaps such a report should be prepared by a university task force.

F. National radiation control

I would urge that the Congress continue its investigations of radiation hazards, extending them into the broader area of peacetime uses of radiation. I believe that the ever-increasing uses of radiation must be subject to legislative controls. Radiation protection in the United States needs uniform legal status.



Rough Estimate of Annual Additions to Stratosphere From Nuclear Weapons Tests of U.S.A And U.S.S.R [in Megatons of Fission Products]

Senator ANDERSON. Dr. Selove, we are happy to have you here today, proceed please.

STATEMENT OF DR. WALTER SELOVE, DEPARTMENT OF PHYSICS, UNIVERSITY OF PENNSYLVANIA [1]

Dr. SELOVE. I would like to thank the Joint Committee for inviting me to present testimony here. I would like to put before you insofar as time is available the results of some independent studies of the radiation hazards problem made primarily by the radiation hazards committee of the Federation of American Scientists.

The federation, as I believe you probably know, consists of scientists who are concerned with the interrelation between science and public affairs, and at the request of a number of members of the federation, a few of the members took on themselves the task of trying to gather such information as was available on the extent of the radiation hazards.

I have been a member of the radiation hazards committee for a year and a half or so and was recently elected chairman of that committee.

Representative COLE. What is the radiation hazards committee to which you refer, Dr. Selove?

Dr. SELOVE. What is it?

Representative COLE. Yes.

Dr. SELOVE. In the second paragraph of the prepared statement here you will see that at present the radiation hazards committee consists of several nuclear physicists, a biophysicist, a biochemist, a chemist, and a cancer research worker.

In the studies which this group has made of the radiation hazards problem we have also consulted with geneticists, and we have consulted with members of the Atomic Energy Commission, who I should say have been most cordial and helpful.

Representative COLE. What I would like to know, Dr. Selove, who is the sponsoring organization—of what organization is this radiation hazards committee of which you are a member?

Dr. SELOVE. This is a radiation hazards committee concerned with possible hazards from radiation sources of all kinds. It is one of the committees of the Federation of American Scientists which is a volunteer organization of scientists—of some of those scientists who are concerned with the impact and interrelations between science and public affairs.

One of the principal objectives of this statement is to emphasize that, although there is no important disagreement among scientists on the magnitude of the average dose of radiation due to fallout, conclusions which may appear to be in conflict with each other can be drawn with equal accuracy from the available facts.

I would like to quote from a report just prepared by the FAS radiation hazards committee which will illustrate this.

---

[1] Date and place of birth: 1921, Chicago, Ill. Education: Bachelor of science degree, physics, University of Chicago, 1942; doctor of philosophy degree, physics, University of Chicago, 1949. Work history: Assistant instructor, University of Chicago, 1942–43; staff member, M. I. T. Radiation Laboratory (radar), 1943–45; National Research Council predoctoral fellow, 1946–47; physicist, Argonne National Laboratory, 1947–49; instructor, assistant professor, Harvard University, 1950–56; on leave, University of California radiation laboratory, Livermore, 1953–54; member, editorial board, Review of Scientific Instruments, 1955–; National Science Foundation senior postdoctoral fellow, 1956–57; associate professor of physics, University of Pennsylvania, 1956–. (Submitted by witness.)

The committee study of the available scientific facts has led to two conclusions:

First: The added radiation hazard from continued nuclear weapons testing at the present rate is no greater than that from other radiation normally encountered. The radiation from testing will approach a level equivalent to the natural background radiation to which the human race has always been exposed. Similar radiation doses are obtained from annual chest and dental X-rays.

Second: This small added radiation, from whatever source, will cause many deaths.

The committee believes that both conclusions are scientifically correct, and in no way contradict each other.

Unfortunately, those who believe that we should continue testing in order to maintain a lead in nuclear weapons often emphasize the first conclusion and ignore the second. Similarly, those who believe that a test ban is desirable, since it may lessen international tension, often emphasize the second and ignore the first. The committee believes that both statements must be taken together since either alone is misleading.

Rather than try to cover fallout problems exhaustively I would like to restrict my remarks to just a few aspects of the problem, primarily concerned with the global and long-lasting effect of fallout and not taking up the question of close-in fallout effects.

One of the most important aspects of the problem I would like to emphasize is the uncertainties that remain as to just what the effects will be and how large the effects will be. That is, how many individuals will be affected.

There simply has not been sufficient time to learn what all the effects of fallout radiation may be. It is likely to be several decades before we know with much certainty. At present, however, it appears likely that the two principal effects of fallout will be the genetic effects from general gamma radiation and the production of leukemia and bone cancer by strontium 90. With regard to these effects, that is, the somatic effects, there is considerable disagreement as to whether small doses such as are involved in widespread fallout from tests will produce any effect at all. A considerable body of scientific opinion holds that it is very likely that even small doses will produce proportional effects and believes that there is some support for this conclusion both from experimental data and from theory.

Other scientists, as you have heard, are of the contrary opinion and believe there is probably a threshold for somatic effects.

All are agreed, however, that the true behavior cannot be determined from presently available data. I believe it is fair to say that on the basis of the data the pessimistic interpretation seems at least as reasonable as the optimistic interpretation.

In this connection, it should perhaps be emphasized that even if one takes the pessimistic interpretation; namely, that there is no threshold——

Senator ANDERSON. Are you reading from your prepared text?

Dr. SELOVE. No. I have some other statements here.

In connection with this question of pessimistic versus optimistic interpretation, I think it is worth emphasizing that even if one takes the pessimistic approach and assumes that production of leukemia and bone cancer are proportional to even small doses, it is still true that the probability that any given individual would be affected by the levels of radiation received from fallout from tests so far, is very small indeed.

For example, I have made a rough calculation for the type of effect to be expected from the radiation from a wristwatch. I come out with

the following number, regarding which I can go into more detail for you if you wish. The dose of radiation to the wrist from a wristwatch, according to the theory that even small doses may produce bone cancer, would have a certain probability of producing bone cancer, primarily a probability of producing it in the wrist, since the radiation from a wristwatch is concentrated in that area. I have calculated the effects of the dose from a typical wristwatch according to measurements which have been made of radiation from wristwatches. Suppose one had a million individuals not wearing wristwatches, on the one hand and a million individuals wearing wristwatches on the other. Now one would expect that among those not wearing wristwatches some 1,000 out of a million would eventually die, when they die, with bone cancer. This is a number of 1 in about 1,000 of all deaths normally occurring which is due to bone cancer.

Representative COLE. By that you mean that the cause of the death would have been the bone cancer?

Dr. SELOVE. That is correct. Those are statistics for the United States. Two thousand people in this country die every year from bone cancer. Then of the million who do not wear wristwatches, 1,000 would die of bone cancer.

Among those who wear wristwatches I calculate that 1,003 would die of bone cancer.

There would be a certain small effect. It is obviously such a small increase over the normally existing bone cancer and so small an effect on an absolute basis that obviously one individual has absolutely no worry as far as he himself is concerned as to the magnitude of this effect.

As has been emphasized before you by many witnesses, when one takes a very small percentage increase in some normally occurring effect and applies it to the very large population of the world then a considerable number of individuals may still be affected. It might be worthwhile giving a second example for the case which Mr. Holifield raised the other day, as to whether a mother should be concerned about letting her children drink milk which has the amount of strontium 90 in it that has been found to exist at present.

I calculate also on the basis of further numbers that I may present if you wish, and if time is available, that if you have again a number of children or adults drinking milk which has no strontium 90 in it and another group drinking milk which has the amount of strontium 90 currently present, then among those who drink milk with no strontium 90, 1 in 150 of them when they die will die of leukemia. This is a number based on statistics for the United States. It may be surprising to you that every year in the United States 10,000 people die of leukemia among a million and a half deaths. This is 1 in 150.

I calculate that if one drinks milk which contains the present amount of strontium 90 and if one uses the assumption that the effects of strontium 90 even in small doses are that proportionate amount of bone cancer and leukemia will be produced, then the probability that an individual will develop leukemia will be increased from 1 in 150 to 149.

Obviously from the standpoint of an individual, this is an extremely small number. This represents about a 1 percent increase in the probability that one would develop leukemia. The 1 percent increase translated into numbers which apply to a large part of the world popula-

tion, for example a country of the size of Japan means the following: If again we take for Japan—I do not have statistics available for this but I think we can use probably reasonably accurate numbers approximately the same as those in the United States—a number of deaths of leukemia per year which is probably 5,000 or 10,000 and if we have a 1 percent increase in that effect—and a 1 percent increase is the amount that one calculates from the kind of data presented to you by the expert witnesses in these fields—it means that in Japan as a result of the tests so far one may have each year at the present time 10,100 deaths from leukemia instead of the 10,000 which would normally occur.

Again this 100 additional deaths in one country—one should perhaps multiply this by some larger factor if one wants to consider the world as a whole—is a very small percentage compared to the million or so people who die in Japan every year, and even compared to the 10,000 or so who die of leukemia every year in Japan.

It is one purpose of this statement to gather together these numbers which have been presented to you by the various witnesses here and to show that depending on which aspect of the data one emphasizes one gets a result which has quite a different appearance, from the result which appears when the data is presented in another way.

To summarize this point, then, it has been estimated that some 10 percent of normal leukemia cases are due to natural background radiation, and a similar figure may also hold for bone cancer. The fallout radiation from past tests represents only a small percentage increase over natural radiation and consequently the effects of fallout radiation represent only a small percentage of increase over normal effects.

For example, even without fallout some 10 million people would develop leukemia or bone cancer over the next 30 years. This is an estimate based on the United States statistics. Because this number is so large, even the small percentage increase in radiation resulting from tests still can affect a considerable number of people.

Senator BRICKER. What percentage of those bone cancers are caused by natural background radiation?

Dr. SELOVE. There is not sufficient data to draw a conclusion. The conclusion has been reached by Dr. Lewis who testified before you that in the case of leukemia some 10 percent of the leukemia cases are very likely due to natural background radiation. Leukemia is a cancerlike disease of the blood. If the theory as to how cancer develops held by many geneticists, namely, that it is a mutationlike process, is correct, for one cancerlike disease leukemia, probably the same type of effect would hold for bone cancer.

Senator BRICKER. Is that generally accepted?

Dr. SELOVE. I am sure it is not widely accepted. These matters are just coming to be thought about.

Senator BRICKER. There may be other causes of bone cancer.

Dr. SELOVE. Even according to Dr. Lewis' testimony only 10 percent of leukemia would be due to radiation.

Senator BRICKER. Your statement is based on the premise that they are all caused from radiation?

Dr. SELOVE. No. I should say in this connection that the statistics for the United States are that each year some 10,000 individuals die of leukemia, and 2,000 from bone cancer. If we assume that approxi-

mately the same percentage of bone cancers are due to radiation as one would assume from Dr. Lewis' work is the case for leukemia, then we would have a very small number of bone cancer cases due to radiation as compared to the number of leukemia cases due to radiation.

If one lumps together leukemia and bone cancer it will not make much difference how accurate our estimate is of how much bone cancer is caused by natural radiation.

I would like to note that these figures I have presented are consistent with the recent estimate made by the radiation hazards committee of the British Atomic Scientists Association. I do not have the professional affiliation of the various members of that radiation hazards committee but I recognize one name on the list, that of Dr. Alexander Haddow, who is the director of a cancer research institute in Britain. That committee made an estimate, subject to the same assumption we have been discussing, that even small radiation doses produce proportional amounts of bone cancer. They estimated that some 50,000 cases of bone cancer might be expected to develop as a result of nuclear tests already carried out. This is essentially the same type of number I presented.

You have heard estimates of larger numbers of individuals affected. This number I believe represents a sort of median value. These numbers, it should be emphasized are extremely crude and approximate. There is simply not enough data to obtain very accurate numbers. The true effects might be easily 10 times larger than the numbers I am presenting. They might also be easily 10 times smaller. It is the very uncertainty that needs the strongest consideration.

A great deal of apparent disagreement on the dangers of fallout has been due to a difference in emphasis. The AEC has emphasized that the radiation from strontium 90 from tests so far will represent only a few percent increase from the natural background radiation, on the average. The AEC has further emphasized that this average increase in radiation due to strontium 90 is small compared to the additional radiation exposure many people receive simply as a result of living with a higher background radiation level than average or X-rays. Relative to other sources of radiation it is perfectly true that fallout radiation contributes at the present level of testing only a small additional increment. On the other hand it can be stated that even a small percentage increase over the natural background radiation is likely to harm a considerable number of individuals.

The likelihood that even a small percentage increase over background radiation will cause many deaths applies to radiation from other sources as well as to fallout radiation. It should be remarked that the very rapidly increasing awareness of the effects of radiation is already leading to greatly improved X-ray techniques.

Next is the effect of fluctuations. I think this affects the impact of the fallout problem on the people of the world.

The fact that fallout is not distributed with absolute uniformity among all the people of the world affects the evaluation of the fallout hazard in two important ways. First, as has been emphasized by many people, and particularly by Doctor William Neuman, if a "maximum permissible" level of fallout material, say of strontium 90, is agreed upon, then the world average level should not be allowed to go beyond a fraction of it, or else an appreciable part of the world pop-

ulation would be subjected to more than the "maximum permissible" level.

Second, the detectability of fallout effects, which to a considerable extent determines the psychological impact of the effects, will be increased as a result of variations in the amount of fallout, in the diet, and in the nature of the soil. Some 50,000 individuals may develop leukemia and bone cancer from strontium 90 due to tests so far. If these 50,000 were fairly uniformly spread over the world, they would probably be undetectable among the 3 million or so "normal" cases of bone cancer and the 10 million or so "normal" cases of leukemia which would be present even if there had been no fallout. But in actual fact some world areas will suffer more heavily than others, due to diet and soil nature, and due to the nonuniform distribution of fallout. Although the world average increase in leukemia or bone cancer, for 50,000 total cases due to fallout, would be only 1 in about 60 normally occurring cases of bone cancer, or 1 in about 500 normally occurring cases of leukemia, the relative increase in some areas might be as high as about 1 case for every normally occurring case of bone cancer. Even such a 100 percent effect, from tests so far, might be hard to detect, because the normal incidence of bone cancer is so low, and the number of people subjected to extreme levels is probably very limited.

Is there a danger from test fallout?

The two principal effects of fallout appear to be the genetic effects from general gamma radiation, and the production of leukemia and bone cancer by strontium 90. Scientists are agreed that genetic effects are produced even by small amounts of radiation. With regard to somatic effects, such as cancer, a considerable body of scientific opinion holds, with some support from experimental data and with some basis in theory, that these, also, can be produced even by small doses of radiation. On the assumption that this is true, it can be estimated that the number of individuals likely to be directly harmed in the coming generation by strontium 90 will probably be greater than the number showing genetic effects in that generation—although, for a given amount of fallout, the genetic effects will persist for many generations and will eventually cause direct injury to a total number of persons comparable to that affected by strontium 90. In terms of the immediate impact, therefore, we can concentrate our attention on strontium 90.

In regard to the genetic effect of fallout, Warren Weaver, vice president of the Rockefeller Foundation, and Chairman of the National Academy of Sciences Committee on the Genetic Effects of Atomic Radiation, estimated before a Senate subcommittee on January 16 that radioactive fallout from nuclear weapons testing to date will account for some 6,000 of the 30 million "handicapped" babies to be born in the coming generation. In regard to the effects of strontium 90, estimates quoted previously indicate that some 50,000 individuals over the next 30 years may develop leukemia or bone cancer as a result of tests to date.

How much should one be concerned about fallout effects of this magnitude? This is not a question which can be answered on scientific grounds. Even if one accepts the interpretation of the data presented above as a basis for estimating the number of individuals who will show genetic or somatic effects as a result of the 50 megatons—mil-

lions of tons of TNT equivalent—fission yield of tests so far, there are a number of other factors that must be carefully weighed before final judgment can be reached on the significance of the hazard. Probably no absolute evaluation will be possible, but instead, the evaluation will have to be made relative to the problem of securing peace in the world and in relation to the moral problems involved.

A discussion of some of these questions would take us out of the area at which this statement is directed, but it does seem desirable to emphasize that three of the pertinent factors which must be considered are:

(1) The uncertainties as to the number of individuals affected. The number may be smaller than 50,000, or larger. And the remaining uncertainties as to what all the effects of fallout radiation will be;

(2) The size of the number, 50,000, relative to the numbers of people harmed by other reducible effects; and

(3) The fact that the fallout effects are global, involving citizens primarily of countries other than the testing countries.

The global effects of fallout—as opposed to the more local effects—come almost exclusively from explosions of large nuclear weapons; a single one of these can produce as much worldwide fallout as a thousand bombs of the size used at Hiroshima. Those who consider the further development of large nuclear weapons necessary for military security view the probable effects of fallout as a small addition to other hazards of day-to-day living. Those who consider that the further development of large nuclear weapons moves the world away from peace rather than toward it of course do not feel that the military desirability of such weapons justifies the probable fallout effects of tests. Finally, there is a third group who, while not certain whether the further development of large weapons is likely to be useful, feel that in view of the uncertainties as to the nature and magnitude of fallout effects, and in view of the international political impact that testing of large nuclear weapons produces in the involuntarily exposed majority of the world, the nuclear testing powers would be well advised to exercise strong restraint with regard to tests producing further significant amounts of fallout.

As new nations enter the nuclear testing program, it can be expected that they will be interested in testing bomb types which produce a great deal of fallout. There are two dominant reasons for this:

First, about the most economical way possible to increase the yield of a large bomb is to use an outer shell of natural uranium. This leads to an inexpensive large energy release, but also to a large release of fission products—the worst kind of fallout.

Second, a large amount of fallout increases the devastating power of a nuclear bomb. The addition of a shell of natural uranium to a large thermonuclear bomb can increase the devastating fallout to a very much greater degree, for example, than the addition of cobalt to make a cobalt bomb, and moreover can at the same time increase the energy release by a large amount, which a cobalt shell will not do.

Although the United States appears to have turned its efforts to designing nuclear weapons which produce less fallout the U. S. S. R. does not appear to have followed suit as yet. While the United States Pacific tests of last spring did not add very appreciably to the stron-

tium 90 in this country, the first four Russian test explosions of last fall resulted in an increase of about one-third in the strontium 90 concentration in United States soil, within a period of a few weeks. Since that time, the U. S. S. R. has set off at least eight additional nuclear explosions—as of May 15. No information of any precision has as yet been released on the size of these explosions and on the amount of radioactive fallout resulting from them.

The amount of testing which can be tolerated.

I would like to ask Senator Anderson what sort of time scale you would like to proceed on. I can cut this short.

Senator ANDERSON. I have been trying to find out whether we should go ahead with the seminar we had scheduled. If it is agreeable for the other members and participants I would like to suggest that we do not go ahead with it. After all, 6 hours of hearing in 1 day is quite a little bit. If we are not going ahead with it, then I would say if you could conclude in the next 5 or 10 minutes, then there might be questions and we can conclude this afternoon.

Representative COLE. Mr. Chairman, I can only say I exhausted my absorptive powers an hour or so ago.

Senator ANDERSON. I would like to say to the participants that it is very difficult to those of us who are not scientists to try to keep up with papers that are presented and not do what Congressman Cole has just suggested. He has been a faithful attender of these hearings and trying his best to absorb it. It is unjust to him and to the participants.

If there is no vigorous and violent exception I will announce now that the seminar will not be held at the conclusion of this, but we hope to start off with it in a preliminary fashion tomorrow. Maybe it will give us a chance to therefore develop it.

I would ask the witness if he does not mind to try to conclude in 10 minutes.

Is there any objection? If not, you may proceed.

As you know, I am very much interested in the Federation of American Scientists and devoted to their program.

Dr. SELOVE. I would like to introduce in the record at the end of this testimony a copy of the report which the Radiation Hazards Committee has prepared, and if there are any questions we can discuss it further tomorrow.

I think it is useful to put the fallout problem in perspective through a look at some of the numbers of individuals affiected by fallout problems as compared to those affected by other related effects and some of these numbers are written out in this report.

(The report above referred to together with two statements by Dr. Selove follow:)

WORLDWIDE FALLOUT FROM H-BOMB TESTING—ALARMING OR NEGLIGIBLE?

Written by the Radiation Hazards Committee of the Federation of American Scientists

For almost a year highly qualified scientists have been making apparently conflicting statements on the hazards of fallout from continued nuclear weapons testing. Because of the existence of such conflicting statements, the radiation hazards committee has felt it desirable to make an independent and objective study of this important issue.

After 6 months of study and discussion the committee has reached complete agreement on the scientific facts of worldwide fallout and its hazard, and on the scientific conclusions that can be drawn from these facts.

The members are also agreed that arguments for or against the banning of nuclear weapons tests must be based primarily on moral grounds and on considerations of international affairs, and not purely on a scientific evaluation of the radiation hazards.

The committee study of the available scientific facts has led to two conclusions:

First: The added radiation hazard from continued nuclear weapons testing, at the present rate, is no greater than that from other radiations normally encountered. The radiation from testing will approach a level equivalent to the natural background radiation to which the human race has always been exposed. Similar radiation doses are obtained from annual chest and dental X-rays.

Second: This small added radiation, from whatever source, will cause many deaths.

The committee believes that both conclusions are scientifically correct, and in no way contradict each other.

Unfortunately, those who believe that we should continue testing in order to maintain a lead in nuclear weapons often emphasize the first conclusion and ignore the second. Similarly those who believe that a test ban is desirable, since it may lessen international tension, often emphasize the second and ignore the first. The committee believes that both statements must be taken together, since either alone is misleading.

How is it that both statements are correct? Why are they not contradictory? Only by an examination of the facts can these questions be answered.

1. OUR NORMAL RADIATION BACKGROUND

One must first recognize that the human race has always been subjected to a continuing radiation dose from natural, unavoidable causes such as cosmic radiation from outer space and natural radioactivity in earth and rocks. For comparative purposes we have taken as standard the normal, unavoidable, radiation experienced by a person living in a frame house in Germantown, Pa.

The figures below show that a person living in a stone house in Denver experiences a background radiation 35 percent higher than the Germantown resident. Other similar variations occur, owing to differences in the radium content of the drinking water, the wearing of a luminous dial wristwatch, etc. Many of us experience even larger doses from annual dental or chest X-rays.

How will the radiation dose from continued H-bomb testing compare with these "normal" radiations?

	Added radiation dose above the natural background dose received by a Germantown resident living in a frame house (percent)
"Natural" causes:	
Living in a brick or stone house	20
Living at 5,000 feet altitude (Denver, Colo.)	15
Total additional background for a person living in a stone house in Denver	35
Other causes:	
Wearing a luminous dial wristwatch	20
Having an annual chest X-ray	From 10 to 200

2. ADDED RADIATION FROM NUCLEAR WEAPONS TESTING

The radiation hazards from weapons testing that are the most serious are first the hazard to individuals due to the accumulation of radioactive strontium in the bone, and second the genetic hazard to future generations due to increased numbers of harmful mutations produced by the general rise in the external radiation background.

Considering first the bone radiation from strontium, it has been verified that radioactive strontium from weapons already tested is now accumulating on the ground. Since strontium is chemically similar to calcium, it is being taken up by plants and through our food chain is passing into our bodies and is being stored in our bones. Here it emits radiations, causing an increase in our bone dose over that from natural causes.

	Percent
Present (1955) average annual dose to human bone from strontium from past tests	0.2
Average annual strontium bone dose in 1970 if no more weapons are exployed	5
Average annual strontium bone dose by the year 2000 if tests continue	35

If weapons testing continues at the present rate, our children and grandchildren will on the average receive a bone dose of radiation 35 percent again as much as that received from natural causes.

Turning next to the radiation that may be genetically harmful to future generations, it is mainly the increase in external radiations that must be considered rather than internal radiations such as bone strontium. If nuclear weapons testing continues at the present rate the genetic dose will rise by 2 percent of the normal background dose. On the average we receive a 60 percent genetic dose from annual medical and dental diagnostic X-rays.

What will be the effect of fallout radiation doses as small as a bone dose 35 percent of natural background, or a genetic dose 2 percent of natural background?

Cause:	*Added radiation dose—Above the natural background dose received by a Germantown resident living in a frame house (percent)*
Average added genetic dose if tests continue at the present rate	2
Present average added genetic dose per year per person in the United States from X-rays	60

8. THE EFFECTS OF RADIATIONS

(a) The strontium hazard

Radiations in large doses are definitely known to produce leukemia and bone cancer, and at high dose levels the incidence is proportional to the dose. But what is the effect of small doses? For leukemia, evidence has been obtained that indicates that the incidence is directly proportional to the radiation received, and that about one-tenth of all present cases are due to normal background radiation. It is reasonable to assume that the same is also true for bone cancer.

While these interpretations are not yet rigidly proved, they are reasonable conclusions from the data and we can use them as the basis for estimating the extent of radiation effects.

There are 12,000 deaths annually in the United States from leukemia and bone cancer. If one-tenth of these cases are due to the natural radiation background, then an added radiation dose 35 percent of natural background, whether from continued H-bomb testing or from annual chest X-rays for all people, would be estimated to produce 400 additional deaths from bone cancer and leukemia in the United States per year. Over one generation of 30 years this figure would total 12,000 deaths. Many more deaths will be produced outside the United States by continued testing.

Deaths per year per 5 million people in the United States. (Approximately the population of greater Philadelphia)

Deaths per year from all causes	[1] 50,000
Deaths per year from motor accidents	1,100
Deaths per year from bone cancer and leukemia	380
Estimated present deaths per year from bone cancer and leukemia caused by natural background radiation	38
Estimated added deaths per year from bone cancer and leukemia for those living in stone houses at 5,000 feet altitude	13
Estimated aded deaths per year from bone cancer and leukemia from bone cancer and leukemia by 2000 A. D. due to continued testing	13
Estimated added bone cancer and leukemia deaths per year in 1970 from weapons already exploded	[2] 2

[1] Per 5 million people.
[2] Per 5 million people.

The above figures are for the United States only. They are probably also correst for the rest of the western world, but may be incorrect for those parts of the world where the death rate from other causes is much higher than in the western world.

(b) The genetic hazard

Mutations, or changes in the hereditary material passed on from parent to offspring, are known to be produced by radiation as well as by chemical and other causes, many unknown.

Geneticists are agreed that any increase in the mutation rate is bad, and they are also agreed that any added radiation dose will increase the mutation rate; the larger the dose the greater the number of mutations produced.

By how much does the mutuation rate in man increase, for an added dose of 2 percent of the natural background (from continued testing)? Or for an added dose of 60 percent of the natural background (the average X-ray dose per person under 30 in the United States)?

The absolute answers to these questions are not known. We can say, though, that in the United States the increasing use of X-rays and fluoroscopy for medical and dental diagnosis is a very much greater genetic hazard than is the increased radition from nuclear-weapons testing.

4. NUCLEAR WAR

Both those who favor continued H-bomb testing and those who favor a test ban are agreed that a major nuclear war would be a world catastrophe from which civilization might not rise again. They only disagree on the best method of averting such a war. Apart from the death, injury and destruction caused at the time, the radiation background would be raised many times above that from continued testing, and would result in further hundreds of millions of deaths in the years following the war.

CONCLUSIONS

While the scientific evidence discussed above is admittedly not complete, it is complete enough to allow reasonable conclusions to be made.

First, the radiation hazard from continued nuclear-weapons testing at the present rate is no greater than that from other radiations normally encountered.

Second, even this small added radiation will cause many deaths.

Third, even more lives are endangered by other radiation hazards such as from X-ray examinations. In the case of X-ray examinations the advantages are usually important enough to outweigh the disadvantages. The medical profession is alert to this problem and is taking active measures to reduce the exposure to a minimum.

Will continued testing lead to a hazard that is alarming?

If one believes that even one death from fallout is too many, and that no nation should subject other peoples to the effects of fallout without their consent, then the answer to the question is "Yes."

If, on the other hand, one believes that the security of the country depends on continued testing, then the answer to the question is, "No; it is a small price to pay for security."

Each person, in trying to answer these questions for himself, must make personal judgments based on the moral problems involved and on the problem of maintaining peace.

THE RADIATION HAZARDS COMMITTEE OF THE FEDERATION OF AMERICAN SCIENTISTS

Dr. Walter Selove, associate professor of physics, University of Pennsylvania, Philadelphia (chairman).

Dr. Richard L. Burling, lecturer in physics, University of Pennsylvania, Philadelphia.

Dr. Stanley C. Glauser, biophysical chemist, postdoctoral research fellow, University of Pennsylvania, Philadelphia.

Dr. Donald L. Glusker, research chemist, Rohm & Haas Co., Philadelphia.

Dr. Norman Goldberg, assistant professor of physics, University of Pennsylvania, Philadelphia.

Dr. Philip Grant, research associate, Institute for Cancer Research, Fox Chase, Philadelphia.

Dr. Rosalie C. Hoyt, biophysicist, associate professor of physics, Bryn Mawr College, Bryn Mawr, Pa.

Dr. John R. Pruett, nuclear physicist, associate professor of physics, Bryn Mawr College, Bryn Mawr, Pa.

SUMMARY OF REMARKS ON RADIOACTIVE FALLOUT PRESENTED BY WALTER SELOVE TO THE JOINT COMMITTEE ON ATOMIC ENERGY AT ITS OPEN HEARINGS

Although there is no important disagreement among scientists on the magnitude of the average dose of radiation due to fallout, conclusions apparently in conflict with each other can be drawn with equal accuracy from the available facts. As stated in a report just prepared by the radiation hazards committee of the Federation of American Scientists, the available scientific facts lead to two conclusions:

"First, the added radiation hazard from continued nuclear weapons testing at the present rate is no greater than that from other radiation normally encountered. The radiation from testing will approach a level equivalent to the natural background radiation to which the human race has always been exposed. Similar radiation doses are obtained from annual chest and dental X-rays.

"Second, this small, added radiation, from whatever source, will cause many deaths.

"The committee believes that both conclusions are scientifically correct, and in no way contradict each other.

"Unfortunately, those who believe that we should continue testing in order to maintain a lead in nuclear weapons often emphasize the first conclusion and ignore the second. Similarly, those who believe that a test ban is desirable, since it may lessen international tension, often emphasize the second and ignore the first. The committee believes that both statements must be taken together, since either alone is misleading."

There has not been sufficient time to learn what all the effects of fallout radiation may be. It is likely to be 10 or 20 years or more before we know with much certainty. At present, however, it appears likely that the two principal effects of fallout will be the genetic effects from general gamma radiation, and the production of leukemia and bone cancer by strontium 90.

Rough estimates can be made of the number of individuals likely to show these effects. The estimates are that, from tests through 1956, some 6,000 individuals throughout the world will show serious genetic effects in the next 30 years, and probably also some 50,000 will develop leukemia or bone cancer. (The genetic effects of a given amount of radiation will persist for many generations, and the total number of individuals ultimately affected genetically, from a given amount of fallout, will be comparable to the total number affected by strontium 90.) These estimates can only be made approximately—the correct numbers may be several times larger or several times smaller.

The fallout radiation from past tests represents only a small percentage increase over natural radiation (cosmic rays, natural radioactivity), and consequently the effects represent only a small percentage increase over "normal" effects. For example, even without fallout some 10 to 20 million people would develop leukemia over the next 30 years. Because this number is so large, even the small percentage increase in radiation, resulting from tests, still can affect such a considerable number of individuals as 50,000.

Should fallout effects of this magnitude be called "large," or "small"? This is not a question which can be answered on scientific grounds. A personal judgment is necessarily involved, based on the problem of securing peace in the world and on moral questions. Some of the factors which must be considered are (1) the uncertainties as to the nature and magnitude of fallout effects, (2) the size of the number 50,000 relative to the numbers of people harmed by other reducible effects, and (3) the fact that fallout effects are globel, involving peoples primarily outside of the testing countries.

As new nations enter the nuclear testing program, it can be expected that they will be interested in testing bomb types which produce a great deal of fallout. There are two dominant reasons for this: First, about the most economical way possible to increase the yield of a large bomb is to use an outer shell of natural uranium. This leads to an inexpensive large energy release, but also to a large release of fission products—the worst kind of fallout. Second, a large amount of fallout increases the devastating power of a nuclear bomb. The addition of a shell of natural uranium to a large thermonuclear bomb can increase the devastating fallout to a very much greater degree, for example, then the addition of cobalt to make a "cobalt bomb," and moreover can at the same time increase the energy release by a large amount, which a cobalt shell will not do.

Although the United States appears to have turned its efforts to designing nuclear weapons which produce less fallout, the U. S. S. R. does not appear to have followed suit as yet. While the United States Pacific tests of last spring

did not add very appreciably to the strontium 90 in this country, the first four Russian test explosions of last fall resulted in an increase of about one-third in the strontium 90 concentration in United States soil, within a period of a few weeks.

Official evaluations of the strontium 90 hazard have generally been given in terms of a "maximum permissible concentration" (MPC). The use of such a term is unfortunate, since it implies that the immediate and long-term effects of small amounts of strontium 90 in human bones are known with precision. They are not. Furthermore, it has been noted by many specialists that a "maximum permissible" dose is not necessarily a safe dose. It is possible to estimate from available data that, if large numbers of people are given a "permissible" dose, or one-tenth microcurie of strontium 90, then it is likely that about one in a thousand will develop bone cancer in his lifetime.

The uncertainty in the specification of a "permissible" level is indicated by the fact that the AEC has recently felt it advisable to revise downwards the strontium 90 level considered "permissible," to one-tenth of the value formerly used—namely, to one-tenth microcurie for the "standard" man. This has brought the AEC "permissible" level into agreement with that recommended for "large populations" by the International Commission on Radiological Protection. No recommendation has been given for the case that the entire world population is exposed; for this case, there is good reason to take the "acceptable" value as being still smaller.

The setting of a "tolerance" level for strontium 90 is arbitrary. On the assumption that even small amounts of radiation will produce proportionate amounts of leukemia and bone cancer, it can be estimated that a worldwide average strontium 90 level equal to the (new) "permissible" level would be likely to produce several million cases of leukemia and bone cancer. (A worldwide average level equal to the old "permissible" level would correspondingly be likely to produce several tens of millions of cases of leukemia and bone cancer.) On the basis of these estimates, one would hardly be willing to permit the world average strontium 90 level to come at all near the "permissible" level.

Although the setting of a "tolerance" level is arbitrary, the strontium 90 hazard from further testing can be measured against the yardstick of the amount of strontium 90 already released by tests to date. By such a yardstick, it could reasonably be argued that an increase of, say, 1 part in 1,000 in the amount of strontium 90 already distributed globally would hardly be considered catastrophic. An increase of that amount would be produced by some 50 kilotons (thousand tons of TNT equivalent) of fission yield—2 or 3 of the small bombs used to destroy Hiroshima.

The explosion of megaton-range weapons may or may not produce large amounts of fallout, depending on the bomb design—in particular, depending on whether a large part of the energy release comes from fission. It would take only a few bombs of a type releasing some 10 megatons of fission energy to double the total global fallout now existing. It is clear that so far as radiation hazard is concerned, for bombs of the types which have been tested so far, attention can be confined to the large bombs. As to the question of how much we should be concerned over a doubling, say, of the fallout produced so far, that is a matter into which many complex factors enter, as discussed previously, and no answer can be given on scientific grounds alone.

Although there is much room for disagreement on evoluation of the available data as to the results of fallout from tests, there is no disagreement as to the utter catastrophe that would result from a full-scale nuclear war. Hundreds of millions of people would be killed outright by blast, fire, and radiation, hundreds of millions more would die from the later effects of radiation injury, further comparable numbers in succeeding generations would suffer from genetic effects resulting from the radiation, and large parts of the devastated countries would be not only destroyed but made uninhabitable for extended periods.

The AEC is to be highly commended for the detailed measurements it has made on fallout, and for the steady release of the results of these measurements. However, the AEC has the dual responsibility of conducting a weapons development program and of evaluating the fallout hazard. It can readily be seen that decisions felt to be necessary in one area might conflict with and unduly influence decisions in the other. Because of these conflicting responsibilities, it may legitimately be questioned whether both functions belong in the same agency.

The suggestion has been made that an independent group of qualified scientists be appointed to take over the study and evaluation of fallout hazard and other radiation problems as well. In 1955, the National Academy of Sciences appointed a group to conduct such a study. The report submitted by that group was prepared over a year ago. Since that time, many new data have become available and many highly qualified specialists have expressed criticisms of the AEC's treatment of fallout problems. It is very desirable that appraisal of these problems be brought up to date. It can be expected that a group chosen to include representation of differing points of view can arrive at agreement on an interpretation of the data which can serve as an authoritative basis for policy decisions regarding future weapons testing.

We are at the present time forced into the difficult position of having to make decisions on a problem that we do not fully understand. The data on hand, however, do tell us this much: The fallout radiation from past tests will constitute over the next few decades a small percentage increase over background radiation. It is estimated that even this small percentage increase will cause genetic changes affecting some tens of thousands of individuals over a number of generations, and will probably also produce leukemia or bone cancer in a comparable number of individuals over the next few decades. The fallout effects will increase with the amount of fission yield in future tests. Each small explosion will contribute only a small addition to the fallout radiation already produced; each large explosion can contribute an appreciable addition.

It should be emphasized that the effects of nuclear tests are thousands of times smaller than the effects which would result from a nuclear war. While there is no disagreement that nuclear war would be a disaster which we cannot afford, there is disagreement on the best means of avoiding such a war, and some of that disagreement seems to have spilled over into the area of evaluation of the fallout hazard.

An objective scientific evaluation of the radiation hazard from fallout can be, and should be made independent of any policy or military considerations. The extensive information being compiled at these hearings will be of considerable help in arriving at such an evaluation.

Testimony on Radioactive Fallout

By Prof. Walter Selove, chairman, radiation hazards committee, Federation of American Scientists before the Joint Congressional Committee on Atomic Energy

I am Walter Selove, associate professor of physics at the University of Pennsylvania. I have been a member of the radiation hazards committee of the Federation of American Scientists (FAS), and was recently elected chairman of that committee. The prepared statement which follows is presented with the approval of the national executive committee of the FAS.

INTRODUCTION

A principal object of this statement is to emphasize that, although there is no important disagreement among scientists on the magnitude of the average dose of radiation due to fallout, conclusions which may appear to be in conflict with each other can be drawn with equal accuracy from the available facts. I should like in a moment to quote from a report just prepared by the FAS radiation hazards committee.

The FAS radiation hazards committee consists of several nuclear physicists, a biophysicist, a biochemist, a chemist, and a cancer research worker. This committee has felt it desirable to make an independent study of the available facts on fallout, and has tried to understand the basis of the apparent disagreement in the various statements on fallout hazards from qualified scientists. I now quote from the report just prepared:

"The committee study of the available scientific facts has led to two conclusions:

"First: The added radiation hazard from continued nuclear weapons testing at the present rate is no greater than that from other radiation normally encountered. The radiation from testing will approach a level equivalent to the natural background radiation to which the human race has always been exposed. Similar radiation doses are obtained from annual chest and dental X-rays.

"Second: This small added radiation, from whatever source, will cause many deaths.

"The committee believes that both conclusions are scientifically correct, and in no way contradict each other.

"Unfortunately, those who believe that we should continue testing in order to maintain a lead in nuclear weapons often emphasize the first conclusion and ignore the second. Similarly, those who believe that a test ban is desirable, since it may lessen international tension, often emphasize the second and ignore the first. The committee believes that both statements must be taken together, since either alone is misleading."

I shall shortly discuss these apparently conflicting conclusions in more detail; I wish here, however, to quote further from the final part of this report:

"While the scientific evidence * * * is admittedly not complete, it is complete enough to allow reasonable conclusions to be made:

"First: The radiation hazard from continued nuclear weapons testing at the present rate is no greater than that from other radiation normally encountered.

"Second: Even this small added radiation will cause many deaths.

"Third: Even more lives are endangered by other radiation hazards, such as from X-ray examinations.

"In the case of X-ray examinations, the advantages are usually important enough to outweigh the disadvantages. The medical profession is alert to this problem and is taking active measures to reduce the exposure to a minimum.

"Will continued testing lead to a hazard that is alarming?

"If one believes, as does Dr. Albert Schweitzer, that continued weapons development will not contribute to security, and that no nation should subject other peoples involuntarily to the effects of fallout, then obviously even one death from fallout is too many and the answer to the question is 'Yes.'

"If, on the other hand, one believes, as does AEC Commissioner Libby, that the security of the country depends on continued testing, then the answer to the question is 'No, the hazard is a small price to pay for security.'

"Each person, in trying to answer these questions for himself, must make personal judgments based on the moral problems involved and on the problem of securing peace."

The members of the committee are agreed that:

" * * * arguments for or against banning nuclear weapons tests must be based primarily on moral grounds and on considerations of international affairs, and not purely on a scientific evaluation of the rediation hazards."

I should like to confine this written statement to a discussion of the scientific questions involved, and will not present in it any evaluation as to whether the fallout hazard from tests is alarming or negligible. I omit presenting any such evaluation in this statement because it cannot be made on scientific grounds alone, and I wish to try to follow the Joint Committee's request to distinguish clearly between fact and opinion.

Rather than trying to cover fallout problems exhaustively, I wish to focus these remarks on a few aspects of the matter, primarily concerned with the global and long-lasting part of fallout.

UNCERTAINTIES IN NATURE AND MAGNITUDE OF EFFECTS

Strontium 90 is generally agreed to be the most worrisome component of fallout at the present time. The energy resulting from the radioactive decay of strontium 90 is only about one-two-hundredths of all the radioactive energy in fallout. Nevertheless, strontium 90 is one of the most important of the fallout components because of its long persistence and its concentration into a relatively small part of the human body—the skeleton. In regard to worldwide effect, it does not appear at present that any other fallout component presents a hazard comparable to that of strontium 90, but I do not believe it has been demonstrated beyond reasonable doubt that no other fallout component is concentrated, perhaps in some particular organ, perhaps through the food chain, to a level likely to present an appreciable hazard.

It must be said that no one knows with certainty what effects will be produced in humans by small amounts of strontium 90 carried for long periods of time. There is no direct experience with strontium 90 in humans under these conditions. We are therefore limited to extrapolations from animal studies and from effects of other radiation sources on humans. On consideration of the physical and chemical effects of strontium 90 radioactivity, there seems good reason to believe that no new type of effect will turn up, but again I believe this cannot be said with absolute certainty.

STRONTIUM 90 DOSE LEVELS AND INTERPRETATIONS IN TERMS OF NUMBER OF INDIVIDUALS AFFECTED

With the support of the AEC, data have been collected from any parts of the globe which permit a fairly accurate statement of the average dose of radiation from strontium 90. There is no important disagreement about this data per se. Here it should be stressed first of all that, in some areas, because of differences in fallout patterns, differences in the nature of the soil and differences in dietary habit, the dose of radiation from strontium 90 may be considerably higher than the average. However, accepting the average figure as a basis for discussion, what are the expected effects? Here, there is some disagreement, involving differing scientific opinion.

The principal effect of strontium 90 will probably be the production of bone cancer and leukemia. Although we cannot know the full effects of strontium 90 in humans until 20 years or more have passed, estimates of the number of new cases induced can be made, as already mentioned, by reference to animal experiments and to certain data on humans who have been exposed to radiation of other sorts.

That radiation can induce cancer is known. Animal experiments show that the number of induced tumors is directly related to the total amount of radiation given. It is true that these data are obtained with high doses of radiation and it is not certain that the results can be extrapolated to low doses of radiation. Such an extrapolation is, on the other hand, a reasonable one and results reported in a recent paper by E. B. Lewis (Science, May 17, 1957) strongly support the validity of such an extrapolation in the case of radiation-induced leukemia in man. Dr. Lewis' work suggests, in fact, that 5 to 10 percent of all present cases of leukemia are due to normal "background" radiation reaching the bones—cosmic rays, and natural radioactivity from our surroundings and from internal sources. If this is true for the cancerlike disease, leukemia, it is reasonable to assume that, for bone cancer as for leukemia, a fraction of present cases is due to normal background radiation. The total number of deaths due to bone cancer, in the United States, is only one-fifth of the number due to leukemia, so if we lump bone cancer and leukemia effects together, estimates of the production of these two diseases by radiation will not be far in error, even if we do not know with any accuracy what fraction of bone-cancer deaths is normally due to background radiation. We therefore assume that about 10 percent of the normally occurring cases of bone cancer as well as of leukemia are due to background radiation. (See p. 962.)

If natural background dose to the bone is responsible for 5 to 10 percent of normal leukemia and bone cancer, then even a small percentage increase over background would harm many individuals. The average bone dose of radiation from strontium 90 derived from tests already conducted will rise to 5 to 10 percent of natural background. The incidence of leukemia and bone cancer would consequently rise one-quarter to 1 percent. Since some 10 million individuals in the next generation would normally die of leukemia or bone cancer (estimate based on statistics for the United States), this one-quarter to 1 percent increase represents 25,000 to 100,000 individuals. Thus, although normally only about 1 in 150 or so of all deaths (statistics for United States) would be due to leukemia or bone cancer, an increase as small as one-quarter to 1 percent in this rate still represents many individuals.

It may be noted that these figures are consistent with a recent estimate made by the Radiation Hazards Committee of the British Atomic Scientists Association. That committee estimated that, subject to the assumption that even small radiation doses produce proportionate amounts of bone cancer, some 50,000 cases of bone cancer might be expected to develop as a result of nuclear tests already carried out.

A great deal of apparent disagreement on the dangers of fallout has probably been due simply to a difference in emphasis. The AEC has emphasized that the radiation from strontium 90 from tests so far will represent only a few percent increase over natural background radiation, on the average, and the AEC has further emphasized that this average increase in radiation due to strontium 90 is small compared to the additional radiation exposure many people receive as a result of living with a higher background radiation level than average, or as a result of medical X-rays. Relative to other sources of radiation, then, it is perfectly true that fallout radiation contributes, at the present level of testing, only a small additional increment. On the other hand, it can be stated that even a small percentage increase over natural background radiation is likely to harm a considerable number of individuals, as discussed above.

The likelihood that even a small percentage increase over background radiation will cause many deaths applies to radiation from other sources as well as to fallout radiation. It should be remarked that the very rapidly increasing awareness of the effects of radiation is already leading to greatly improved X-ray techniques.

THE EFFECT OF FLUCTUATIONS

The fact that fallout is not distributed with absolute uniformity among all the people of the world affects the evaluation of the fallout hazard in two important ways. First, as has been emphasized by many people, and particularly by Dr. William Neuman, if a "maximum permissible" level of fallout material, say of strontium 90, is agreed upon, then the world-average level should not be allowed to go beyond a fraction of it, or else an appreciable part of the world population would be subjected to more than the "maximum permissible" level.

Second, the detectability of fallout effects, which to a considerable extent determines the psychological impact of the effects, will be increased as a result of variations in the amount of fallout, in the diet, and in the nature of the soil. Some 50,000 individuals may develop leukemia and bone cancer from strontium 90 due to tests so far. If these 50,000 were fairly uniformly spread over the world, they would probably be undetectable among the 3 million or so "normal" cases of bone cancer and the 10 million or so "normal" cases of leukemia which would be present even if there had been no fallout. But in actual fact some world areas will suffer more heavily than others, due to diet and soil nature, and due to the nonuniform distribution of fallout. Although the world-average increase in leukemia or bone cancer, for 50,000 total cases due to fallout, would be only 1 in about 60 normally occurring cases of bone cancer, or 1 in about 500 normally occurring cases of leukemia, the relative increase in some areas might be as high as about 1 case for every normally occurring case of bone cancer. Even such a 100 percent effect, from tests so far, might be hard to detect, because the normal incidence of bone cancer is so low, and the number of people subjected to extreme levels is probably very limited.

IS THERE A DANGER FROM TEST FALLOUT?

The two principal effects of fallout appear to be the genetic effects from general gamma radiation, and the production of leukemia and bone cancer by strontium 90. Scientists are agreed that genetic effects are produced even by small amounts of radiation. With regard to somatic effects, such as cancer, a considerable body of scientific opinion holds, with some support from experimental data and with some basis in theory, that these, also, can be produced even by small doses of radiation. On the assumption that this is true, it can be estimated that the number of individuals likely to be directly harmed in the coming generation by strontium 90 will probably be greater than the number showing genetic effects in that generation—although, for a given amount of fallout, the genetic effects will persist for many generations and will eventually cause direct injury to a total number of persons comparable to that affected by strontium 90. In terms of the immediate impact, therefore. we can concentrate our attention on strontium 90. In regord to the genetic effect of fallout, Warren Weaver, vice president of the Rockefeller Foundation and Chairman of the National Academy of Sciences Committee on the Genetic Effects of Atomic Radiation, estimated before a Senate subcommittee on January 16 that radioactive fallout from nuclear weapons testing to date will account for some 6,000 of the 30 million "handicapped" babies to be born in the coming generation. In regard to the effects of strontium 90, estimates quoted previously indicate that some 50,000 individuals over the next 30 years may develop leukemia or bone cancer as a result of tests to date.

How much should one be concerned about fallout effects of this magnitude? This is not a question which can be answered on scientific grounds. Even if one accepts the interpretation of the data presented above as a basis for estimating the number of individuals who will show genetic or somatic effects as a result of the 50 megatons (millions of tons of TNT equivalent) fission yield of tests so far, there are a number of other factors that must be carefully weighed before final judgment can be reached on the significance of the hazard. Probably no absolute evaluation will be possible, but instead, the evaluation will have to be made relative to the problem of securing peace in the world and in relation to the moral problems involved. A discussion of some of these questions would take us out of the area at which this statement is directed.

but it does seem desirable to emphasize that three of the pertinent factors which must be considered are: (1) The uncertainties as to the number of individuals affected (the number may be smaller than 50,000, or larger), and the remaining uncertainties as to what all the effects of fallout radiation will be; (2) the size of the number 50,000 relative to the numbers of people harmed by other reducible effects; and (3) the fact that the fallout effects are global, involving citizens primarily of countries other than the testing countries.

The global effects of fallout (as opposed to the more local effects) come almost exclusively from explosions of large nuclear weapons; a single one of these can produce as much worldwide fallout as a thousand bombs of the size used at Hiroshima. Those who consider the further development of large nuclear weapons necessary for military security view the probable effects of fallout as a small addition to other hazards of day-to-day living. Those who consider that the further development of large nuclear weapons moves the world away from peace rather than toward it of course do not feel that the military desirability of such weapons justifies the probable fallout effects of tests. Finally, there is a third group who, while not certain whether the further development of large weapons is likely to be useful, feel that in view of the uncertainties as to the nature and magnitude of fallout effects, and in view of the international political impact that testing of large nuclear weapons produces in the involuntarily exposed majority of the world, the nuclear testing powers would be well advised to exercise strong restraint with regard to tests producing further significant amounts of fallout.

As new nations enter the nuclear testing program, it can be expected that they will be interested in testing bomb types which produce a great deal of fallout. There are two dominant reasons for this: First, about the most economical way possible to increase the yield of a large bomb is to use an outer shell of natural uranium. This leads to an inexpensive large energy release, but also to a large release of fission products—the worst kind of fallout. Second, a large amount of fallout increases the devastating power of a nuclear bomb. The addition of a shell of natural uranium to a large thermonuclear bomb can increase the devastating fallout to a very much greater degree, for example, than the addition of cobalt to make a "cobalt bomb," and moreover can at the same time increase the energy release by a large amount, which a cobalt shell will not do.

Although the United States appears to have turned its efforts to desiging nuclear weapons which produce less fallout, the U. S. S. R. does not appear to have followed suit as yet. While the United States Pacific tests of last spring did not add very appreciably to the strontium 90 in this country, the first 4 Russian test explosions of last fall resulted in an increase of about one-third in the strontium 90 concentration in United States soil, within a period of a few weeks. Since that time, the U. S. S. R. has set off at least 8 additional nuclear explosions (as of May 15). No information of any precision has as yet been released on the size of these explosions and on the amount of radiactive fallout resulting from them.

THE AMOUNT OF TESTING WHICH CAN BE "TOLERATED"

It is not practical to speculate here on what new kind of fallout radiation might be produced by new types of nuclear weapons. Consequently the "tolerable" level of testing will be discussed in terms of the kinds of weapone which have already been exploded. As discussed previously, attention will be concentrated on the effects of strontium 90.

Official evaluations of the strontium 90 hazard have generally been given in terms of a "maximum permissible concentration" (MPC). The use of such a term is unfortunate, since it implies that the immediate and long-term effects of small amounts of strontium 90 in human bones are known with precision. They are not. Furthermore, it has been noted by many specialists that a "maximum permissible" dose is not necessarily a safe dose. It is possible to estimate form available data that, if a large number of people are given a "permissible" dose, of one-tenth microcurie of strontium 90, then it is likely that about one in a thousand will develop bone cancer in his lifetime. (This estimate follows directly from the results of the British Atomic Scientists Association study of strontium 90 referred to above.)

The uncertainty in the specification of a "permissible" level is indicated by the fact that the AEC has recently felt it advisable to revise downwards the strontium 90 level considered "permissible," to one-tenth of the value formerly used—namely, to one-tenth microcurie for the "standard" man. This has

brought the AEC "permissible" level into agreement with that recommended for "large populations" by the International Commission on Radiological Protection. No recommendation has been given for the case that the entire world population is exposed; for this case, there is good reason to take the "acceptable" value as being still smaller.

Strontium 90 decays relatively slowly (half life of 28 years), and so the effects of amounts produced within a span of only a few years are cumulative. The amount of strontium 90 distributed over the world in fallout is proportional to total fission yield for a nuclear explosion. How many megatons (millions of tons of TNT equivalent) of fission yield can we "tolerate?" No simple answer can be given to this question.

As discussed previously, the setting of a "tolerance" level is arbitrary. It can reasonably be expected that for leukemia and bone cancer due to strontium 90, just as for genetic effects, even small amounts of radiation will produce proportional effects. According to the estimates given previously, a worldwide average strontium 90 level equal to the (new) "permissible" level would be likely to produce several million cases of leukemia and bone cancer. (A worldwide average level equal to the old "permissible" level would correspondingly be likely to produce several tens of millions of cases of leukemia and bone cancer.) On the basis of these estimates, one would hardly be willing to permit the world-average strontium 90 level to come at all near the "permissible" level.

This points up the urgency of obtaining a more accurate evaluation of the level of strontium 90 which can produce injurious effects. It can be expected that it will be some time before such information can be obtained, because the full effects of small doses will probably appear only after 10 or 20 years, or more.

This discussion also points up the fact that use of the term "maximum permissible level" does not give an accurate impression either of the uncertainties involved or of the fact that, if the world-average strontium 90 level were to become equal to the (new) "permissible" level, then the damage likely to be produced is measured in millions of cases of bone cancer and leukemia, and is hardly likely to be "permissible." It would be better to express the magnitude of fallout radiation in arbitrary units—say, in "strontium units"—or else relative to the average magnitude of background radiation.

Let us return to the question of how many megatons of fission yield we can tolerate. It is frequently useful to use the magnitude of an existing hazard as a yardstick for evaluating a contemplated hazard. In this case, we may use as a yardstick the amount of strontium 90 already released by tests to date. By such a yardstick, it could reasonably be argued that an increase of, say, 1 part in 1,000 in the amount of strontium 90 already distributed globally would hardly be considered catastrophic. An increase of that amount would be produced by some 50 kilotons (thousand tons of TNT equivalent) of fission yield—2 or 3 of the small bombs used to destroy Hiroshima.

The explosion of megaton-range weapons may or may not produce large amounts of fallout, depending on the bomb design—in particular, depending on whether a large part of the energy release comes from fission. It would take only a few bombs of a type releasing some 10 megatons of fission energy to double the total global fallout now existing. It is clear that so far as radiation hazard is concerned, for bombs of the types which have been tested so far, attention can be confined to the large bombs. As to the question of how much we should be concerned over a doubling, say, of the fallout produced so far, that is a matter into which many complex factors enter, as discussed previously, and no answer can be given on scientific grounds alone.

FALLOUT IN WAR

Although there is much room for disagreement on evalution of the available data as to the results of fallout from tests, there is no disagreement as to the utter catastrophe that would result from a full-scale nuclear war. Hundreds of millions of people would be killed outright by blast, fire, and radiation, hundreds of millions more would die from the later effects of radiation injury, further comparable numbers in succeeding generations would suffer from genetic effects resulting from the radiation, and large parts of the devastated countries would be not only destroyed but made uninhabitable for extended periods.

REAPPRAISAL OF FALLOUT PROBLEMS

The AEC is to be highly commended for the detailed measurements it has made on fallout, and for the steady release of the results of these measurements.

However, the AEC has the dual responsibility of conducting a weapons development program and of evaluating the fallout hazard. It can readily be seen that decisions felt to be necessary in one area might conflict with and unduly influence decisions in the other. Because of these conflicting responsibilities, it may legitimately be questioned whether both functions belong in the same agency.

The suggestion has been made that an independent group of qualified scientists be appointed to take over the study and evaluation of fallout hazard and other radiation problems as well. In 1955, the National Academy of Sciences appointed a group to conduct such a study. The report submitted by that group was prepared over a year ago. Since that time, many new data have become available and many highly qualified specialists have expressed criticisms of the AEC's treatment of fallout problems. It is very desirable that appraisal of these problems be brought up to date. It can be expected that a group chosen to include representation of differing points of view can arrive at agreement on an interpretation of the data which can serve as an authoritative basis for policy decisions regarding future weapons testing.

CONCLUDING REMARKS

We are at the present time forced into the difficult position of having to make decisions on a problem that we do not fully understand. The data on hand, however, do tell us this much: The fallout radiation from past tests will constitute over the next few decades a small percentage increase over background radiation. It is estimated that even this small percentage increase will cause genetic changes affecting some tens of thousands of individuals over a number of generations, and will probably also produce leukemia or bone cancer in a comparable number of individuals over the next few decades. The fallout effects will increase with the amount of fission yield in future tests. Each small explosion will contribute only a small addition to the fallout radiation already produced; each large explosion can contribute an appreciable addition.

It should be emphasized that the effects of nuclear tests are thousands of times smaller than the effects which would result from a nuclear war. While there is no disagreement that nuclear war would be a disaster which we cannot afford, there is disagreement on the best means of avoiding such a war, and some of that disagreement seems to have spilled over into the area of evaluation of the fallout hazard.

An objective scientific evaluation of the radiation hazard from fallout can be, and should be, made independently of any military or policy considerations. The extensive information being compiled at these hearings will be of considerable help in arriving at such an evaluation.

Dr. SELOVE. On the question of how much testing can be tolerated, you have had testimony here as to a possible permissible testing level of a number of megatons per year. I would like to emphasize that in arriving at a permissible level the question of whether or not there is a threshold is a crucially important one. For those effects for which there is no threshold, for which effects are produced by even small amounts of radiation, then certainly without question from anyone effects are produced by even the smallest amount of testing and if one wants to continue a testing program it can only be on the basis of balancing the presumed advantages from testing against the certain disadvantages in terms of individuals affected.

With regard to the specification of a permissible level, I think it should be said that the use of the term "maximum permissible concentration" is rather unfortunate, especially with respect to strontium 90, since this term implies that the immediate and long-term effects of small amounts of strontium 90 in human bones are known with precision. They are not known with precision.

Furthermore, it has been noted by many specialists that a "maximum permissible" dose is not necessarily a safe dose. It is possible to estimate from available data that if a large number of people are given a permissible dose such as is now used for the population and

if the effects of strontium 90 are proportional to the dose, then it is likely that about one in a thousand will develop bone cancer in his lifetime.

Senator ANDERSON. Does that mean one additional person?

Dr. SELOVE. One in a thousand of all individuals. One in a thousand normally die of bone cancer. I am saying it can be estimated from the numbers which have been given to this committee, that if the entire world population were to receive what is now called a maximum permissible dose of strontium 90 for the population, that is 10 times less than the level prescribed for occupational exposure, then it can be estimated that one in a thousand of all the people in the world would develop bone cancer. That means that several million cases of bone cancer and leukemia would be developed. On this basis one would hardly be willing to let the world level of strontium 90 to come to the permissible level.

Senator ANDERSON. I am sorry I did not get the question clearly. You said a certain number would develop bone cancer independent of the fallout?

Dr. SELOVE. Yes.

Senator ANDERSON. Did you say an additional number would develop it?

Dr. SELOVE. Yes.

Senator ANDERSON. One out of a thousand additionally would develop it.

Dr. SELOVE. That is correct. In other words, even without fallout, during the next 30 years when some half of the present world population is dying, some 3 million cases of bone cancer would normally develop. Some 10 million cases of leukemia would normally develop. If the world were to receive a maximum permissible dose of strontium 90 it can be estimated that these normal effects might be almost doubled.

On that basis we would hardly be willing to have a world average level equal to the permissible level. This rests on the crucial question of whether the effects of small doses of strontium 90 are visible or whether there is threshold. This point you have heard much discussion on. I think you have seen by now that there is not at present sufficient data to know whether there is or is not a threshold behavior.

Senator ANDERSON. Would I be safe in concluding that most of the geneticists believe that there was not a threshold?

Dr. SELOVE. I think that would be a fair interpretation.

Representative COLE. No threshold as far as genetic effects are concerned?

Dr. SELOVE. There is complete agreement on that question. Comparable numbers of individuals will be affected genetically to those affected by strontium. It is true the genetic effects are spread over a large number of generations; so in the coming generation, for a given amount of fallout, the number of individuals who show genetic effects may be 10 times smaller than the number who will show the effects of strontium 90, if they show them at all.

Therefore, as far as the immediate impact of the fallout is concerned I would say we can confine our attention to strontium 90.

The AEC has made very fully——

Representative COLE. On that last statement, Doctor, does that mean that in your opinion we should be considerably more concerned about the effect of strontium 90 than be concerned with the genetic effect of fallout?

Dr. SELOVE. No. I think according to the estimates one can make from the present limited data, about comparable number of individuals will be affected genetically and by strontium 90.

However, during the next 30 years one can estimate that some 6,000 babies will show serious genetic effects from fallout so far, whereas some 50,000 individuals may develop leukemia or bone cancer from fallout so far.

Representative COLE. Of what population?

Dr. SELOVE. The world population.

Chairman DURHAM. That is based on the amount of strontium at the present time in the stratosphere.

Dr. SELOVE. From tests so far. Most of the strontium 90 produced is now on the ground.

The AEC has made very fully available its measurements on fallout, and it should be commended for this. However, the AEC has the dual responsibility of conducting a weapons-development program and of evaluating the fallout hazard. This is a very difficult position for anyone to be placed in. I would not want the job. It can readily be seen that decisions felt to be necessary in one area might conflict and unduly influence decisions in the other. Because of these conflicting responsibilities it may legitimately be questioned whether both functions belong in the same agency.

The National Academy of Sciences has made an independent study of these problems with a group appointed in 1955. The report prepared by that group was prepared about a year and a half ago. Since that time, many new data have become available and many highly qualified specialists have expressed criticism of the AEC's treatment of fallout problems. I think it is very desirable that an appraisal of these problems be brought up to date. It can be expected that a group chosen to include representation of different points of view can at least arrive at agreement on what can be positively concluded from the available data.

I would like to summarize that we are at the present time forced into the position of having to make decisions on a problem that we do not fully understand. That data on hand, however, do tell us this much: The fallout radiation from past tests will constitute over the next few decades a small percentage increase over background radiation. It is estimated that even this small percentage increase will cause genetic changes affecting some tens of thousands of individuals over a number of generations, and will probably also produce leukemia or bone cancer in a comparable number of individuals over the next few decades. The fallout effects will increase with the amount of fission yield in future tests. Each small explosion will contribute only a small addition to the fallout radiation aready produced. Each large explosion can contribute an appreciable addition.

It should be emphasized that the effects of nuclear tests are thousands of times smaller than the effects which would result from a nuclear war. While there is no disagreement that nuclear war would be a disaster which we cannot afford, there is disagreement on the best

means of avoiding such a war, and some of that disagreement seems to have spilled over into the area of evaluation of the fallout hazard.

An objective scientific evaluation of the radiation hazard from fallout can be, and should be, made independently of any military or policy considerations. The extensive information being compiled at these hearings will be of considerable help in arriving at such an evaluation.

Senator ANDERSON. Thank you, Doctor. I do want to assure you that this committee has been trying to make some sort of objective scientific evaluation of it.

Dr. SELOVE. I want to commend it for its effectiveness in bringing out such a vast amount of data in a way which will be very understandable to very many people in such a short time.

Senator ANDERSON. We appreciate very much the contribution which the scientists have made and are making to this, you included. I am very sorry that you had to wait to the end of a long day for your paper.

Dr. SELOVE. I am sorry you had to sit through it.

Senator ANDERSON. We are happy to do it.

The committee will meet tomorrow in the House caucus room at 10 o'clock.

(Thereupon, at 5 p. m., Wednesday, June 5, 1957, the committee recessed, to reconvene at 10 a. m., Thursday, June 6, 1957, in the House caucus room.)

[illegible] scientific evaluation of the radiation hazard [illegible] and should be made independently of any initial [illegible] The extensive information being compiled [illegible] will be of considerable help in arriving at such [illegible]

Senator ANDERSON. I thank you, Doctor. I do want [illegible] that this committee has been trying to make some [illegible] scientific evaluation of it.

Dr. SELOVE. I want to commend it for its effective [illegible] out such a vast amount of data in a way which will be understandable to [illegible] people in such a short time.

Senator ANDERSON. [illegible] read a very fine [illegible] which the scientists have made and are making. [illegible] I am very sorry that you had to wait to the end of [illegible] paper.

Dr. SELOVE. I am sorry you had to sit through it.

Senator ANDERSON. We are happy to do it.

The committee will meet tomorrow in the Senate caucus room [illegible] o'clock.

(Thereupon, at 5 p.m., Wednesday, June 5, 1957, the [illegible] recessed, to reconvene at 10 a.m., Thursday, June 6, 1957, in the caucus room.)

THE NATURE OF RADIOACTIVE FALLOUT AND ITS EFFECTS ON MAN

THURSDAY, JUNE 6, 1957

Congress of the United States,
Special Subcommittee On Radiation
of the Joint Committee on Atomic Energy,
Washington, D. C.

The special subcommittee met, pursuant to recess, at 10:05 a. m., in the caucus room, Old House Office Building, Hon. Chet Holifield, chairman of the subcommittee, presiding.

Present: Representatives Holifield, Durham (chairman of the Joint Committee), Price, Cole, Van Zandt, Jenkins; Senators Anderson and Bricker.

Also present: Professional staff members: James T. Ramey, executive director; George E. Brown, Jr., Hal Hollister, staff technical adviser, and Paul C. Tompkins, consultant.

Representative Holifield. The subcommittee will be in order.

We have planned to start the meeting this morning with a discussion on the present state of affairs, and we have invited Dr. Libby, Dr. Selove, Dr. Neuman, Dr. Machta, Dr. Crow, Dr. Brues, and Mr. Eisenbud to take part in this seminar. I understand Dr. Shields Warren is here, and we will be delighted to have you come up.

Will you gentlemen come forward to the table?

Mr. Hollister will start off the seminar with a statement or question.

DISCUSSION BY DRS. WILLARD F. LIBBY, WALTER SELOVE, WILLIAM F. NEUMAN, LESTER MACHTA, JAMES F. CROW, AUSTIN BRUES, MERRIL EISENBUD, SHIELDS WARREN, CHARLES L. DUNHAM, BENTLEY GLASS, AND WRIGHT LANGHAM

Mr. Hollister. Mr. Chairman, I would like to suggest, because of the long and tiring day we had yesterday, and because Dr. Selove has made special arrangements to be with us this morning for about an hour, that he be given about 10 or 15 minutes to summarize very quickly the points that he was making yesterday afternoon.

Representative Holifield. Will you do that, Dr. Selove, in order that we may regain the points in your testimony. Also I understand you have to leave at 11 o'clock.

Dr. Selove. Yes.

Representative Holifield. So we will let you start off.

Dr. Selove. Thank you.

This discussion yesterday and this morning is intended to provide a summary discussion of the interrelationships and implications on pol-

icy of fallout matters. I would just like to simply itemize the principal points that I would like to make at this time.

First, I think it has been brought out clearly before this committee that there is some effect from any amount of radiation. There is complete agreement among geneticists and others who have studied the problem that genetic effects are produced even by very small amounts of radiation. Of course, with extremely small amounts, there are correspondingly small effects.

But the major point is there is no safe level for genetic effects in the sense of the existence of a level below which no effects would occur. There is no such level.

As to the somatic effects, and perhaps the ones that have received the most attention are the production of leukemia and bone cancer by strontium 90, there is not sufficient scientific evidence at present to determine whether or not there is a safe level for such effects.

I believe it is fair to say, as I said yesterday, that on the basis of the available data, the interpretation that effects are produced even by small amounts of strontium 90 seems to be at least as reasonable as the interpretation that they are not.

The second major point I would like to make is that I believe the term "permissible level" is an unfortunate one to use in discussing fallout effects. These effects, to be sure, may be very small on a percentage basis. At the same time they may involve many individuals. I think "permissible level" is a term which is generally understood to mean a level not such that there is no risk at all, but a level which is taken to be acceptable by those subjected to the hazard in return for some presumed benefit.

Now this is true, for example, in the case of X-ray exposure where there is certainly some genetic effect.

Representative HOLIFIELD. May I interrupt you just a moment? I understand Dr. Glass is in the audience. Dr. Glass, we have one empty chair up here. Would you join us?

Dr. GLASS. Yes.

Representative HOLIFIELD. Continue, Dr. Selove.

Dr. SELOVE. To continue, then, in the case of X-rays, for example, the permissible level has been established——

Senator ANDERSON. Just a second. I would hope if we are going to fill this extra chair, we would add another one and ask Dr. Dunham to come up.

Representative HOLIFIELD. Dr. Dunham, we can find a chair for you, too.

Of course, getting this many here is not to make it mandatory that each one of you take 20 minutes. It is only if you have something of real importance to add that you feel like you should add. Otherwise, it might become unmanageable. But we want to show you the courtesy of an opportunity in case you do wish to speak.

Go ahead, Dr. Selove. Excuse me.

Dr. SELOVE. I would like to reemphasize that a permissible level should be understood to be a level which the people who are subjected to it, or their representatives, have decided is an acceptable level in view of the advantages associated with whatever hazards we are concerned with.

In the case of radioactive fallout, although the effects represent a very small percentage of similar effects occurring normally, in view

of the worldwide concern over this effect, it is not correct to speak of permissible level meaning no risk. It should be understood just what is meant by permissible level.

Senator ANDERSON. In other words, you think there ought to be a balance. You weigh the dangers?

Dr. SELOVE. Against the advantages. Ordinarily, in the case of X-rays, for example, a doctor and his patient decide together—actually, of course, the doctor decides— whether the advantages to be obtained from an X-ray are worth the disadvantages, which are relatively small from an individual X-ray as in the case of radioactive fallout. I do not mean to draw any conclusions here. I just wanted to point out that the peoples of the world are subjected to the radioactive fallout produced by tests from all powers, and they may not be quite so convinced that they are receiving advantages to compensate for the disadvantages. In that sense, the term "permissible," I think, is a misnomer.

The third point I would like to make concerns the size of the effects. This is a very complex question. One simply cannot describe it with a single sentence, or even a pair of sentences. One has to take many aspects of the problem to obtain a balanced view of what the size of the effects is.

I would be glad later to make some further comments on this, but at the moment let me make some very short ones.

Effects from fallout from tests so far are small compared to the effects of other radiation sources to which people always have been exposed, and are currently exposed. The effects are so small they may be undetectable. However, the numbers still constitute many individuals affected even if we just concern ourselves with genetic effects. If we concern ourselves with the possibility of somatic effects in addition, this adds some number of individuals.

The effects are global. They are roughly calculable. They can be calculated by scientists of other countries as well as by scientists of our own.

The next point I would like to make concerns the uncertainties, first, in the magnitude of the effects.

The numbers presented to this committee represent, in general, best estimates as to how many individuals will be affected. No one is in a position to say how accurate these estimates are. I would think it is believed that the estimates that are given you are accurate within 10 times. The true numbers may be 10 times larger, and may be 10 times smaller.

There are uncertainties also in the nature of the effects produced by radiation, by fallout radiation in particular. I do not mean to hint here that there are going to be some serious effects turning up that we do not suspect as yet. I just want to repeat what many witnesses have said—that because many of these things are new to the human race, it is likely to be a generation, until people have carried these radioactive materials in them all their lives, before we really know what the effects are.

It has to be pointed out also that we have good reason to believe we know what the principal effects will be.

Next I want to point out that, so far as radioactive fallout is concerned, one should make a clear distinction between the effects produced by large bombs and the effects produced by small bombs. A

large bomb can produce on the order of a thousand times as much fallout as a small bomb. And if one goes into the question of test bans, then one should make a clear distinction between the effects of small weapons and the effects of large weapons. It is actually, of course, the total amount of fission products released that is important.

Next I want to reemphasize what has been brought out here at these hearings, and a point I tried particularly to make here yesterday, that it can be strongly expected that new nations which enter the testing program will be very interested in testing bomb types which produce a great deal of fallout. There are strong reasons why they will want to do so.

We have seen in the tests of Russia last fall that in just a few explosions, the strontium 90 in this country was increased by one-third, in the soil of this country. This does not represent an increase on a worldwide basis, because of the question of uniformity of distribution and storage in the stratosphere, but the level of the soil in this country was increased by that amount by just a few explosions.

Representative HOLIFIELD. Would you hazard a guess at this time—I do not like to say "guess." Would you hazard an opinion that, in view of the fact the Soviets have tested large bombs on their own territory, there may be an appreciably greater extent of strontium deposit from the local fallout on their own territory?

Dr. SELOVE. In Russia?

Representative HOLIFIELD. In ratio to the stratospheric fallout which has occurred here and raised our level one-third.

Dr. SELOVE. I think probably many of us saw in the Washington Post yesterday morning a Russian report indicating that strontium 90 levels, at least in some areas of Russia, were twice as high as the highest levels in this country.

Senator ANDERSON. Which might explain the sudden Russian interest in stopping nuclear tests and preventing nuclear warfare.

Dr. SELOVE. The decision concerning balance of factors in deciding whether or not to try to obtain an agreement for a test ban—these decisions, of course, involve many other factors other than radiation hazards. I am trying to restrict myself here to that one.

I would next like to reemphasize, as many witnesses here have done, that the effect of a nuclear war would be enormously greater than the effects of fallout from tests.

Again, this remark on this matter: In balancing various factors involved in decisions about test bans, one really has to include such things as the question of whether continued weapons testing will actually help us to avoid a nuclear war. No one disagrees that a nuclear war would be just utter devastation. We have to avoid it. There is considerable disagreement as to the best way to avoid it, but there is no reason for that disagreement to extend into the area of evaluation of radioactive fallout hazards. I think we can and should make that an objective evaluation.

Next, as to the evaluation of fallout hazards, this is anything which has been carried out by the Atomic Energy Commission, by the National Academy of Sciences in an independent study last year, by the British Medical Council, and by various other less formally organized groups.

I think with regard to the Atomic Energy Commission, as I stated yesterday, there is likely to be considerable conflict in the dual re-

sponsibility which the Commission has for evaluating radiation hazards and for concerning itself with the weapons development program of this country. It is a very unfortunate and burdensome problem. I do not think any of us would want to be saddled with it.

I think it can legitimately be questioned whether these two functions—the evaluation of radiation hazards, and concern with the weapons development program—belong in the same agency.

As for the question of independent studies of fallout problems, I would like to make my next point the fact that the National Academy of Sciences report, which is still referred to up to the present time as the latest authoritative report in this country, is seriously out of date. That report was prepared about a year and a half ago. It was issued about a year ago.

The National Academy has, of course, been continuing to study these problems. I think further reports from them at this time would be very much in order nad very welcome. I am sure they are working on them. I think it might be of interest to hear how soon one can expect such further reports to appear.

Finally, I would like to repeat a conclusion of the radiation hazards committee of the Federation of American Scientists, a report from which committee I am inserting in the record. It contains, I think, several numbers of interest.

It is a principal conclusion of that committee, representing some eight people who have studied the radiation hazards problem from fallout quite a long time as such committees go, I think—it is a principal conclusion of theirs that a decision on testing nuclear weapons cannot be based purely on the scientific problems involved, cannot be based purely on radiation hazards, but must involve primarily moral questions, and questions of the effect on international relations and international affairs of our decision on a test ban.

I would like to summarize, then, by saying that the view I have tried to present of fallout problems I think is neither an alarming one nor a calming one. The problem simply does not reduce to such simple terms as those. It is a very complex problem. I do not think a balanced study of it delivers so spectacular a conclusion as that the problem is either of alarming proportions or of negligible proportions. It deserves a lot of serious study. You gentlemen have given it much of that here, and I believe it will be extremely useful.

Representative HOLIFIELD. Thank you very much, Doctor.

Dr. Libby, do you have any comment?

Dr. LIBBY. I find myself in agreement with much of what Dr. Selove said. We certainly must continue to take this problem very seriously, and study it.

In reading the testimony before the committee, I am impressed with the disparity in our knowledge of the biological effects as compared to our knowledge of the physical facts about fallout, which leads me to suggest that we ought to try harder to get some answers to the questions of the genetics, the effects of low radiation doses taken over long periods of time in causing cancer and leukemia, from experimental data.

But we certainly ought to continue the program.

I certainly agree with Dr. Selove this is not a negligible factor. We must take it seriously.

My testimony yesterday shows that I agree with him that nonscientific considerations are involved in the final decision.

Chairman DURHAM. Dr. Libby, I think you are in general agreement that the biological studies should be increased. Does the Commission have facilities available at the present time—and when I speak of facilities, I mean brains and everything else connected—to step this up in the biological field?

Dr. LIBBY. I believe so, but I would like to ask Dr. Dunham to answer that question.

Dr. DUNHAM. Mr. Chairman, we do have facilities. We do have some manpower. This can be stepped up. It cannot be stepped up suddenly by a matter of several orders of magnitude either within the Commission or without the Commission, because there still is, as you hinted in the way you put the question, a limited number of available competent scientists to work in this field.

This is a very real problem, because, if the work is not done competently, it will cause more confusion.

But there definitely could be a step-up in the program over the next few years. There is no question about it.

Chairman DURHAM. Dr. Dunham, since you are in charge of this primarily, do you have funds available, or will you have with what you requested this year from the Congress?

Dr. DUNHAM. We have, as the Congress knows, in our 1958 budget an increase in the request. As you know, these budgets are prepared nearly a year and a half in advance of the coming before the Congress for appropriations.

The cost of scientific living, I mean the cost of doing experiments, is constantly going up. So I would be less than candid if I said that what seemed like a reasonable budget in May of 1956, looks so reasonable today.

Chairman DURHAM. Doctor, since you made that statement, I wish you would send up to the committee your recommendation on it as early as you can. It will not be long before Congress adjourns here, and I think this committee feels like, of course, you should have whatever funds are necessary to carry on the investigation in the biological field.

Senator ANDERSON. May I just ask there: Dr. Selove suggested that the report of the National Academy of Sciences is out of date, and he wanted to know when the next report might be ready. Is there anyone here who can give us any indication of when the new data will be studied and a report made on it, since he says the old report is seriously out of date?

Dr. WARREN. If I might comment on that, Mr. Chairman.

Senator ANDERSON (presiding). Very well.

Dr. WARREN. There is a continuing study made by the various groups assigned by the National Academy of Sciences to all this program. At the present time there are no available facts which would warrant a change to a significant degree in the reports for the pathological effects, the somatic effects. I think Dr. Glass would probably agree with me that there is no very large increment of data in the genetics field.

Senator ANDERSON. The testimony of the geneticists would not indicate that, certainly, would it?

Dr. WARREN. I would rather have Dr. Glass comment on that particular point.

Dr. GLASS. I will later.

Dr. WARREN. Both of the committees have been asked by the president of National Academy of Sciences to meet periodically to keep in close touch with new data as it becomes available, and to issue reports as often as it seems wise to them.

I would say that at the present time, so far as the report on the pathological effects are concerned, that there are no points that as yet are sufficiently well established to warrant a significant change in that report as it stands.

Senator ANDERSON. Now, Dr. Selove, if you think the report is out of date, and Dr. Warren says there are now new facts that have been discovered to change it in any degree, could you give us briefly the basis for your statement?

I rather got the impression there were some new facts, and I thought these scientists had been presenting them. I judge from Dr. Warren's statement, as far as the group, they are going to stand pat on what they put out a year and a half ago.

What do you think about it?

Dr. SELOVE. Dr. Warren is, of course, the chairman of the pathology section of the National Academy of Sciences' study, and is certainly in a position to speak for that group. Some 8 months ago, I communicated with the president of the National Academy, Dr. Bronk, asking whether, in view of the renewed attention that some of the fallout effects were receiving, particularly in view of the attention to strontium 90, whether the National Academy group had any expectation of producing a new report at any time in the near future. And he replied that the matter was under consideration; that a certain number of members of the study group had asked for a renewed study of this problem; and he expressed agreement with the idea that the problem needed more attention, it being understood, of course, that the radiation committees, the subcommittees of the National Academy, have been organized on a continuing basis.

I think there may be some statements in the pathology committee's report which perhaps, in view of the testimony that has been presented to this committee here, might not represent a sort of average view or balanced view of the various notions that specialists in the field now have.

I recall at the moment, not having the report here with me, only one. I think there is a discussion, in the report of the pathology committee, of an unequivocally safe level. I think, in view of the testimony that has been presented here, it is clear that the scientific evidence presently available is not sufficient to say whether or not there is a threshhold level, a level which would be absolutely safe. If the interpretation presented by many specialists in the field that even small doses of strontium 90 can produce bone cancer and leukemia is correct, then I think this point, which I tried to make earlier, that there would be no safe dose at all, is a point which might well be expressed so as to revise that particular statement.

Senator ANDERSON. I know you have to catch a plane, and a member of the staff, Mr. Hollister, has some questions to direct to you. I guess we had better proceed with those.

Representative COLE. If I may come back in where Mr. Durham was discussing with Dr. Dunham the possibility of stepping up this inquiry in the field of biological aspects.

The impression has been created, I fear, that in this field there has been a party line, what might be called a party-line concept of disclosure of the consequences of radiation, in that the Commission has laid out a policy, and other people in the laboratories have tried to find answers which would support that policy.

On the other hand, to disprove that the witnesses, many of them, indicated a large degree of independence in their research, that there is no control by the Commission with respect to their areas of inquiry or conclusions.

Now my question of Dr. Dunham is: In the event that, Dr. Dunham, you do find there is opportunity for stepping up this inquiry, do you see any need or opportunity for enlarging that aspect of independent research beyond what it is now?

Dr. DUNHAM. I do not feel that there is a need to enlarge the independent aspects, because essentially there is a total independence, as Dr. Brues will, I am sure, testify later today when he discusses the research program.

Representative COLE. You mean the present program is essentially completely independent?

Dr. DUNHAM. That is correct, as far as directing or limiting in any way what the scientists, either in the Atomic Energy Commission national laboratories, the major projects, are doing, or university scientists which we support through contracts with our universities.

Senator ANDERSON. Mr. Hollister.

Mr. HOLLISTER. Dr. Selove, I wonder if you would be willing to state for the record, either speaking for yourself or for the Federation, what you feel are the essential differences and similarities in the conclusions of the National Academy report and the British Medical Council report, as far as such questions as genetic effects, MPC, and so on, are concerned?

Dr. SELOVE. As far as what?

Mr. HOLLISTER. As far as such questions as genetic effects, MPC, and so on, are concerned.

Dr. SELOVE. I have to speak for myself, although I believe what I will say reflects the views of the Radiation Hazards Committee. Nothing I say reflects the views of the Federation of American Scientists. This is, of course, a technical matter, and the federation has no policy on such a matter.

I believe the British Medical Council report, which is a report by an organization in Britain separate from the organization which has to do with weapons-testing decisions, just as the National Academy here is, of course, separate from the AEC—I think that report, in my opinion, tends to be perhaps slightly more conservative than the National Academy report.

I recall, for example, that in the British Medical Council report it was suggested that, if the general level of strontium 90 in the population should show signs of increasing greatly beyond one one-hundredth of the occupational permissible concentration, that it would indicate the necessity for a strong reconsideration of the problem.

I think that was a somewhat lower level at which the British Medical Council expressed concern than the level suggested in the National Academy report.

Senator ANDERSON. How much would that be in sunshine units, Doctor?

Dr. SELOVE. That would be 10 sunshine units. The level in the world from tests so far is expected to rise to 2 to 3 sunshine units on the average.

Representative COLE. Mr. Chairman, I wonder if you would mind just experimenting for a moment to see if we cannot do better with these microphones cut off, and everybody speaking up a little louder.

(Discussion off the record.)

Dr. SELOVE. The British Medical Council report, I believe, took a slightly more conservative tone than the report of the National Academy of Sciences. The report of the National Academy group was also conservative, let me hasten to say.

Representative COLE. Speak up so that the audience hears.

Dr. SELOVE. I am not sure I can. I am trying to speak to the committee. I am not sure I can speak to the committee and have the audience hear it at the same time.

I believe in the British Medical Council report another point of difference which occurs to me, with the conclusions that one would draw from reading the National Academy report—another point of difference was that with regard to the possibility that the effects of even small doses of strontium 90 would be felt in terms of the production of a certain amount of bone cancer and leukemia, this possibility was brought out somewhat. I do not recall whether that possibility was discussed in the National Academy report. Perhaps Dr. Warren can answer.

Dr. WARREN. I think probably Dr. Brues can answer this better than I, because his particular group of scientists were the ones who had special knowledge with regard to strontium and the internal emitters.

Dr. BRUES. I am sorry I do not have a copy of the report with me at the present time. I know that the possibility was very seriously considered. I know that it is in some way mentioned in the report, but I am sorry that I cannot say exactly how.

I think that the report looked at the problem from the standpoint of the fact that some radiation is present, that therefore if——

Senator ANDERSON. Is this it [indicating document]?

Dr. BRUES. I am sorry. I am afraid this is not the one which contains the subcommittee report. Perhaps the committee would like to entertain another question while I consult the report.

Dr. NEUMAN. I wonder if it would be appropriate, since we are going to find out what was intended to be meant in the report, to consider what a very interested reader got out of the report, and have that question while we are waiting?

Senator ANDERSON. We are just trying to get through Mr. Hollister's last question before Dr. Selove will have to leave for his plane.

Go ahead.

Dr. NEUMAN. I came away with a little less drastic view than Dr. Selove. I think that the British report expressed this figure of 10 sunshine units essentially as a worry dose—recognizing that, if the strontium levels in the population rose, that we should seriously recon-

sider the matter of MPCs. This is only a suggestion. The British report does not really differ from the National Academy's position.

In reading the NAS report, I do not know, of course, what was intended, but I came away with the feeling that there was a division of opinion in the committee, and that this report indicated some people's feeling that the threshold dose did exist, (the MPC then might be really an MPC) while others did not accept the concept of threshold.

I think there were areas of disagreement, and that a compromise was reached. I hope that, before we leave, this group might consider the matter of the MPC. This finally (the MPC) is the operational number that you use in translating these discussions into real action.

Senator ANDERSON. Thank you, Dr. Neuman.

Dr. SELOVE. I would like to toss out again at that point that I believe, on the basis of available data, the interpretation that small doses will produce effects of strontium 90 is at least as reasonable as the interpretation that they will not, even though no one is in a position to say certainly. However, if one takes the conservative view and assumes that small doses may produce effects until proven otherwise, one is then in a position to estimate roughly how many individuals will be affected by a given amount of strontium 90. And the dose which has been specified as permissible for the population or for a large part of the population, namely a hundred sunshine units, such a dose can be estimated to produce in the world several million cases of leukemia and bone cancer, if that becomes the average world level.

I think it would be generally agreed that, if the occupational dose, which is ten times that level, if the entire world population were subjected to an occupational permissible level, there would be several tens of millions of cases of leukemia and bone cancer, on the assumption that small doses do produce effects.

The difference between the occupational level, and the population level is a matter about which a question was put yesterday, and I think it should be pointed out, in the answer to that question, that the principal reason which the International Commission on Radiological Protection has for specifying a smaller level as a permissible level for the population—the principal reason is the uncertainty that exists as to just what the effects will be.

The International Commission on Radiological Protection has tried to arrive at a level which, on the basis of the limited data available, it thinks will not injure a large percentage of people exposed to that level.

If one considers exposing the entire world population to that level, then, in view of the uncertainties, one simply has to recommend a lower level. That is the basis of the difference.

I am just pointing out that, on the basis of estimates that can be made from presently available data, even the lower population permissible level, if the entire world is subjected to it, could be expected to produce several million cases of leukemia and bone cancer. So we would hardly want to approach that level until we know more surely whether or not small doses do produce effects.

Senator ANDERSON. Do you have another question, Mr. Hollister?

Mr. HOLLISTER. I would like to ask Dr. Selove, do you have any other comments concerning the numerical magnitude of possible effects?

Dr. SELOVE. I would like to make one further brief remark, which I think may be of help to put these things in perspective.

We have talked here about numbers of individuals, such as 50,000 individuals in the world who may develop leukemia and bone cancer from tests so far.

To reduce these numbers to terms where one can more readily feel the impact they have on an individual, we might talk about the situation in, say, the city of Washington, or take Washington and Baltimore together, which I would say probably have a total population of perhaps 3 million.

In Washington and Baltimore together at the present time there are approximately 200 deaths per year from leukemia from normal causes. On the basis of the estimates that have been discussed here, one arrives at the conclusion that, for the next several decades, the results of fallout from tests so far will be that there will be, instead of 200 or so cases of leukemia a year, 202. So there will be that increase, from tests so far, in deaths from leukemia per year in a population of 3 million.

When this is translated to a country such as Japan, with a population of a hundred million, one finds that, instead of the normal 10,000 or so deaths from leukemia in Japan, there might be expected to be about 10,100; and translated into effects for the world as a whole, as compared to this 100 extra deaths in Japan per year, and 2 extra deaths in Washington and Baltimore per year, one comes to some 1,500 extra deaths in the world per year. And over 30 years that adds up to 50,000.

So one should look at all these numbers to try to get some perspective.

Fifty thousand. Is it many individuals?

In terms of impact on a parent in Washington or Baltimore, it means that if a child dies of leukemia, there may be one chance in a hundred that that particular death was due to fallout from tests so far.

Senator ANDERSON. Now, Doctor.

Dr. BRUES. I think perhaps, sir, if I read what was said in the report it may also clear up some other points.

Senator ANDERSON. You are talking about independence, and this is independent. You are perfectly free to do what you feel like. Go right ahead.

Dr. BRUES. The report said:

Permissible dosage to large populations: This is a matter on which no complete agreement was reached by the subcommittee. First responses to this question ranged all the way from the permissible industrial level down to no radiation at all.

These were all people who have given a great deal of thought to the problem.

The uncertainty existing here stems from our ignorance as to whether there is a true threshold for such late effects as malignant tumors, and as to the degree of variation in response of equally exposed individuals.

It is agreed that the only rational approach must take into account the natural radiation background to which the population is exposed.

Then this is tabulated.

It is noteworthy that considerable differences exist from place to place, due mainly to differences in gamma radiation from the environment and in part to

variation to radium content of individuals. Since these existing variations have not given rise to any changes in incidences of tumors or other pathologic states sufficient to attract attention, it was felt that an amount of internal radiation sufficient to double the large population background could certainly be considered safe.

Unfortunately, the word "safe" is in there, and I have less and less respect for the word "safe" as this discussion goes on.

Senator ANDERSON. Thank you very much.

Dr. Neuman, did you have a comment?

Dr. NEUMAN. No.

Senator ANDERSON. Does someone else have a comment?

Dr. Glass, you are a geneticist.

Dr. GLASS. I would like to comment on two matters that have been brought up already: One in respect to whether the genetics portion of the National Academy Committee's report is out of date, and whether an additional supplement is being planned.

We were so much impressed with the relative magnitude of the exposure of the population to X-rays from medical and dental diagnostic and therapeutic practice, in contrast to the relatively minor amount due to fallout on the basis of the current estimates, that most of our attention over the course of the past year has been devoted through our consultants to a more accurate estimation of the medical and dental exposure of the population to radiation; and the report on this study has been given a preliminary release already. I do not know whether it is in the hands of the committee or not, but it should receive general release soon.

As far as the conclusions in our report which relate to the effect of fallout are concerned, I might read that:

Since any additional radiation is genetically undesirable, the fallout dose is genetically undesirable.

Second, the fallout dose to date and its continuing value, if it is assumed that the weapons testing program will not be substantially increased, is a small one, as compared with the background, or as compared with the average exposure in the United States through medical X-rays. From the point of view of this committee, there are two summary remarks that should be made.

Senator ANDERSON. Any dose is undesirable, you say? Read that again, will you please?

Dr. GLASS (reading):

Since any additional radiation is genetically undesirable, the fallout dose is genetically undesirable.

Now, I believe that the testimony of the geneticists at these hearings is simply an amplification of those two statements, and that our position has not been changed at all. It is in accordance with the views that Dr. Libby has expressed. It is also in accord with the views that the geneticists expressed. It depends on how you look at these things.

Senator ANDERSON. Dr. Selove, I know you are in a hurry. Do you have a comment?

Dr. SELOVE. I am in complete agreement with what Dr. Glass has said. I think the genetics part of the report would probably not require serious modification.

I would like to repeat, however, that a very important factor in discussing levels in terms of a permissible level is the question of who decides what is permissible for whom. If it is a matter of a physician giving an X-ray to a patient, the physician decides whether the X-ray disadvantages are compensated for by the advantages.

If one of the several nuclear-testing powers in the world is subjecting the entire world to fallout, even in terms of very small percentage effects—and it is widely agreed, as Dr. Libby has said, that the scientific data indicate that the effects are only a small percentage increase over normal effects, even in such terms, the term "permissible level" implies that it is acceptable by the people subjected to it.

I think that is the important point on which further clarification is needed in the National Academy report.

I will have to leave. Thank you.

Senator ANDERSON. Dr. Machta, you had some calculations on uniformity and nonuniformity you were working on. Are those ready now?

Dr. MACHTA. Yes; if you would like to hear about them.

Mr. Hollister, do you have something else you thought should come first?

Mr. HOLLISTER. I had planned to go next into the strontium 90 question, but it is not necessary.

Senator ANDERSON. Go ahead, and we will come to this. Go ahead with the strontium 90 question.

Mr. HOLLISTER. I would like to do this with the understanding that, if time allows, we will come back to the question of MPC, and its setting later.

Senator ANDERSON. We probably will have time to get back.

Dr. GLASS. Mr. Chairman?

Senator ANDERSON. Dr. Glass.

Dr. GLASS. May I make one remark before we get too far away from the comments that Dr. Libby and Dr. Dunham made earlier?

I would like to testify personally to the complete freedom of investigators who receive support from the Atomic Energy Commission as to the direction of their own work, and also as to the choice of the problems on which they work. This is so important that, in the stepping up of the program, it actually becomes a problem.

The Atomic Energy Commission has leaned over backward to such an extent that it has never even suggested, except in the most informal and free way, to any person what kind of problems we really need answers to.

There is a need, as these hearings have brought out, for the answers to specific questions, which I hope we can find within the framework of the independence given investigators by the Atomic Energy Commission to encourage them to work on particular things.

Chairman DURHAM. Dr. Glass, of course, the reason the question arose was the fact that Dr. Libby said it should be stepped up, in his opinion, as I recall his statement. I think I am in complete agreement that there has been perfect freedom in this field of investigation. There was no implication of my thinking at all that it has been suppressed at any level.

Representative VAN ZANDT. Mr. Chairman, could we have Dr. Dunham fill that gap out?

Dr. LIBBY. Mr. Chairman, would it be in order to ask the people at the table here whether they agree with me about the need for additional emphasis on the biological effects of radiation?

Senator ANDERSON. I think it would be very much in line to do that. Why not just start around the table.

Dr. Libby said why do we not ask the panel whether they agree with him for the need of additional emphasis on the biological aspects.

Dr. Machta, do you want to comment?

Dr. MACHTA. I do not believe I am in a position to comment.

Senator ANDERSON. Dr. Neuman?

Dr. NEUMAN. Because you are not a biologist?

I feel a biologist is not qualified because he would obviously say "Yes."

Senator ANDERSON. We were talking about complete freedom, so we are going to allow a man to disqualify himself as a juror if he wishes to do so. If you wish to, Dr. Machta, we would be happy to have your comments.

Dr. MACHTA. I have no comment.

Senator ANDERSON. All right.

Dr. NEUMAN. I think it is very important, but how to go about it is also very important. I think we should not be swept away by the urgent need for information to just dump money into a program. Very careful consideration should be given to other aspects, such as future procurement of scientists, maybe not just in the field of biology, but also in many allied fields on which we are dependent. I guess that is all, except to urge careful study be made on the way you would proceed.

I would also say, in my own personal opinion, the Division of Biology and Medicine has certainly had a very balanced program, and certainly my own testimony attests to the freedom of scientists within the Division.

Senator ANDERSON. Would you not think, however, that an announcement by the Atomic Energy Commission that it felt this was a field in which there should be a speeding up might result in many other universities, such as Rochester University has done in this field, coming in and saying, "We would like to have a chance to do something, and here is what we would like to explore"?

Dr. NEUMAN. I think a policy statement would be very important; yes.

Senator ANDERSON. I think that is what I was trying to say.

If Dr. Libby made such an announcement across the country there might be many schools which have not taken part that might at least start to survey to see if there are any contributions they might make.

Dr. NEUMAN. I think from just reading the fallout information, a lot of people have not yet done that.

Senator BRICKER. Dr. Libby, are there many of these students that participate in the research programs in the universities—I know you have many that do—are there many of them who continue in the field or related fields to the atomic-energy program?

Dr. LIBBY. I think so. Our interests are so broad, Senator Bricker, that the answer to your question is undoubtedly "Yes." If they move from one discipline to another, the chances are we are interested in the other one, and they do not often move.

Senator BRICKER. They do not get very far away from the field?

Dr. LIBBY. No.

Senator BRICKER. Thank you.

Senator ANDERSON. Dr. Brues.

Dr. BRUES. I think Dr. Dunham and Dr. Libby are both well aware of the fact I have agreed with this point of view for some time, and

much of my concern has been how to implement, in an acceptable way, the progression of knowledge. Sometimes I think there are people who feel that the biological work is a small tail on the rather large animal.

It is true that you cannot expand biological work as you can a reactor program, just by drawing some designs and setting about it. But I fully agree.

Senator ANDERSON. Dr. Dunham.

Dr. DUNHAM. I do not know that I have anything to add to what these people have said. I think the suggestion of the AEC coming out with a fairly strong policy statement as to the needs in certain areas for scientists to work is a good one.

We have tried, as Dr. Glass indicated, through the staff of the division, by direct contact in talking with scientists as we travel around visiting our own projects, and attending meetings, to sort of seduce people into working on things we have felt were of primary importance.

As I will say later, I think we know, and particularly as a result of these hearings, have a better perspective on where the emphasis ought to be during the next few years.

Senator ANDERSON. Thank you.

Dr. Crow?

Dr. CROW. I agree. I have nothing further to say.

Senator ANDERSON. Dr. Warren?

Dr. WARREN. There are two added points that I would like to bring out.

First, I will say that I am in agreement with the other speakers in this field. However, I think one must guard against swinging a program to concentrate on any one phase of activity to a great extent. We have many other problems than the fallout problem with which to concern ourselves in the biological effects of radiation.

I feel that, while it is highly desirable to expand the research program in relation to fallout, there are many other aspects of the program which may be just as essential as fallout now is within a few years' time.

Senator BRICKER. For instance?

Dr. WARREN. Well, let me take the fallout thing itself. When our program was first started in the Atomic Energy Commission we examined very carefully the work that the Manhattan District had been carrying on, and the Manhattan District had very wisely become interested in the problem of strontium 90 quite early in its existence. We already had a backlog of information with regard to strontium 90.

There have been close to, I would say, 14 years of work already in this field, which has brought us up to the state of knowledge that we have at the present time, and makes a large program with regard to radioactive fallout a perfectly sound and reasonable thing at the present time.

Now there are problems in regard to the effect of alpha radiation, the effect of neutrons. There is the problem of how to protect individuals against radiation effects. There are a whole range of things which must not be overlooked in favor of one aspect of the program only.

I am very heartily in favor of an orderly expansion of the program with regard to fallout, but I would hate to see it at the expense of the present program, which I regard as a well-rounded and effective program.

Senator ANDERSON. Dr. Libby, you raised the question so you do not have to comment. Do you care to comment again now, or go on around?

Dr. LIBBY. No comment.

Senator ANDERSON. Mr. Eisenbud?

Mr. EISENBUD. As a nonbiologist who needs answers from biologists, I would heartily agree that some of these answers have got to be obtained before we can proceed much farther. On the other hand, I am impressed by Dr. Warren's remarks concerning the need for a balanced point of view.

I do not think it has been brought out in these hearings, Mr. Chairman, that one of the reasons why so many of these unanswered questions exist is because we know so much rather than so little. No substance to which human beings are exposed, either in the air or in the food that they eat has been investigated so thoroughly as radiation and radioactive materials. The total investment of the United States Government and private industry in the whole field of air pollution research has been something like $5 million in the last 5 years. Dr. Dunham can tell us how much has been spent in this field, but certainly it is in the order of $200 million or great in the last 5 years. It is simply characteristic of good research that answers beget questions, and for every answer that we find, we raise 1 or 2 or 3 new questions.

I feel intuitively that if we had full information about organic chemical heavy metals, and other things to which we are exposed in our environment, we should be asking the biologists many of the same kind of questions we are asking them today in regard to radiation effects.

Senator ANDERSON. Thank you, Doctor, for a very fine statement.

Senator BRICKER. I have not been able to follow the conclusions that some of you have reached. I have asked the question a time or two as to how much of the leukemia and how much of the bone cancer at the present time, regardless of imposed radiation, is due to the background radiation, and how much is due to other metabolic and biological factors, or facts. I have not had an answer to that question yet.

How in the name of sense are you able, from an indefinite base of that kind, to extrapolate, like one doctor did a moment ago, and say there would be 102 cases of leukemia in Baltimore and Washington, rather than a hundred cases, because of the imposed additional radiation from strontium 90? There is a gap in there someplace I have not been able to fill in in my own thinking. If somebody would explain it to me I would appreciate it.

Dr. WARREN. I do not think I can explain it entirely satisfactorily, Mr. Senator. But I would like to stress that when you establish a hypothesis, you can draw conclusions from that hypothesis, but, as you point out, those conclusions have no more validity than the hypothesis itself.

Senator BRICKER. No more validity than the hypothesis itself?

Dr. WARREN. And I think you will recall that I said on Monday I am not at all satisfied that strontium 90 will cause any additional cases of leukemia. I would think there is a possibility of an increased amount of bone cancer, but I am very skeptical as to leukemia.

This again, you might say, is only a hypothesis.

Senator ANDERSON. We did have, did we not, the statement from Dr. Selove yesterday, in which he quoted Dr. Lewis as saying that 5 to 10 percent of all present cases of leukemia are due to natural background radiation?

Dr. WARREN. I know of no way in which that can be established or proved, Mr. Anderson.

Senator ANDERSON. I do not know either. I just simply say that we have scientists who think they know.

Dr. WARREN. Yes. That is, I think that is a fair and reasonable assumption, but I do not think we are warranted in accepting it as an established fact.

Senator BRICKER. It is nothing more than an educated guess.

Senator ANDERSON. When you say, also, that one microcurie or one-tenth of a microcurie is a safe background, that is also an educated guess, is it not?

Dr. WARREN. No. I feel—well, yes, it is an educated guess.

Senator ANDERSON. Yes. That is right.

Dr. WARREN. But I think it has a little more foundation.

Senator ANDERSON. There is not a particle of difference between the two educated guesses.

Dr. WARREN. I think there is. If I could say one more word here. We know there is nothing unique about the radiation from strontium 90. We also know that there are much greater variations in radiations which have not produced clearly measurable effects. There may be some shadowy effects. We cannot say there are not any, but we can say there are none that are significant and measurable.

I would have no hesitancy in moving my family, myself, to your own State, sir, which happens to have a higher background, I believe, than Washington, and the strontium radiation is only a small part of this difference.

Senator ANDERSON. Now we are back on something we can agree on. I will be happy to say that I am willing to face a slight degree of leukemia and bone cancer for the privilege of living in my State.

Senator BRICKER. May I ask one more question?

Senator ANDERSON. Senator Bricker.

Senator BRICKER. On the assumption, Dr. Libby, or anyone else, that we had a hundred million dollars to spend in biological research this coming year, from what kind of an expenditure would you receive the greatest benefit, an expenditure in this field, or an expenditure in the field I think Dr. Eisenbud mentioned a minute ago, in the field of pollution of the atmosphere, the effect of automobile exhausts, smog in Los Angeles, and many other things that are present in contamination of foods, waters, and the like?

Have we neglected the one for the other? And should we give more attention to the general biological human effects of other deleterious things?

Dr. DUNHAM. My answer to that, Mr. Senator—anybody else can add their comments—we have not neglected one for the other. We have neglected the general effects you are talking about of all chemical

elements and things that are in the environment on man. I think that one should not, by that statement, then say that we should stop all work on radiation effects when we are just on the brink of learning a lot of important things that will help resolve some of the radiation problems. But there is no question but what the other activity dealing with all of these other substances about which we really know relatively little should be stepped up.

Senator BRICKER. You know there is one result of this meeting and this hearing—I think it has a good result generally—people are getting scared.

I said here a while ago that as a result of cigarette smoking and radiation, I am surprised we have got so many healthy people around. In other words, the other field is, to my mind, the more important in numbers, and we have overemphasized the radiation effects insofar as we know what they might be at the present time. I think we ought to turn some attention to the general and not neglect this, mind you, but we ought to give more attention to the general field of biology.

Dr. DUNHAM. I think there is no question about it, sir.

Senator ANDERSON. We got as far around as Dr. Glass.

Senator BRICKER. I am sorry.

Senator ANDERSON. I appreciate, Senator Bricker, your coming in here.

Dr. GLASS. I have already expressed myself on this question sufficiently in my testimony, I think, so I will just pass it on.

Senator ANDERSON. Dr. Langham?

Dr. LANGHAM. I had decided more or less what I was going to say until Mr. Bricker asked his question.

I do not think there is any doubt but that radiation has been the most emphasized environmental hazard that man has ever been subjected to. Why? Because it has a radioactive tag on it, which makes it easy to discover, and it is also new. Therefore, it has received considerable interest. So I would heartily agree with Mr. Bricker.

On the other hand, I would like to point out one thing which applies to radiation specifically, and perhaps to man's industrial environment in general, and that is that probably for the first time in the history of man he has advanced technologically to the point where he is beginning to become the weak link in what he can accomplish in the future.

I mean by that—let's take the human heart. It can wear out any mechanical pump of the same size that can be built by man.

If we want to, we can say that a man can ride a horse until it drops, or he can fly airplanes until they fall out of the sky with mechanical failure.

Now, however, we are reaching the point where man can build machines and gadgets in which he is the weak link in their operation. In fact, I might say that man has now reached the stage where he is the weak link in his ability to wage war, because he is reaching the point where in so doing he might even annihilate a large portion of his own population. This is even true of the man who wins the war.

So I would say, yes, by all means we should emphasize biological and medical or, let me say, human factors investigations. I do not mean we should do this without adequate planning, but I think we

should emphasize more the human factors in all aspects of our technological advancement.

I think for the first time radiation has called this to our attention.

Senator ANDERSON. Thank you very much, Dr. Langham.

We went around the circle. Now we shall start again by asking Dr. Machta if he wants now to present his information.

Chairman DURHAM. Mr. Chairman, I would assume from Dr. Langham's statement, then, that he believes it is a good thing for the human race that we do have radiation. Is that correct?

Dr. LANGHAM. Not necessarily.

Chairman DURHAM. Because of the fact that it has caused us to study all of these related things, such as biology and medicine and everything else. Now we have been studying the cancer cell here for generations. Of course, it is nothing new. We have failed to reach any decisions as to the cause of cancer prior to strontium 90 or prior to any fallout; had we not?

Dr. LANGHAM. Oh, yes, very definitely.

I would not say it is necessarily good that we have radiation, but I should say, also, that I do not think it necessarily means we have to stop further technological advancements because radiation is a factor in that advancement.

Chairman DURHAM. We have to have it.

Senator ANDERSON. I am very happy to see so many heads nodding in agreement. We are happy to find something on which these scientists agree.

Thank you, Dr. Langham, for producting that motion.

Chairman DURHAM. I raised the question because of the fact we have lived under sunshine here for thousands or millions of years, and we still have a future, I think. With the radiation that we are trying to determine—of course, that is the purpose of all of these hearings—can we continue to live with this thing, with the manmade process we have come to today? We have accepted the others for years because there was very little we could do about it.

Is that not essentially correct, or is it?

Dr. LANGHAM. You are very definitely correct. We have lived with many things in the past that are of our own doing.

For example, the smog from automobile exhaust. And I think we will continue to do it.

I am certainly in favor of saying we have faced many things that we have had to face because they were a part of our environment, and we could do nothing about them. We have also faced many things which are of our own creation in the interest of technological advancement. I say, yes, let's continue to face them, and by all means let's not think that radiation is the only one, and let's not think that radiation is the insurmountable one.

Chairman DURHAM. That is right. I agree with you.

Senator ANDERSON. Mr. Van Zandt.

Representative VAN ZANDT. Dr. Warren, did I understand you to say a moment ago that the data we have at the present time represents a spread of 40 years?

Dr. WARREN. It actually represents a still wider spread than that. We began to get some understanding of the effects of radiation in the late 1890's, very soon after the discovery of the X-rays, and the isolation of radium, and our knowledge of the field advanced very

slowly only recognizing the more extreme cases in the early days.

Then, with the concerted attack on the problems of atomic energy in the Manhattan project, there was added a great deal of additional information, which is being kept up and added to all over the world at the present time.

I also mentioned that there is one population in India on the Monazite sands which has lived there for many generations. We do not yet know the medical and biological effects of this, but these will be studied by the Indian Government, and the data made available to the world.

Senator BRICKER. Is there any indication that those people who did live in the Monazite hills have had a shorter life span?

Dr. WARREN. I do not believe that the vital statistics are sound enough——

Senator BRICKER. They are not sound enough yet to determine?

Dr. WARREN. To draw conclusions at the present time.

Senator BRICKER. Either on that, or leukemia or cancer?

Dr. WARREN. No. I think we will have to have pretty careful studies for a fairly long period of time to answer this.

Senator BRICKER. Are there not also some places in South America where the same conditions exist?

Dr. WARREN. Yes, there are. There are Monazite areas in Brazil. But here the inhabitants are chiefly Indian tribes who do not stay in any one area very long. And while studies are being carried out by the Brazilian Government, there will not be as large a population available for study.

Senator BRICKER. There is no evidence, is there, that there is any resistance in the human body built up to this radiation like there is to bacteria?

Dr. WARREN. No, there is no evidence there is any resistance. In fact, there seems to be an indication that, if there has been a significant amount of radiation given at one time, that individual will actually stand somewhat less radiation.

Senator BRICKER. Is that because of the accumulation or because of the weakening of the cells?

Dr. WARREN. That is probably because some of his cells have been injured or killed.

Senator BRICKER. Weakening, yes.

Senator ANDERSON. Now, Dr. Machta.

Dr. MACHTA. Do you want at this time to go into detailed numbers, sir? This is what has been suggested that I do.

Mr. RAMEY. In some detail. Not necessarily in absolute detail. I think the idea was that you made certain statements in your testimony last week, and from that information on the possible nonuniformity of deposits that you could make some calculations on future predictions of what might be deposited.

Dr. MACHTA. Thank you.

There are essentially three reasons why the fallout over the globe should be nonuniform. Two of these have never been in question. These are that tropospheric fallout from tests occurs primarily in the band of latitude in which the tests take place.

(Discussion off the record.)

Senator ANDERSON. Proceed.

Dr. MACHTA. I mentioned that there are essentially three reasons I believe why there might be nonuniform fallout over the globe. Two of these have never been in question and have been well accepted.

First, the tropospheric fallout occurs primarily in the band of latitude in which the test takes place.

Second, rainfall being the primary mechanism by which particles are removed from the atmosphere, will give rise to greater fallout in rainy areas than nonrainy areas.

The new element which has been added, and on which there may be some question, is the point I wish to talk about. This is the fact that fallout may be coming from the stratosphere preferentially in the temperature latitudes of Northern Hemisphere.

My contention is that I am not sure to what extent this occurs, and in view of the possibility, we ought to take into account a very conservative as well as, let us say, a very optimistic picture, in the hope that the truth will lie between the two.

What I would like to offer now are the numbers that one gets out of two pictures; one which essentially gives rise to complete uniformity from now on, and one which gives rise to extreme nonuniformity.

The latter is supposed to represent an absolute upper bound.

In order to compute my numbers, I have to make an assumption of the amount of debris still in the stratosphere, and from the previous testimony, up to mid-1956 there are still about 24 megatons equivalent of fission products left in the stratosphere. When it comes down, it will decay, and consequently there will be a smaller amount deposited on th ground. However, one might say that the tests which have been held since mid-1956 may balance the decay.

I can talk about two conditions: One, there are no more tests, and we want to find the fallout in terms of what has been put in the stratosphere already; and, second, the tests continue at whatever rate you wish to specify.

The curve which I drew several days ago describing the nonuniformity of fallout from all sources—and one need not ascribe it to either tropospheric or stratospheric fallout—shows about 10 millicuries per square mile as the average peak in the north temperate latitude as of 1956. If you have 24 megatons—this is equivalent to 12 millicuries per square mile when spread uniformly over the globe—by adding 10 and 12 one gets 22 millicuries per square mile in the north temperate latitude for the case of uniform future fallout.

Now, superimposed on——

Mr. RAMEY. How much is that in subshine units? Is it the same?

Dr. MACHTA. No; it is not the same.

Dr. LIBBY. You get the answer for the average soil pretty well by multiplying by two.

Dr. MACHTA. But these sunshine units are not in the human bone. There is still the discrimination factor.

Dr. LIBBY. These are sunshine units in the soil. So with Dr. Machta's 22, that is the average soil will be 40-some sunshine units.

Dr. MACHTA. This, then, would represent a sort of lower bound for the average temperate latitude of the amount that would have fallen out from tests conducted essentially to date. One can put this into perspective. According to the latest information, the norhern tier of the United States has about 33 millicuries per square mile.

However, superimposed on a value of 22 millicuries per square mile for the average band in the north temperate latitude, one should take into account the fact that there are areas of heavier rainfall than average. I have stated that the factor taking weather into account should be no more than 2 or 3. I think a reasonable number is two.

Therefore, I have suggested that the lower limit of the total fallout which will occur in the northern temperature latitude will be in the order of 40 or 45 millicuries per square mile, or converted to something under 90 sunshine units in the soil.

There is another way of looking at the matter. In a recent letter from Dr. Brewer, he suggests that stratospheric debris will come out almost entirely in the temperate latitudes of each hemisphere. This reasoning would yield a marked peak in the temperate latitudes.

The number I am about to quote is the upper bound. Mixing in the stratosphere must of necessity make this lower. I am not offering it as a number that would occur. I would like to make this clear. But since this an absolute extreme, the true number would be less than it.

The number I would like to offer is something of the order of 60 or 70 millicuries per square mile on the average. And again multiplying by the factor of 2, would bring the answer up to 120 or 140 millicuries per square mile.

As I say, I am not quoting this as a number we expect to have. This is a thing we can expect as the absolute upper bound.

This is what will happen from the tests to date. Fallout of strontium 90 will lie somewhere between 40 and 120 millicuries per square mile.

If we continue our tests at about the same rate, one can also make a calculation. Dr. Campbell, formerly of the National Academy of Sciences, has published an article in Science which shows how to take into account not only the radioactive decay, but the storage in the stratosphere. (See p. 1338.)

Presuming the test rate to continue at approximately 10 megatons per year—and if one desires any other test rate the answer is proportional to it. The assumption is made that the mean stratosphere storage time is 5 years—and I prefer this to 10 years, since it gives a slightly more conservative answer, although the difference is less than 10 percent, which is negligible. Then the answer at equilibrium, which will take many years to achieve, would be something in the order of 350 millicuries per square-mile or a little less, to something in the order of 850 millicuries per square mile. A very conservative picture is one in which the nonuniformity is no greater than a factor of 2 as the ratio of peak to average. A guess on my part—and I must admit this is a guess since I have not gone through any quantitative calculations—calls for a factor of 5 for the case of extreme nonuniformity for the ratio of peak to world average.

These give rise, as I indicated, to a range of 350 to 850 millicuries per square mile.

Mr. HOLLISTER. Is the concept of a mean storage time, in your mind a valid one for stratospheric fallout?

Dr. MACHTA. Well, meterologically speaking, it is not necessarily a valid one. I think it is useful for the purposes for which we are using it.

Mr. HOLLISTER. That is, empirically it is valid, although essentially we agree it offers some problems?

Dr. MACHTA. That is correct, sir.

Mr. HOLLISTER. The numbers 350 and 850 correspond essentially to equilibrium after a long period of time? Many years?

Dr. MACHTA. That is so.

Mr. HOLLISTER. Could we not say that within, shall we say, 35 to 100 years we will be so close to the equilibrium value that from then on we can say we are at equilibrium, assuming a constant rate?

Dr. MACHTA. I think this is roughly in the order of magnitude of time to achieve equilibrium; yes.

Mr. HOLLISTER. What is the factor you used to multiply by, to get this equilibrium number?

Dr. MACHTA. In the case of the most conservative picture, I assumed the peak was in the order of twice the average for the world. At the present time, the peak is something, I think, about two and one-half times the average of the world.

In the case of the least conservative picture, which I again view as an extreme and not likely to happen, it is of the order of five times the average for the world.

Mr. RAMEY. What are those figures, then, translated from your microcuries per square mile to bones, for example? Is that possible?

Dr. MACHTA. It is possible, if the gentleman here would provide the numbers. The first step would be to convert to subshine units in soil, which Dr. Libby has done very simply. The next step would be the discrimination factor. This I do not know how to do.

Mr. RAMEY. Perhaps we might carry on from there, and let possibly Dr. Libby, Dr. Neuman, or Dr. Eisenbud apply these discrimination factors, and see how we come out on these upper and lower limits.

Dr. NEUMAN. In my previous testimony, I said that my personal choice of the discrimination factor, and one that I thought represented the best guess, perhaps, was 8; and that I firmly believe the true discrimination factors lie between twice or half of these limits, between 4 and 16.

As far as I know, the other testimony of people using discrimination factors involved figures that also lay between these limits. I think some used 14.

I think, also, that this represents, as I mentioned, the view of Dr. John Lautit, of England. I believe he is on record as choosing eight for the present.

Really this is pretty good, considering some of the spread we have heard in statements. A factor of 2 is relatively good.

I think we are weak on estimates of effects, but in determining levels we can pretty well agree that it lies between 4 and 16.

Dr. LIBBY. I would comment on Dr. Machta's calculations as follows:

Dr. Machta and I agree that the question of the uniformity of stratospheric mixing is one that has to be settled by further experimentation.

There is a great deal of evidence for a certain amount of stratospheric mixing, as I remarked yesterday, since we do find strontium 90 in fallout, even at the South Pole and we have never found any place in the Southern Hemisphere where it rained at all where we did not find strontium 90.

So I think the extreme calculation is really rather extreme. It is all right to put it down, but I am sure Dr. Machta would agree it is rather an extreme calculation.

We will know this answer, as I said yesterday, by our stratospheric monitoring program. That is, we will know the matter of the horizontal mixing.

Now the possibility of concentration in terms of leakage at a certain latitude is an additional point which we will also learn about in connection with our studies of the age of the fission products that come down in our washtubs. We can determine simply whether the material that we collect is old or young. If it is coming from the stratosphere, it must be old; if it is coming from the troposphere, it must be young. And this program will settle it.

Representative VAN ZANDT. Dr. Libby could you include in your statement a time factor? And if that is not possible, will you know in a year or 2 years?

Dr. LIBBY. I think we will know in less than a year. Maybe I am being optimistic, but a year ago we would not have made the flat statement that rain is it, as far as fallout is concerned. And we all make it now. We have established that firmly, we think, during this past year. I think so.

Representative VAN ZANDT. Can you definitely establish the age of the fallout?

Dr. LIBBY. Yes, sir. It is not the easiest thing in the world to do, and it has not been done very often, but it can be done.

Representative COLE. Does not this fallout have any weight at all?

Dr. LIBBY. No, sir.

Representative COLE. None whatever?

Dr. LIBBY. Negligible. We think the particle size is so small you cannot see them, even by the highest powered microscope. We think the particle sizes are well down in the hundredths of a micron range. They may be as large as a tenth of a micron. They are very, very tiny, too small to see, but we can measure them by virtue of their radioactivity.

Now the matter of the discrimination factor. I would not argue with as eminent an authority as Dr. Neuman, and I certainly think his statement is a fair one from all that I know about this difficult subject, that the discrimination factor between average American diets and the bones should be between 4 and 16. I think certainly that must be right.

There is one little point I would like to make that seems not to have been made by anyone. I think it is just accident.

We find the fallout in the top 2½ inches of the soil; therefore, deep-rooted vegetables get less strontium. This is a point worth thinking about. If you plow it to distribute it, it will still get less strontium, for there will be more calcium to dilute it.

So the short-rooted grasses which produce the milk give us about the worest situation.

So I think there is another factor. It is not what we should call a discrimination factor, I think, in the human body, but there is a sort of physical factor as to the depths of the strontium 90 deposits relative to the length of the roots which pick it out of the soil. I do not know what that number should be, but I think it is certainly a significant number.

Maybe like—I do not know. Certainly a number greater than 1, and probably less than 2. So there is a factor which could be multiplied by the discrimination factor which Dr. Neuman gives.

Senator ANDERSON. Mr. Hollister.

Mr. HOLLISTER. Dr. Libby, then you would agree that we should use equilibrium discrimination factors at the moment rather than, for example, to try to relate soil data to, let us say, Kulp's bone data?

Dr. LIBBY. I think you should do both, Mr. Hollister. That is, you should figure out—we know now, as Dr. Kulp has said, that fresh bone is, I think he testified, 0.7 sunshine units in this country, and this at a time when the average soil was—well, it is hard to say just when the children got their average food—but say, 50 sunshine units. Between 40 and 50 sunshine units probably was the ground concentration.

Now all of these factors have come in to give us a ratio in excess of 50 between the ground concentration and new bone at the present time.

Now, the equilibrium discrimination factors are only part of it. I just mentioned another one.

There is another factor I have not mentioned, and that is that the Imperial Valley and desert regions supply truck crops through irrigation methods where the strontium 90 is lower. So this leads to a decrease in the strontium 90 content of the average diet, you see. That should be in.

Mr. HOLLISTER. I am going to go from an MPC in the body, and let's beg the question whether there is one at the moment. If I want to go from MPC to testing rate, which I think Dr. Neuman showed we could do in principle——

Dr. LIBBY. Yes.

Mr. HOLLISTER. How do we get there? Do we go by way of a factor of 50 or 30, or by a factor of 8?

Dr. LIBBY. I do not think we know the right answer to that question. I think you had better do both of these calculations. It looks to me like we have to admit to a certain ignorance as to the correct way. That is, we have 4 to 16 as the range here. I really think Dr. Neuman knows the factor a little better. But that is a fair range. Then we have to estimate these various additional things I have mentioned, and there are some unknowns in this calculation.

Mr. HOLLISTER. Could I ask Dr. Neuman to comment here?

Dr. NEUMAN. I think everyone will admit to uncertainty, and I would be the last one to say that we are certain of these figures. I stated 8 as the best number and estimated the range of 4 to 16, in terms of the variability in results reported thus far. There may be something basically very wrong with the results on final equilibrium factors, in which case our models are wrong. There may, indeed, be another factor of perhaps as much as 2 either way. If we multiply 16 by 2, we are up to 32. I think the uncertainty over how soon equilibrium is achieved means that the higher relation found in the survey data is not really in disagreement with the experimentally determined discrimination factors.

I would like also to say that I felt the best model to use is natural strontium, something with which we truly are in equilibrium. However, I feel the data are much less certain than some of our experimental data with isotopes, because I have distrust of emission spectro-

scopy as applied to a wide variety of samples and the difficulty of estimating what is actually the average diet.

I think there is an important document that received no attention (but it is in the record) submitted by Norman McDonald, of UCLA, comparing his best estimate of the Sr/Ca ratio of average diet and of human bone, determining natural strontium by suspected emission spectroscopy. He came out with an estimate of the equilibrium discrimination factor as 15. This is right in between my estimate of 8 and some of the higher estimates from the survey data. (See p. 720.)

So I will confess to this uncertainty, and I think, for purposes of calculation, that we should take them at several levels. I would suggest 8 be considered one reasonable value, and 32. Well, that's too high. I would say 16, right now, is not an unreasonable guess. It might also be 4.

Would you agree, Dr. Libby?

Dr. LIBBY. I think so. If you have to pick a best number, something like that (8 or 16) might be the best one.

Senator ANDERSON. Dr. Langham, I wonder if you would like to run through that table now of the calculations showing strontium 90 in the bone in relation to maximum permissible concentration?

Dr. LANGHAM. This is a repeat of numbers in essence that were placed before you last week. What it amounts to is taking the various estimates of present levels in the bone, or present levels in the soil, and applying ecological discrimination factors, and estimating what the equilibrium level will be when our entire environment is in equilibrium with the present test rates, assuming 10 megatons of fission yield per year injected into the biosphere.

All this amounts to is taking the various estimates of present bone and soil levels and applying Dr. Libby's factor of 8, which is about what these levels will be increased by when equilibrium is reached, assuming that we can continue to test at 10 megatons fission yield per year.

First, we can take Dr. Libby's estimate given in his recent speech, in which he estimated that the United States or rather the north temperature population belt, would reach equilibrium bone levels of somewhere between 5 and 20 sunshine units. These values were derived by assuming different ecological discrimination factors: 1 in the region that Dr. Neuman is talking about, and 1 in the region which Dr. Libby has talked about on occasion, up around 80.

If we take Dr. Kulp's estimate of two for the equilibrium bone level assuming no more tests, which, of course, is for the general population of the latitudinal belt, and multiply it by 8, we obtain an equilibrium bone level of 16 for continued testing at the present rate.

I have taken Kulp's bone data and applied certain corrections that I think refine it to some degree.

Mr. HOLLISTER. At the present rate of testing, and assuming the rate continues, or at the present rate of testing, assuming no more testing?

Dr. LANGHAM. These are numbers assuming we continue to test at the equivalent of 10 megatons of fission products injected into the biosphere per year until we reach equilibrium. This is until the decay of strontium is equal to the amount we are putting up.

So we have 5 to 20 sunshine units by Dr. Libby. Dr. Kulp's numbers give 16.

Our numbers from Dr. Kulp's bone data is 25.

Mr. Eisenbud, in this meeting, estimated about 40 from New York milk data.

My estimate on the basis of other milk data gives an equilibrium bone level of 29.

If we take the soil data, which is the 22 millicuries per square mile that Dr. Libby has estimated at present, multiply by 1.8 to give sunshine units in the soil and apply a discrimination factor of 10 in going from soil to bone, we come out with 42.

Now, Dr. Machta's numbers that he just quoted are handled in this fashion. That is, we take the soil numbers he gave, multiply them by about 2 (1.8 to be exact) to get them to sunshine units in the soil, and then take one-tenth of that, assuming a discrimination factor of 10, and we come out with an estimate of from 70 to 170 sunshine units ($\mu\mu c$ Sr–90/g bone Ca) as the equilibrium bone level that will be reached if biospheric contamination continues at the present rate.

Mr. HOLLISTER. Which soil data are you taking now?

Dr. LANGHAM. The ones Dr. Machta just gave as the equilibrium level with continued testing (350–850mc/mi^2). Is that not right?

Dr. MACHTA. Yes.

Dr. LANGHAM. The ones he just gave would predict, then, for the United States on this basis, assuming a discrimination factor of 10, 70 to 170 sunshine units in bone at equilibrium.

Now the maximum permissible level that we have been talking about is 100 of these units. So I think you can see that at the present rate of testing, if we assume all numbers other than Dr. Machta's are correct, the average is a factor of 3 below what we have accepted as a maximum permissible level, meaning that we can assume that some people will be 3 times the average value, and still not exceed what we have set as a maximum permissible level.

If we take Dr. Machta's number of 70 sunshine units which is his best or average number—is it not—for this?

Senator ANDERSON. The lowest number.

Dr. LANGHAM. The lowest number. Then our present test rates would not allow a factor of three safety. If we take his most pessimistic guess, then our present test rates could not be permitted.

Senator ANDERSON. Actually, Dr. Neuman's testimony of 2.2 megatons of fission products to be somewhat safe—I hope I do not misstate you, Doctor—would indicate also we would be a little bit above what he regarded as a safe level. I should not have said it. Maybe you should.

Dr. NEUMAN. I made two calculations, one using the MPC of 50, and one using the MPC of 100. Using the 100 value, I came out with a maximum equilibrium test rate of 4.4 megatons of fission products injected annually into the stratosphere. But in that calculation I assumed Campbell's model, and 20 percent overall decay due to the reservoir, and I used a factor only of 2 to take care of the variation in individuals, but I used a factor of 3 for nonuniformity.

So, in a sense, my calculation really anticipated Dr. Machta's, although I did not know I was anticipating it at the time I made it.

Senator ANDERSON. It is very interesting that the two worked out as they did. Mr. Hollister.

Mr. HOLLISTER. I would like to introduce for the record an article by Dr. Charles I. Campbell, entitled "Radiostrontium Fallout from Continuing Nuclear Tests."

(The matter referred to is as follows:)

[Reprinted from Science, vol. 124, November 2, 1956]

RADIOSTRONTIUM FALLOUT FROM CONTINUING NUCLEAR TESTS

In spite of widespread comment on the problem of the fallout of radiostrontium from testing thermoneuclear weapons, confusion persists in the public mind and perhaps among many of the readers of Science as well regarding the relationship of Sr–90 accumulation on the ground to such factors as assumed mean storage time in the stratosphere and the rate of testing of thermonuclear weapons. Libby's recently published report on the AEC's studies of the Sr–90 problem (1) was not addressed to the effects of continuing weapons tests. Yet his conclusions have recently been quoted in the press as if they were valid if tests continue provided only that test rates remain unchanged.

Libby's analysis considered essentially the question whether nuclear weapons tests to date may have committed us already to an intolerable accumulation of Sr–90. Happily they have not. Speaking to that point, the meteorologists on the National Academy of Sciences study of the biological effects of atomic radiation stated, "At present, the amount of Sr–90 in the stratosphere from nuclear weapon tests is far too small to approach maximum permissible concentration even if it all were to be deposited now. However, if the testing programs of the several countries producing thermonuclear weapons were to intensify, stratospheric storage time may become a critical item in terms of hazard to mankind. For this reason, a continuing program to investigate this phenomenon is needed, including actual measurements of the radioactivity in the stratosphere and improved and more representative methods of observing fallout" (2, p. 60).

The consequences of continued tests can be discussed in terms of a simple mathematical model which is generally accepted by Libby and others in this country as well as in England (3). Assume that Sr–90 is introduced at a constant rate n into the stratosphere, where it is immediately mixed uniformly over the entire glode. According to British data, mixing is evidently reasonably rapid (3, p. 11). Assume further that fallout occurs at a rate $R=kQ$, where Q is the instantaneous stratosphere storage and k is the reciprocal of the mean stratospheric storage time.

Accumulated radiostrontium on the ground, M, can then be shown to be

$$M=\frac{n}{\lambda}\left[\frac{k}{k+\lambda}+\frac{\lambda}{k+\lambda}e^{-(k+\lambda)t}-e^{-\lambda t}\right] \tag{1}$$

where λ is the radioactive decay constant of radiostrontium. If the constants are expressed in years and the rate of testing is expressed in terms of millicuries of Sr–90 per square mile of the earth's surface introduced per year into the stratosphere, M is given in terms of millicures of Sr–90 per square mile at t years. When $t=\infty$

$$M_{\max}=\frac{nk}{\lambda(k+\lambda)} \tag{2}$$

and the maximum accumulation of fallout is seen to be proportional to the test rate.

Using Libby's best estimate for the mean stratospheric storage time of 10 years and a conservative estimate of the test rate corresponding to the introducion of 2.5 mc/mi^2 yr, per year as a reasonable value for n, the maximum accumulation of radiostrontium would be about 80 mc/mi^2. Libby, *considering only the Sr–90 produced up to 1955*, predicted maximum fallout of less than one-tenth this amount. The two figures should not be confused.

It is not yet known what fraction of the total radiostrontium produced from a thermonuclear weapon reaches the stratosphere and becomes involved in the fallout process discussed here. For this reason, we do not know how to interpret available data on test rates and accumulation of Sr–90—for example, whether little has reached the stratosphere and has subsequently fallen out again relatively quickly or whether much has entered the stratosphere but has been held back by a long storage time. It cannot be said with much confidence, therefore, what rate of weapons testing would result in a given accumulation of Sr–90.

Assuming a 10-year storage time and a continuing test rate about twice that mentioned in a previous paragraph (corresponding to estimates made by Stewart, Crooks, and Fisher in the United Kingdom), the Sr–90 accumulated on the ground after about 35 years would be 80 mc/mi². This would correspond to about 0.14 MPC (maximum permissible concentration) unit in the soil. According to Libby (4), Sr–90 levels in soils are converted to levels in bones of young children at about 70 percent efficiency. This reduces the figure for levels in young children after 35 years of continuous tests to about one-tenth the permissible levels as established for *occupational* exposures. The concentration would not fall much below 0.07 MPC unit even if storage time were found to be 20 years instead of 10. Recently committees of the National Academy of Sciences (2, p. 39) and the British Medical Research Council (5, par. 281) have expressed their belief that only 0.1 MPC unit or less should be permitted for the population at large. In fact, the British report stated (5, par. 360): "So far as radioactive fallout may affect the individual, we believe that immediate consideration would be required if the concentration of radioactive strontium in bone showed signs of rising greatly beyound that corresponding to one-hundredth of the maximum permissible occupational concentration." The rate of introduction of Sr–90 into the stratosphere assumed here is close to that estimated by Libby for the past 3 years. On the assumptions made here, therefore, a long-term test program could conceivably reach or exceed the levels of Sr–90 considered safe for the whole population.

There is little reason to hope that what may be learned about storage time, k, will change this situation much. We must hope that new information may allow us to increase the maximum permissible concentration of radiostrontium in the bodies of the people of the world, that means may be found to decrease the input of Sr–90 to the stratosphere from tests, or, preferably, that a new attitude among the people of the world will permit us to lower the test rate, n.

CHARLES I. CAMPBELL,
Route 4, Box 361–M, Vienna, Va.

REFERENCES AND NOTES

1. W. F. Libby, Proc. Natl. Acad. Sci. U. S. 42, 365 (1956).
2. Biological Effects of Atomic Radiation. Summary reports (NAS–NRC, Washington, D. C., 1956).
3. N. G. Stewart, R. N. Crooks, E. M. R. Fisher, British Atomic Energy Research Establishment (Harwell) Rept. HP/R 1701 (1955).
4. Note added in proof: In an address before the AAAS, on 12 Oct. 1956, Libby presented data indicating that the efficiency is more likely between 10 and 30 percent. He also estimated that in the past 2 years Sr–90 was introduced into the stratosphere at an average rate of 6 mc/mi² yr, or more than twice the rate assumed in the calculations of this paper. Libby's address will be published in Proceedings of the National Academy of Sciences.
5. Hazards to Man of Nuclear and Allied Radiation (Her Majesty's Stationery Office, London, 1956).

Mr. HOLLISTER. I would like to read a paragraph from Dr. Libby's speech before the American Physical Society on April 26, 1957. This is on page 23, the third paragraph.

Of course, as testing continues and more fallout occurs, the levels will rise. The strontium 90 that still resides in the stratosphere at the present will fall out according to our expectations at a rate which just about compensates for the decay of the material already deposited, so that no great additional increase from this source is to be expected from weapons fired in the past. If this testing should continue at about the same rate as it has averaged over the last 5 years, then we should at equilibrium, after an infinite time, approach a level of 8 times the present rate, since the average life of strontium 90 is 40 years. This assumes that the future testing will be conducted so as to give in each future 5-year period the same as the last 5 have. And so we would expect in the United States at that time an average human strontium 90 concentration of 20 sunshine units with the conservative factor of 20 between the topsoil concentration and the concentration in human bone, or 5 sunshine units if the factor of 80 is used. In other words, in the United States something between 5 and 20 sunshine units would be the equilibrium concentration of human bones if testing continued

indefinitely at the average rate of the past 5 years. This level would be approached only after a few decades. After 28 years the level would be half of this equilibrium value, and after another 28 years, 56 years total, from an arbitrary beginning which we have set as 1952, we would expect in the year 2008 three-fourths of the equilibrium figures. So somewhere between 4 and 15 sunshine units of strontium 90 in human bones in the United States might result from the present type of testing being continued for the next 50 years.

I would like to ask Dr. Libby how he derived the factor of 8 that would represent the levels at equilibrium as compared with the levels now.

Dr. Libby. Mr. Hollister, as you probably know, the strict derivation of this is somewhat complicated mathematically. But let me try to express it in words.

If you take a rate of testing which is equal to the average over the last 5 years, then per year you have got one-fifth of the total we have now.

Now, if you continue this indefinitely, you will accumulate a final total amount which is equal to the average lifetime of the radioactive strontium.

The average lifetime of radioactive strontium is 40 years, not 28 years. Twenty-eight years is the halflife. So the calculation is after an infinite length of time you will have 40 years' worth if no strontium decays.

The reason that you do not get more than 40 years' worth is that the decay of the strontium compensates for the new accumulation. Now, this holds for both stratosphere and for the stuff that is on the ground.

Now, actually the calculation is not strictly rigorous, in that, as Dr. Machta pointed out, and as I have pointed out, too, the strict and rigorous calculation is a bit more complicated. But, considering all of the factors that are involved here, and restricting the consideration to the United States, which was the condition of that paragraph—I am not sure that paragraph made that clear. We were talking about the United States——

Mr. Hollister. I think it would be implied in the sentence that, "So we would expect the United States," et cetera.

Dr. Libby. Yes. I consider that this factor of 8 is good enough and a good-enough approximation to the truth to give. The exact truth is not known to us.

We do not know, for example, whether after a long time the strontium 90 that lies in the soil will not act in the way that calcium acts, in that a portion of it will become unavailable to the plant. We do not know whether the strontium 90 that has been there for 5 or 10 years may not be less available to contaminate plants. Chemically, it is quite likely that this will happen. I had in mind, in using this factor of 8, to load the calculation a little bit for these factors which we have not yet known. I was trying to get a number which I think is the solidest number.

Mr. Hollister. You would agree, then, it is a loaded number?

Dr. Libby. It is loaded on the side of truth, in the side of actuality, in the direction of what is most likely to happen.

Now, it is very difficult to estimate the effect of this precipitation out, or removal from the biosphere of fallout strontium, which I think we will discover, and I already see evidence in our actual data.

Mr. HOLLISTER. Would you agree that the Campbell model is correct?

Dr. LIBBY. The Campbell model is strictly correct on the assumptions made, surely. I believe it is. I have not gone through all of the derivations in detail.

Mr. HOLLISTER. I went through the Campbell model in trying to justify this factor of 8, and using a half life of strontium 90 of 28 years, the only way I could justify this factor of 8, 8 now being the ratio of the level on the ground at infinite time, and the ratio now being 5 years after we started the constant rate of testing——

Dr. LIBBY. Right.

Mr. HOLLISTER. The only way I could justify this factor of 8 was to assume the storage time as zero.

Dr. LIBBY. That is strictly mathematically correct.

Mr. HOLLISTER. If I assume the storage time is 10 years, which I believe is sort of an average number that we have seen in the literature, this factor of 8 becomes a factor of 32 or 33.

Dr. LIBBY. That is certainly wrong. Not that your calculation is not right, Mr. Hollister, but for the reasons I have pointed out. You see, we now have on the ground about—what is it—two times as much as we have in the stratosphere, something like that, in the United States. And this is a very important consideration in your calculation. I cannot do it in my head and get the strict mathematical number. But we have to consider the biological aging of the strontium that goes into the soil. It is very difficult to know exactly what to do with this, but is seems to me that it was a reasonable thing to do to say that this aging out was about compensated for by the stratospheric—the fact that the stuff is stored in the stratosphere.

Mr. HOLLISTER. Would you say then that one of the problems here is the Campbell model is not strictly applicable?

Dr. LIBBY. Well, I would not put it quite that way. I would say that we do not know enough to make this calculation with complete certainty. But I do believe that the numbers given in the first row (on the blackboard) are just about right, and that the schedule that will be attained, that is, half of it by 1985, is just about right.

Mr. HOLLISTER. You would agree, though, that if, somehow in this loaded calculation we loaded it wrong, it is possible by virtue of the Campbell model argument that that 5 to 20 could be greater?

Dr. LIBBY. I think this is very unlikely. You see Dr. Campbell did not consider the aging, the removal of the strontium 90 from the biosphere. I believe that is certain to be an appreciable effect. But we do not know enough yet to know how important it is.

Mr. HOLLISTER. Would the others like to comment either on this discussion or on the numbers?

Dr. NEUMAN. I would like to say one thing. If the Campbell model is 32, this is a factor of 4, and yet you said earlier, Dr. Libby, you thought the aging was only a factor of 2. We still have a factor of 2 running loose.

Dr. LIBBY. I cannot check in my head the factor of 32 of Mr. Hollister. I do not distrust Mr. Hollister's figure, though.

Dr. MACHTA. My numbers on the board obviously stick out like a sore thumb. The reason lies partly in the factor of 8 used to increase the other values. The aging factor that Dr. Libby just indicated as

being included in the 8—when you put that in my figures, they would be reduced to the same order of magnitude as the others. So those numbers are not comparable. If you do not take into account Libby's aging factors, and if you make the assumption, for example, that half of what fell out in the United States is tropospheric, and half stratospheric, then instead of a factor of 8 you get something like 14 for the conversion of the 5-year fallout to equilibrium fallout.

Further, if you assume what came out of Castle is only 3 years old instead of 5 years old, the number 114, becomes even higher.

So this is the reason for the difference. Dr. Libby has incorporated in the 8 other factors. These and other differences should be included in the 70 and 170, and the changes will bring my answers down to the others. So as now written they are not comparable and I do not wish the record to show this comparison.

Dr. LIBBY. Should they not be still higher?

Dr. MACHTA. No, I do not think the answers will come out higher if you take into account the aging and other factors.

Mr. HOLLISTER. Do we not all agree on the discrimination factors? So there is only the question of uniformity and nonuniformity, and the upper limit would have to be higher; isn't that so.

Dr. MACHTA. The upper limit, yes.

Senator ANDERSON. I am going to recognize Mr. Durham in just a second.

Would you submit a subsequent calculation for our record that might take into consideration some of these other things? We are trying to find where the truth lies. There are all kinds of truths: There are truths, half-truths, the whole truth and nothing but the truth, so help you God.

Dr. MACHTA. I have given the amount falling on the ground. When that is converted to what goes into the bone, others are contributing, what are the conversion factors? If someone would tell me, then the numbers can be made comparable. But this has not been done, sir.

Senator ANDERSON. I just thought you might say, assuming a certain factor, which everybody seems to agree on, 8 or some other figure, is proper, your figures would come to this point. If you can do it, fine, we will appreciate it. We have appreciated your cooperation thus far so much. If you can do it, fine. If you cannot, nobody is going to put you in the penitentiary.

Mr. Durham.

Chairman DURHAM. My question may be irrelevant, but I am going to ask it anyway. Is it in general agreed by all the scientists worldwide that no strontium existed until we developed a thermonuclear weapon?

Representative COLE. How can you be sure? I am intrigued by the question, because I am curious to know.

Chairman DURHAM. I was thinking of the possibility. Of course, this is getting rather high into the problems above the stratosphere and biosphere and all the rest of the spheres, and getting up into the satellites. Is there any scientific data on the possibility this could have happened over the many, many years of existence of the satellites that some chemical reaction possibly took place, and now we are getting this down on the earth?

Dr. LIBBY. You asked two questions, Mr. Durham.

To answer the first one, well, you stated, and Mr. Cole asked if you knew your statement to be correct.

Representative COLE. No. You misunderstood. You gentlemen were nodding your heads when Mr. Durham asked if there were general agreement worldwide among the scientists that strontium 90 did not exist prior to the atomic age. You were all nodding your heads. It was because of that that I said, "How can you be sure?"

Dr. LIBBY. Yes. I will tell you.

Representative COLE. Referring to strontium 90.

Dr. LIBBY. I will tell you how we can be sure. We looked for it before the atomic bomb was invented, and we could not find it, sir. That is how we know and we can still look for it by taking old material. As a matter of fact, the other day I got a can of old tuna fish from a neighbor of mine, which was canned before July 16, 1945, and we will look for strontium 90 in that. Dr. Langham has been collecting milk samples. I do not know whether he has been successful. But just to be sure that there was no strontium 90 before the atomic bomb was first fired we have done these things.

We have looked in old bodies and there is not any. We have looked in the ground below this 2½-inch level and there is not any. This is how we know, Mr. Cole.

Chairman DURHAM. How high have you looked for it?

Dr. LIBBY. Well, sir, the highest we can look is as high as we can sample air, which is—I do not know the exact answer, but it is a few miles.

Chairman DURHAM. Will we get any data from the satellites we are building?

Dr. LIBBY. Now on your satellite question, it is a very interesting point. A few months ago we would all have given a categorical "No" to your question and said there could not be any up there. But recent evidence has indicated that we did not know as much as we thought we knew about the contribution of the sun to the earth. We learned a year ago last Washington's Birthday during the incidence of a great solar flare that matter comes to us from the sun directly, and that the so-called cosmic rays, which we have been speaking about so much, come in part from the sun. Now, if these can come from the sun, I am not so sure that a tiny little negligible amount of strontium 90 might not possibly come from the sun.

The only thing I can say now is that it must not be too large an amount, or we would find it in preatomic bones and soil, and we have not. So it is not too large, but it still might be up there in detectable quantities. I am quite sure it will not be enough to in any way upset our considerations.

Chairman DURHAM. Then we cannot convict man entirely at the present time of placing this burdensome problem upon the world?

Dr. LIBBY. I am not sure that it is there, either, Mr. Durham, but there is a possibility.

Chairman DURHAM. A possibility.

Senator ANDERSON. Off the record.

(Discussion off the record.)

Senator ANDERSON. I think if there is no violent objection, I am going to take advantage of the suggestion here and adjourn. We will not have a session this afternoon because the House is very busy with

a long series of votes, and it makes it most inconvenient for the Members to come and go. They want to be sure to hear it all.

Therefore, tomorrow morning at 10 o'clock, in the old Supreme Court room in the Capitol, the final session will be held. There will be no afternoon session.

That is the final session only as to these hearings.

The chairman of the subcommittee will undoubtedly, I think, recommend to the full committee that the hearings be adjourned at that time for the development of subsequent testimony, if it seems desirable. Is that not correct?

Representative HOLIFIELD. Yes.

Senator ANDERSON. Therefore, we will meet tomorrow morning in the old Supreme Court room, at 10 o'clock, and we will have only a morning session, because some of these witnesses who have been kind enough to stay with us for several days are going to find it necessary to leave, and we want their contributions to us as we we go along.

Mr. Durham.

Chairman DURHAM. I would like to compliment the panel this morning for a fine display of knowledge. I feel it did a lot of good here and throughout the world.

Senator ANDERSON. Just before we go, Dr. Machta, you said your figures stick out like a sore thumb?

Dr. MACHTA. Yes. I do not think they should be put on the board and quoted. They are not comparable because the proper discrimination factors have not been applied. Therefore, I do not believe the press should quote them in any way whatsoever, sir.

Senator ANDERSON. Let us ask the newspapermen present not to report the Machta figures which he desired not to be put on the board. I would not want to take advantage of any witness here. I know Dr. Langham well enough to know that he put them on with the best of intentions, and only to call attention to the fact discrimination factors is not in it.

If a hundred is permissible, and he has a 170 figure, it might look dangerous, which he does not desire to have it look. I am very anxious we do not distort the comments of a very fine scientist who has been willing to come to us and help us in this discussion.

Thank you very much.

(Below are several items of correspondence following the day's discussion and a comprehensive report on the potential hazards of strontium–90 prepared by Dr. Wright Langham and Dr. E. C. Anderson.)

[Telegram]

JUNE 7, 1957.

HAL HOLLISTER,
Joint Committee on Atomic Energy,
The Capitol, Washington, D. C.:

Regret unable to remain for further discussion at Thursday morning panel. In view of importance of question as to whether National Academy of Sciences pathology report is or is not out of date, suggest Joint Committee request answers to following questions from Drs. Warren, Brues, Neuman, Langham. To wit on page 7 of the pathology report it is stated that "there seems no reason to hesitate to allow a universal human strontium burden of one-tenth the (occupational) permissible level." That is, of 100 sunshine units. Further, on page 2–9, it is stated that "it was felt that an amount of internal radiation

sufficient to double the large population background could certainly be considered safe." That is, of approximately 40 sunshine units. On the other hand, the British Medical Council report stated on page 68 that immediate consideration would be required if the strontium concentration in the human bones showed signs of rising greatly beyond 10 sunshine units.

According to some of the testimony presented to the Joint Committee it is a reasonable although unproven inference from available data that a worldwide average strontium 90 level in human bones of 100 sunshine units could be expected to produce several million cases of leukemia and bone cancer over a few decades. In view of these comparisons, do you feel that the existing pathology reports, prepared in early 1956, gives a balance view of the strontium 90 problem and serves as a proper guide for consideration of it?

Dr. WALTER SELOVE.

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington, D. C., June 7, 1957.

Mr. H. L. HOLLISTER,
Staff Member, Joint Committee on Atomic Energy,
Congress of the United States, Washington, D. C.

DEAR MR. HOLLISTER: The point about the factor by which the soil burden of strontium 90 would be expected to increase if testing were to continue at the average rate and in the same type as during the last 5 years was discussed by yesterday's panel, and you asked why I had given the factor to be 8 in my April 26 address before the American Physical Society when this would be strictly true only if the stratospheric residence time were zero.

I told you that the factor of 8 was given because the aging of deposited strontium 90 in the ground would gradually render a considerable portion of it unavailable to plants and that the lower factor of eight was chosen to compensate for this. The aging effect is analogous to the incorporation of the strontium 90 into insoluble solids form which will not feed the plants. For example, calcium occurs in soils in two forms—the "available" and the "unavailable" and the distinction always is made in soil analysis.

The factor of increase in the absence of any aging appears to be about 11 instead of 32 as given yesterday. (The cause of the difference between our calculations is not clear to me. I did not use Dr. Campbell's equations but derived my own as given below. Perhaps some inadvertent error is involved. I would be pleased to check this out with you.)

I believe that the decrease from 11 to 8 to account for about 30 percent of the strontium 90 becoming unavailable to the plants is reasonable.

The detailed calculation is appended.

Very sincerely yours,

W. F. LIBBY.

APPENDIX

Amount of Sr^{90} in the stratosphere after t years is, y.

Amount of Sr^{90} on the ground is, x.

Since the rate of accumulation in the stratosphere, dy/dt, is the deposition rate, S, less the fallout rate, $y/10$, and the decay rate, $y/40$,

$$dy/dt = S - y(1/10 + 1/40), \text{ or}$$

$$y = S\left(\frac{1 - e^{-t(1/10+1/40)}}{1/10 + 1/40}\right).$$

Similarly,

$$x = 40(S+T)(1 - e^{-t/40}) - y.$$

In these equations, T is the rate of tropospheric fallout. In order that at five years the ratio of the amount deposited (30 mc/mi^2 in the northeastern United States) to that in the stratosphere (12 mc/mi^2) be given correctly the ratio of S to T must be 4 to 7. With this the ultimate amount on the ground relative to that deposited at 5 years is 11.

UNIVERSITY OF ROCHESTER,
Rochester, N. Y., June 12, 1957.

Dr. W. F. LIBBY,
Commissioner, United States Atomic Energy Commission,
Constitution Avenue, Washington, D. C.

DEAR DR. LIBBY: Thank you for the copy of your letter of June 7, 1957. My associates and I have gone over the equations and find them to be mathematically correct. I believe I understand the origin of the difference between your number 11 and Hollister's number 32. Hollister, perhaps, will agree. I believe he was using Campbell's equation which is derived for stratospheric fallout only. The number 32 applies to ground levels due to stratospheric fallout only not to the total on the ground at $T=5$ years.

I think, though probably obvious to most, it should be pointed out that predictions of equilibrium from these equations are based on two assumptions: first, that the overall test rate will be constant and second, that the proportion of trapospheric to stratospheric fallout will remain the same.

Is it correct to say you now predict ultimate ground levels of between 432 to 600 sunshine units (depending on the importance of your postulate of ageing) if testing continues? If so, and if you agree with me that the population average should not exceed 40 S. U. (MPC=100 S. U.), then discrimination factors of 10 to 15 would be required for the present test rate to be permissible indefinitely. A figure of 80 percent of the "present test rate" would seem to me to be a "permissible test rate" compatible with your figures.

I would appreciate your comment since my estimate, though only differing by a factor of two (4.4 Megations), presumed ideal conditions of worldwide fallout.

Sincerely yours,

WILLIAM F. NEUMAN,
Associate Professor, Pharmacology and Biochemistry.

cc: Mr. Hal Hollister.

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington, D. C., June 24, 1957.

Prof. WILLIAM F. NEUMAN,
School of Medicine and Dentistry,
The University of Rochester, Rochester 20, N. Y.

DEAR PROFESSOR NEUMAN: You are correct in finding that my equations predict ultimate ground levels of between 400 and 600 S. U. (depending on the importance of the aging factor) if testing were to continue indefinitely. A population average concentration of 40 S. U. then would correspond to discrimination factors of 10 to 15. Of course, these discrimination factors would include the ploughing factor which I mentioned the other day. Of course, one normally does not plough range land but, over a period of many years, I believe that Sr^{90} would gradually mix itself with deeper lying layers of soil. This factor should be included in the overall discrimination factor. I believe in your discussion of discrimination factors, you begin with vegetation and then consider the discrimination factor between vegetation and human bone. Do you not?

I hope we can meet again before long to discuss these problems.

Sincerely yours,

W. F. LIBBY.

cc: Mr. Hal Hollister.

UNITED STATES DEPARTMENT OF COMMERCE,
WEATHER BUREAU,
June 27, 1957.

Hon. CHET HOLIFIELD,
United States House of Representatives,
Washington, D. C.

DEAR MR. HOLIFIELD: In response to a request by Senator Anderson for information on the morning of June 6, 1957, I am enclosing the following remarks for the clarification of my testimony.

The $strontium^{90}$ bone content at equilibrium (i. e., if tests are continued at the current rate of 10 megatons of fission products per year and after a very long time) can be obtained from the predicted fallout. In my testimony, I estimated

that the fallout would lie between the 350 to 850 millicuries per square mile. These numbers were converted to strontium^{90} bone content in sunshine units by Dr. Langham to provide a range from 70 to 170 sunshine units. They were compared with numbers also estimated by Dr. Langham for other investigators which lay between 5 and 40 sunshine units. There are four reasons why the human bone content of strontium^{90} based on my fallout estimates were higher than the others:

1. The conversion from millicuries per square mile to sunshine units in average soil was a factor of 2. It should have been 1.8.
2. The conversion of fallout in 1956 to fallout after equilibrium has been established was performed by Dr. Langham through a multiplication of 8 for the numbers assigned to the other analysts. The factor of 8 should have been somewhat higher. The 8 was used to compensate for the aging of strontium^{90} in the soil and the effects of plowing.
3. Many of the other investigators (and perhaps all) use discrimination factors in converting from soil to human bone which are greater than 10, that is, gave lower bone content from the same soil. The value of 10 was used by Dr. Langham in converting from soil to human bone for my numbers.
4. The upper bound quoted in my fallout figures is offered only as an extreme. Other investigators, except for Mr. Eisenbud, did not offer upper bounds.

A review of the situation during the past several weeks has indicated two features. First, the use of comparable conversion factors yields final answers for my fallout estimates which are essentially the same as those for Dr. Libby and Mr. Eisenbud. I was unable to check the numbers assigned to the other investigators. The reason for the similarity is evident. Both Dr. Libby and Mr. Eisenbud have made their predictions for future fallout based on fallout in northeast United States where there is nonuniformity of fallout. That is, the fallout in northeast United States is over 3 times the worldwide average in 1956. Second, I am unable to provide a set of predictions for human bone content of strontium^{90} at equilibrium if the test rate is continued at about 10 megatons per year, because I cannot choose between the various conversion factors which have been quoted during the fallout hearings. For example, the discrimination factor from soil to bone has been variously estimated as lying between 8 and 80.

I feel that the uncertainty in nonuniform fallout over the globe represents only one of many of the uncertainties in the final prediction of human bone content. Other factors are: The importance of transportation of food from one area to another, the variability of calcium content of soil, the relative importance of plowing and aging, variability among people, etc. It would seem to me that some kind of a statistical treatment is necessary. The use of extremes of each of these factors does not obtain a realistic upper bound for the likely bone content of human beings.

In conclusion, I should like to repeat that the estimate of 850 millicuries per square mile, the equilibrium fallout if test rate is continued at 10 megatons per year, is offered only to show that even with the uncertainty in meteorological factors, a reasonable upper bound can be provided. It is a guess which we hope will be refined in the next few months.

Sincerely yours,

LESTER MACHTA,
Chief, Special Projects Section, Office of Meteorological Research.

UNIVERSITY OF ROCHESTER,
Rochester, N. Y., July 2, 1957.

Dr. W. F. LIBBY,
Commissioner, United States Atomic Energy Commission,
Constitution Avenue, Washington, D. C.

DEAR DR. LIBBY: This letter has as its purpose the possible resolution of some of the differences in the various calculations presented at the congressional hearings. I understand from Dr. Potts that an attempt is being made to recall at least some of the participants of the roundtable discussion for this same purpose.

Unless I am mistaken, Machta and I derived independent approaches to the problem. Eisenbud, Kulp, Langham, and you gave independent calculations of bone levels, but all of these were based on your predictions of ground levels at full equilibrium.

Machta and I were, I believe, in substantial agreement provided we used similar discrimination factors. All the values based on your ground-level estimates, however, were somewhat lower and the question is "Why?"

I have, to my own satisfaction, answered this question. Using your equations as given in your letter of June 7 to Mr. Hollister, I have substituted in the same values with one exception: I used $t=3$ years for stratospheric fallout and $t=5$ years for tropospheric fallout. After all, there was inappreciable stratospheric fallout prior to test Castle. On this basis, $T=6$ mc/mi²/yr. and $S=4.8$ mc/mi²/yr. and x, at $t=\infty$, is 396 mc/mi², or about 710 sunshine units. Using as I did an overall discrimination of 8, equilibrium bone values would then average 89 sunshine units which is 2.2 times too high (MPC=100, av.=40).

The so-called present "level of testing," said to be 10 megatons per year, should thus be reduced, on these assumptions, to about 4.5 megatons. This agrees with my testimony giving the figure of 4.4 megatons per year, whether we base our predictions on rate equations or on a presumed equilibrium. This is true, of course, only if you find my application of your equations to be acceptable. If you do agree, then, the equilibrium values of Eisenbud, Kulp, and Langham should be multiplied by 13.2/8, before they are corrected for "aging," "plowing," etc.

You asked (June 24) whether the discrimination factor of 8 covered only the area from vegetation to bone. In my testimony, I specified this to be the case. I said further that other more or less favorable factors (fallout to vegetation) may soon be discovered, but the experimental data were, in my opinion, as yet too inadequate to permit the assignment of numerical values. I then asked for further discussion of this point. In summary, then, I used 8 as an overall factor, but it admittedly is based only on the transfer from vegetation to bone.

Your comments will be appreciated.

Very truly yours,

WILLIAM F. NEUMAN,
Associate Professor, Pharmacology and Biochemistry.

cc: Mr. Hal Hollister.

UNIVERSITY OF PENNSYLVANIA,
Philadelphia, Pa., July 23, 1957.

Reference: EM: FN

Division of Biology and Medicine,
Dr. C. L. DUNHAM,
Atomic Energy Commission, Washington, D. C.

DEAR MR. DUNHAM: Thank you for your letter regarding the July 29 symposium. It is very fine that the unfinished business of the congressional hearings is being pursued by the Commission.

In addition to the problem of prediction of future levels of strontium #90, I wonder whether the plans for the meeting include discussion of the "permissible level" of strontium #90 in humans. This subject also was not fully covered in the hearings. Because of its great importance, discussion of this subject should be carried out to a more satisfactory stopping point.

Sincerely yours,

WALTER SELOVE.

cc: H. Hollister.

POTENTIAL HAZARD OF WORLD-WIDE Sr^{90} FALLOUT FROM WEAPONS TESTING

By Wright H. Langham and Ernest C. Anderson

Los Alamos Scientific Laboratory, University of California,
Los Alamos, New Mexico

1. INTRODUCTION

During the past year public attention has been increasingly focused on the potential hazard to the general population of wide-spread, low-level, radioactive fallout from nuclear weapons testing (1–5). Although a number of radioisotopes are present in the fission mixture, Sr^{90} is the major concern. It is believed to be the most important radionuclide because of its similarity to calcium, long physical and biological half-life, and high relative fission yield. These factors

lead to high incorporation in the biosphere and a long residence time in bone. General contamination will result in the bones of the population eventually reaching an equilibrium state with Sr^{90} in the biosphere. The predominance of Sr^{90} over other long-lived radioelements as a potential hazard can be deduced in part from data in Table I, which show that it is the only isotope that combines high fission yield, long half-life, high absorption rate and a low maximum permissible level (MPL). These data suggest Cs^{137} by at least an order of magnitude fission product, and its presence in people and foodstuffs has been reported (6, 7). However, for reasons not discussed here, its potential hazard to the population is believed to be less than that of Sr^{90} by at least an order of magnitude (8).

TABLE I.—*Radioelements of importance to long-term fallout problem*

Radioelement	Type radiation	Fission abundance [1]	Radiological half-life	Absorption on ingestion	MPL (μc)
		Percent		*Percent*	
Pu^{239}	α	----------	24,000 years	3×10^{-3}	0.04
Cs^{137}	β,μ	6.2	28 years	100	98
Sr^{90}–Y^{90}	β	5.1	28 years	35	1
Pm^{147}	β	2.6	3.7 years	3×10^{-2}	25
Ru-Rh^{106}	β,μ	.5	1.0 year	5×10^{-2}	4
Ce-Pr^{144}	β,μ	5.3	275 days	3×10^{-2}	1

[1] Slow neutron fission of U^{235}.

Appraisal of the potential hazard from world-wide fallout of Sr^{90} requires consideration of the extent and rate of fallout, its method of incorporation into the biosphere and the human body, present and predicted levels in soils and people, the basis of presently accepted maximum permissible body levels, and the biological significance of present and future body levels in terms of the megatons of fission weapons detonated. Information on all of these factors is somewhat inadequate at the present time. This paper is an attempt to present a general summary of the present thinking with regard to the above factors.

2. GENERAL WORLD-WIDE FALLOUT FROM BOMB TESTING OPERATIONS

Based on measurements of world-wide fallout, Libby (2, 3) proposed a mechanism by which atomic debris is disseminated throughout the world. This theory leads to three kinds of fallout, which are illustrated in Fig. 1. First is local fallout which is deposited in the immediate environs of the explosion during the first few hours. This debris consists of the large particles from the fireball and includes residues from the soil and structures which are swept into the cloud in wholly or partially vaporized state. The fraction of the total radioactivity which falls out locally depends very much on those conditions of firing which govern the amount of soil and extraneous debris incorporated in the fireball.

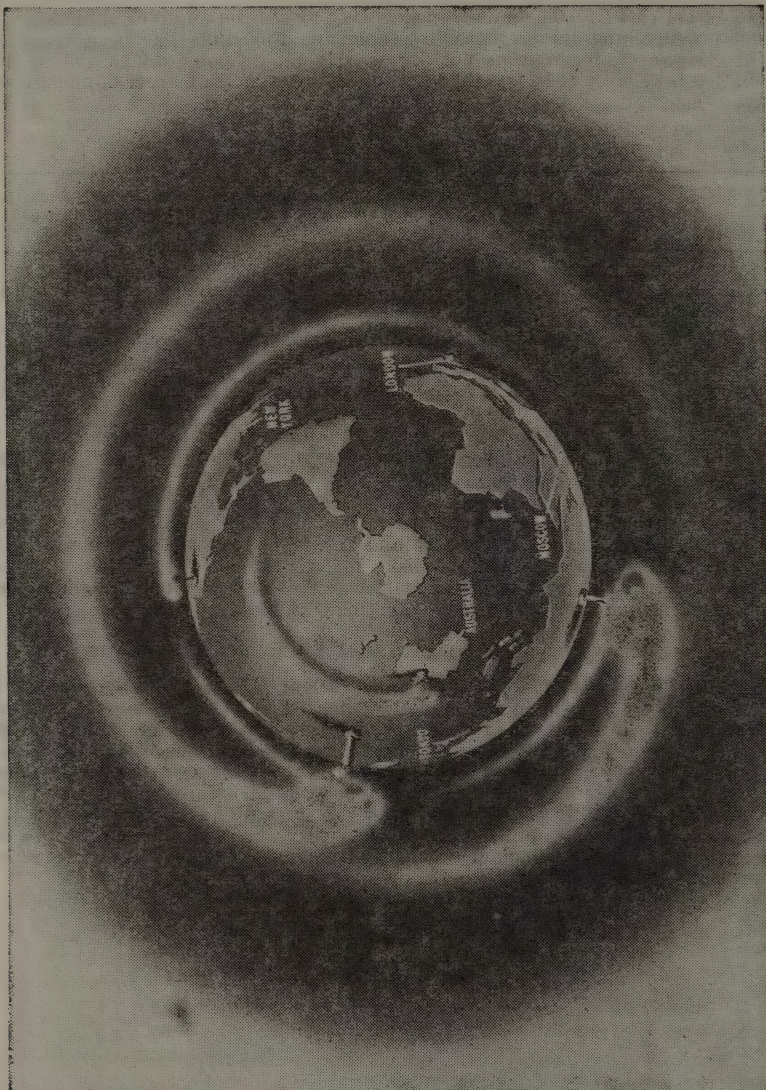


FIG. 1.—Types of radioactive fallout from weapons tests (local, tropospheric and stratospheric).

The second type (strospospheric fallout) consists of that material injected into the atmosphere below the tropopause which is not coarse enough to fall out locally. This debris is sufficiently fine that it travels great distances, circling the earth in the general latitude of the explosion, until removed from the atmosphere by rain, fog, contact with vegetation, and other meteorological and/or physical factors. The average tropospheric fallout time is estimated as 20 to 30 days. The fraction of the fallout which is in this category depends mainly on the size of the explosion and the conditions of firing. If the explosion exceeds a certain minimum size (about one megaton (MT)), the fireball will have enough energy to penetrate the tropopause carrying fission products into the stratosphere. Smaller detonations leave in the troposphere all debris not deposited locally. The fraction of the fission products from a large weapon that remains in the tropopause depends on the size of the explosion, conditions o firing, and meteorological factors.

The third type (stratospheric fallout) is composed of fission products that are carried above the tropopause and can result only from large weapons. Libby (3) has postulated that the activity is mixed rapidly throughout the stratosphere and falls back uniformly into the troposphere (see footnote page 10), where it is deposited over the earth's surface in relation to meteorological conditions. The over-all mean deposition time is estimated at from 6 to 10 years.

The above mechanism leads to a general distribution pattern of radioactivity over the surface of the earth as shown in Fig. 2. Libby's estimates (3) of Sr^{90} levels in the fall of 1956 suggest 22 mc/mi^2 for the midwestern section of the United States, 15 to 17 mc/mi^2 for similar latitudes elsewhere in the world,[1] and 3 to 4 mc/mi^2 for the rest of the world. The higher value for the upper midwestern United States is attributed to greater local and tropospheric fallout because of the proximity of our own continental test site.[2] The 15 to 17 mc/mi^2 deposited between about 60° N and 10° N latitude is due to tropospheric fallout from all shots of less than 1 MT conducted in the northern hemisphere plus stratospheric fallout from all weapons greater than 1 MT. Actually, this general picture is greatly oversimplified. Once fission products are suspended in the troposphere (either directly by the detonation or by air exchange, regardless of mechanism, between the troposphere and the stratosphere), meteorological conditions play a major role in their deposition. Libby (3) has stressed the importance of rainfall, fog, and mist. Within any major fallout area one might expect to find fluctuations in the level of surface deposition which correlate with local meteorological conditions. Higher deposition in a local area does not correlate necessarily with total precipitation but rather with frequency of rainfall.

---

[1] The latitude position and width of the north temperate tropospheric fallout belt is variable and hard to estimate. Earlier Libby mentioned 60°N–10°S (2); later he mentioned 50°N–10°N (3); and most recently he referred to 60°N–10°N (10). The area between 60°N–10°N latitude agrees better with soil data and it is assumed by the authors that the 15 to 17 mc/mi^2 applies to this area.

[2] Machta (47), in testimony given during the Open Hearings of the Joint Committee on Atomic Energy, postulated that stratospheric mixing is slow and that stratospheric distribution of fission products is still nonuniform. He feels that a major portion of the nuclear debris is still in the northern portions of the northern hemisphere, rather than uniformly spread over the entire globe or even uniformly dispersed in the northern hemisphere itself. He feels also that stratospheric movement of the fission products is largely by direct transport from west to east in the general latitude of the point of injection with very slow vertical mixing. Slow polewards circulation of stratospheric air from equatorial regions provides some mixing towards the poles. The higher concentrations of fallout in the temperate latitudes was explained on the basis of air exchange between the troposphere and stratosphere through the break in the tropopause frequently found in the vicinity of the jet stream. A large part of the higher concentration of Sr^{90} found in the northern part of the United States may result from such uneven stratospheric leakage instead of the proximity of the Nevada Test Site. In either case, the general distribution of Sr^{90} is relatively the same qualitatively. Quantitatively, Machta's model predicts a greater degree of nonuniformity of fallout over the earth with higher concentrations in the temperate latitudes.

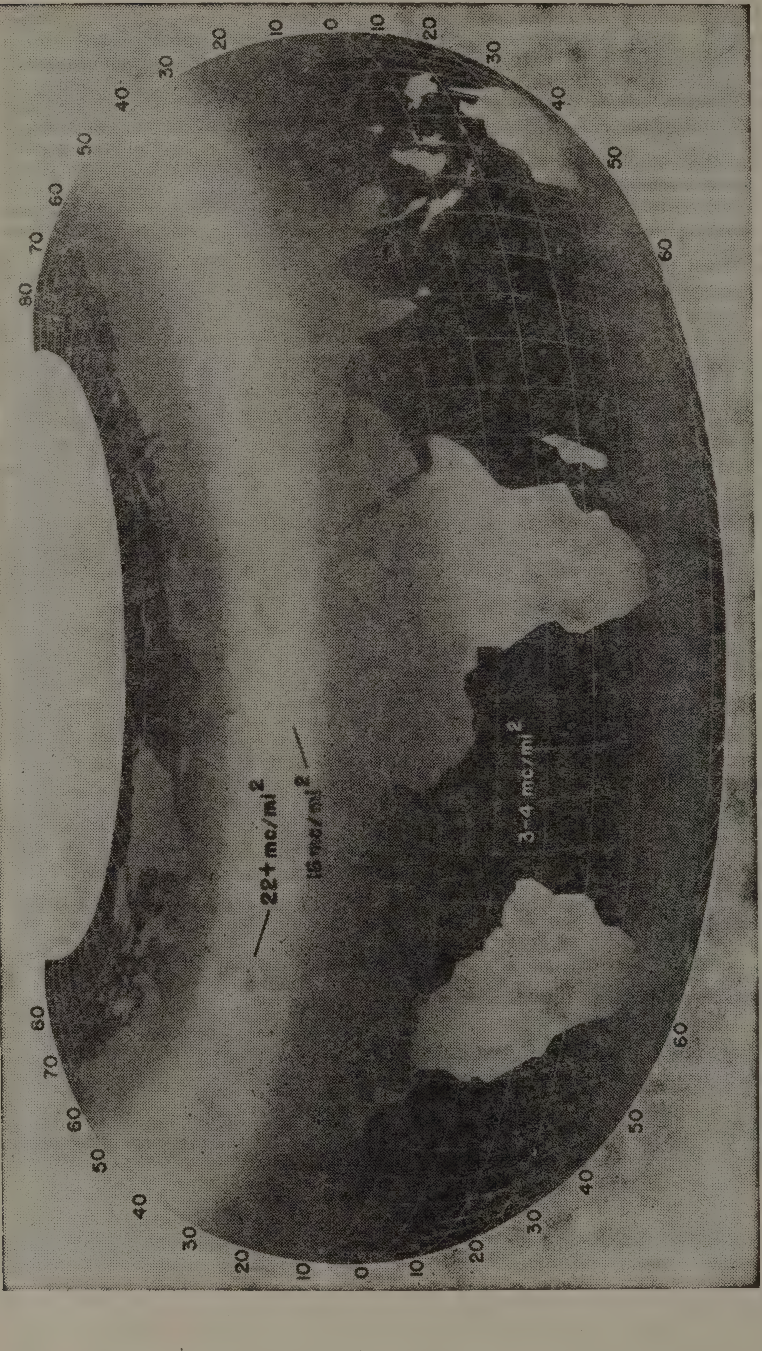


FIGURE 2.—General levels of world-wide fallout deposition.

3. PRESENT AND PREDICTED MAXIMUM LEVELS OF SURFACE DEPOSITION

Libby (3) estimated that the stratospheric reservoir (in the fall of 1956) contained the products of about 24 megatons of fission. One megaton of fission results in the formation of enough Sr^{90} to give a surface deposition of 0.5 mc/mi^2 if uniformly distributed over the entire surface of the earth. If all material presently in the stratospheric reservoir were deposited instantaneously and uniformly over the earth, present values would be increased by 12 mc/mi^2, and the maximum surface deposition of Sr^{90} would result. Maximum deposition, however, will not occur because of the relatively long average stratospheric storage time (6 to 10 years), which will allow some of the strontium to decay before deposition. Figure 3 shows, however, that the predicted maximum level is not highly dependent on the mean time of fallout. Although British investigators (9) appear to favor a fallout half-time of about 5 years, Libby (2) has chosen to use a value of 7, which corresponds to a mean time of about 10 years. With a mean time of 10 years, the maximum predicted level of Sr^{90} surface contamination from fission products alreadmy present should occur in about 1975. Table II shows the estimtaed present levels (October 1956 (3)) and the maximum predicted levels that might be expected at that time.

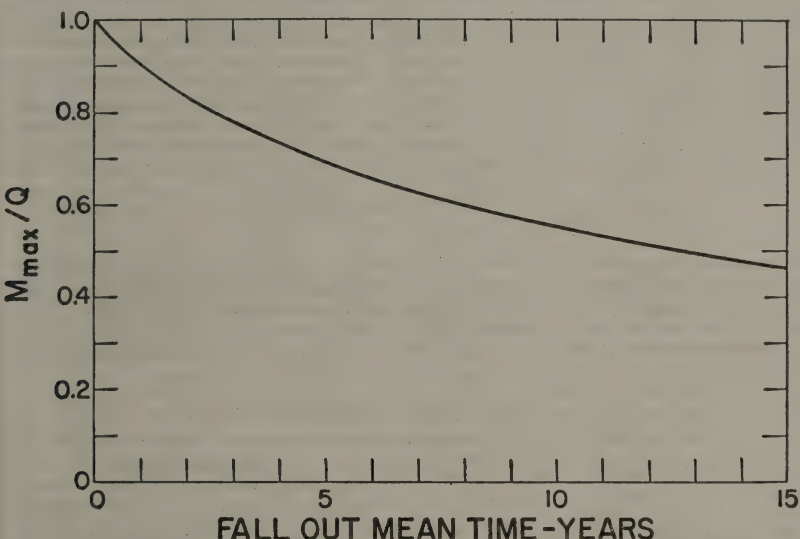


FIGURE 3. Dependence of maximum level of deposition on mean time of fallout.

TABLE II.—*Present and predicted maximum levels of Sr^{90} surface deposition* [1]

Area	Level October 1956 (mc/mi^2)	Maximum level 1975 (mc/mi^2)
Midwestern United States	22	29
Between 60° N. and 10° N. latitude	16	23
Rest of world	3.6	10
World average	[2] 8	[2] 15

[1] Assuming products of 24 MT fission in the stratosphere Jan. 1, 1957, and a fallout mean time of 10 years.
[2] Calculated by weighting for respective areas, taking 35 percent of earth's surface as lying between 60° N. and 10° N. latitude.

As stated previously, these figures assume uniform world-wide distribution of the material now in the stratospheric reservoir and no more weapon tests. Under these conditions, the area in the midwestern United States would be expected to reach a level of about 29 mc/mi^2. The area between 60°N and 10°N latitude may reach about 23 mc/mi^2, and the rest of the world may reach a

level of about 10 mc/mi^2. These values are general levels only, assuming uniform distribution within the respective areas. Local meteorological conditions will produce nonuniformities within these general regions. Recent data (10) suggest that some areas of the United States (South Dakota, Iowa, Michigan, New York) already may have deposition levels of about 29 mc/mi^2 (January 1957).

4. INCORPORATION OF SR^{90} INTO THE BIOSPHERE

When Sr^{90} falls upon the earth's surface, it is taken into plants through the root system in relation to the available calcium in the soil. That which settles directly on vegetation may remain as surface contamination, or a part of it may enter the plant through foliate absorption. When plants are eaten by animals, Sr^{90} deposited directly on the surface or incorporated in the plant (by foliate absorption or from the soil) is absorbed by the animal along with calcium. When plant and animal products (e. g., milk) are eaten by man, the Sr^{90} they contain becomes incorporated with his body calcium and deposits predominantly in the bone.

It is reasonable to assume that strontium may be discriminated against with respect to calcium in passing along the ecological chain. For example, the Sr^{90}/Ca ratio in the bones of people may be expected to be lower than the Sr^{90}/Ca ratio in the soil, which is the beginning of man's food chain.

Information regarding Sr^{90} in relation to man and his environment may be obtained from data on stable strontium. Turekian and Kulp have reported stable strontium to calcium ratios in human bone (11) and in sedimentary and igneous rocks (12). If it is assumed that the average stable strontium to available calcium ratio in the world's soils is essentially equal to that of the rocks from which they are formed, the over-all discrimination ratio (OR) against strontium over calcium in passing along the ecological chain from soils to bone

$$\text{is about } 0.1, \left[\text{i.e., } \frac{(Sr^s/Ca)_{bone}}{(Sr^s/Ca)_{soil}} = \sim 0.1\right].$$

Data on stable strontium content of human bone ash were reported by Hodges *et al* (13) and show conclusively that, under equilibrium conditions, stable strontium is equally distributed throughout the skeleton (Table III). Their results were confirmed by others (11, 14), and leave no doubt but that man's bones will eventually come into equilibrium with the Sr^{90} contamination in his environment.

TABLE III.—*Stable strontium content of human bones*

Sample	Sr in bone ash (percent)			
	Parietal	Vertebra	Rib	Femur
Fetal [1]	0.016	0.016	0.017	0.017
All ages [2]	0.023	0.022	0.022	0.022
1914 cadavers	0.027	------------	0.027	0.025

[1] Fetal bones showed range of 0.015 to 0.019 percent.
[2] All-age group showed no significant increase with age when analyses were compared in 5 age groups.

Alexander, Nasbaum and MacDonald (15) obtained excellent data on the discrimination against strontium over calcium in going from plants to milk. They compared the stable strontium to calcium ratio of cows' milk with that of the feed the cows consumed and found that $\frac{(Sr^8/Ca)\,milk}{(Sr^8/Ca)\,feed} = 0.13$. These authors (16) also studied the relative uptake of stable strontium and calcium from the diet by a variety of rodents, including rabbits and kangaroo rats from the Nevada desert. They found an average $\frac{(Sr^8/Ca)\,bone}{(Sr^8/Ca)\,food} = 0.24$. They suggested that this retention ratio might be used to predict the skeletal uptake of Sr^{90} by humans, through continued consumption of contaminated food.

Attempts are being made also to determine the over-all Sr^{90} discrimination ratio in going from soils to human bone by radioactive tracer studies of the discrimination factors (DF) that occur at the various steps along the ecological chain.

The discrimination factor most difficult to establish is the one from soils to plants (DF_1). It is dependent, among other things, on type of soil, available soil calcium, type of plant and perhaps on rainfall, all of which may vary greatly with geographic location. Menzel (17) obtained a soil-to-plant discrimination factor $\frac{(Sr^{90}/Ca)\,plant}{(Sr^{90}/Ca)\,soil}=0.7$, for four widely different soil types using both radiolactive and stable strontium. Larson (18) and Bowen and Dymond (19) have obtained comparable values.

Comar (20) has determined the discrimination factor from plants to milk (DF_2) using Sr^{85} and Ca^{45} and found that $\frac{(Sr/Ca)\,milk}{(Sr/Ca)\,plants}=0.14$, which is in good agreement with the value of 0.13 obtained from stable strontium data (16). Comar also studied the discrimination factor (DF_3) in going from milk to human bone following single and multiple feeding and found the radio $\frac{(Sr/Ca)\,bone}{(Sr/Ca)\,milk}=0.5$.

Experiments by Laszlo (21) on the discrimination factor (DF_4) from plants directly to human bone gave a value for $\frac{(Sr/Ca)\,bone}{(Sr/Ca)\,plant}=0.25$, which agrees with radioisotope studies in rats conducted by Comar (22) and with stable strontium data on rodents (16).

The over-all ratio ($OR_{bone-soil}$) in going from soil to human bone via the diet may be estimated from the various discrimination factors and the fraction of dietary calcium derived from diary products and directly from other sources. The Department of Agriculture, on the basis of United States retail sales, has estimated that 65 percent of the dietary calcium for all ages comes from dairy products and 35 percent from other sources. On this basis, $OR=(0.65\times DF_1\times DF_2\times DF_3)+(0.35\times DF_1\times DF_4) = (0.65\times 0.7\times 0.13\times 0.5)+(0.35\times 0.7\times 0.25=0.09$. The above value is in agreement with the crude estimate made from stable strontium considerations, and represents a reasonable general average of the values derived at a recent Washington Conference on "Deposition and Retention of Ingested Sr^{90} in the Skeleton" (23). This Committee reported also that the ($OR_{bone-diet}$) for a six-month-old child might vary from 0.041 to 0.13, which would give an average value for ($OR_{bone-soil}$) of 0.06. The above information on the over-all discrimination factor against strontium during passage up the ecological chain from soils to human bone via the diet is summarized in Figure. 4 and may be used to estimate the average present and future maximum Sr^{90} levels in bone as a result of weapons tests to date. In so doing, however, it must be re-emphasized that these values are for *ecological* discrimination and apply only to discrimination against Sr^{90} in progressing up the life cycle. An ecological discrimination factor automatically assumes that the calcium and strontium are uniformly mixed in soil and are equally available to the depth of the plant feeding zone. No allowance is made for direct foliar absorption of Sr^{90}, for its dilution with a greater reservoir of available soil calcium through plowing, or for the possibility that it may become less available with time through soil binding and leaching. Much of the disagreement between the over-all discrimination factors reported by Libby (10) and those given above is more apparent than real, because of failure to make clear distinction between over-all discrimination and over-all *ecological* discrimination.

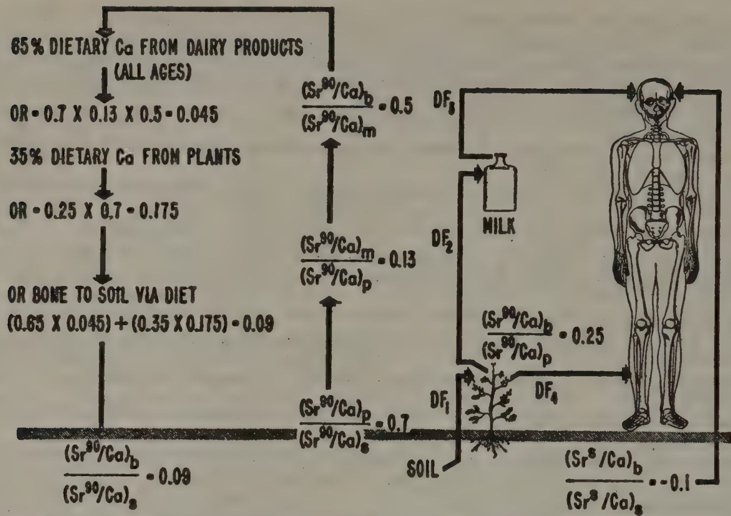


FIGURE 4. Discrimination against strontium with respect to calcium in passing up the food cycle from soil to man.

5. PREDICTED PRESENT AND FUTURE SR^{90} AVERAGE MAXIMUM LEVELS IN BONE

Sr^{90} content of the bones of young children at the time of maximum biospheric contamination is assumed to be of major concern because children are believed to be more sensitive to radiation and their entire skeletons will be formed under steady state conditions with the Sr^{90} contamination of the environment.

From the present and predicted maximum Sr^{90} surface deposition levels (Table II) and the Sr^{90} soil-to-bone discrimination ratios derived in the previous section, present and future average maximum Sr^{90} levels in bone can be predicted.

Assuming an average of 20 g available Ca/ft^2 of soil to a depth of 2½ in. (2), 1 mc of Sr^{90}/mi^2 is equivalent to 1.8 $\mu\mu$c/g available calcium. Multiplication of the Sr^{90} surface deposition levels in Table II by 1.8 gives the specific activity of the available soil calcium. The specific activity of the soil calcium times the Sr^{90} discrimination ratio of 0.06 (for six-month-old child) should give the average maximum specific activity (in $\mu\mu$c/g) of the bone calcium of young children under steady state conditions. Multiplication of the specific activity of the available soil calcium by the Sr^{90} discrimination ratio of 0.09 should give the average maximum specific activity of calcium laid down in the adult skeleton through exchange and bone remodeling during the period of environmental contamination. Present and future average maximum Sr^{90} levels in the skeletal calcium of young children at equilibrium and in newly formed adult bone calculated in the above manner are given in Table IV. As the diet of children changes, they may be expected to approach a Sr^{90} equilibrium level comparable to the level predicted for newly formed adult bone.

TABLE IV.—*Predicted present and future average maximum Sr^{90} levels in bone*

Area	Predicted maximum, fall 1956		Predicted maximum, 1975	
	Children at equilibrium [1] ($\mu\mu$c/g Ca)	New adult bone [2] ($\mu\mu$c/g Ca)	Children at equilibrium [1] ($\mu\mu$c/g Ca)	New adult bone [2] ($\mu\mu$c/g Ca)
Midwest United States	2.4	3.6	3.1	4.7
60° N. to 10° N. latitude	1.7	2.6	2.5	3.7
Rest of world	0.4	0.6	1.1	1.6
World average	0.9	1.3	1.6	2.4

[1] Assuming steady state conditions, 20 g available soil Ca/ft^2 of soil to depth of 2½ in. and ($OR_{bone-soil}$) = 0.06.

[2] Specific activity of calcium of new bone formed by exchange plus skeletal remodeling, assuming $OR_{bone-soil}$) = 0.09.

These data suggest a present average maximum equilibrium level of 1.7 $\mu\mu c/g$ Ca in bones of young children and 2.6 $\mu\mu c/g$ Ca in newly formed adult bone in the world population belt between 60°N and 10°N latitude. Calculation of average maximum equilibrium levels in about 1975 from the predicted soil levels gives 2.5 and 3.7 $\mu\mu c/g$ Ca for young children and adult bone, respectively, assuming no more detonations after Operation Redwing in the summer of 1956.

The data in Table IV are subject to the uncertainties in predicted present maximum levels of surface deposition and to the uncertainties involved in the derivations of the bone-to-soil discrimination ratios. The greatest uncertainty in the values is probably due to their dependence on available soil calcium with which the Sr^{90} is mixed.[8] Available soil calcium may vary within the United States from about 1 to 100 g/ft^2 to a depth of 2½ in. The relative Sr^{90} uptake would be higher in areas with abnormally low available soil calcium. The available calcium with which the Sr^{90} is actually mixed is dependent also on the average depth of the feeding zone of all the various types of plants responsible for the introduction of calcium into man's food chain.

Derivations of the discrimination ratios assume also that average infant and "all-age" diets of the world population are comparable to those of the United States and assume Sr^{90} is in equilibrium in the soil and make no allowance for the fraction entering the food chain through direct fallout on vegetation.

Kulp *et al.* (24) recently reported Sr^{90} analyses of 484 bone samples from persons of all ages collected at 17 stations in a worldwide sampling network. Most of these samples came from the area between 60°N and 10°N latitude, and the majority were collected during 1955 and the spring of 1956. The average Sr^{90} value for all ages was 0.12 $\mu\mu c/g$ Ca. A few results were ten times the average and a definite age effect was observed. The bones of young children showed Sr^{90} values three to four times the average, which was attributed to the greater portion of active bone in children. The average Sr^{90} content of 64 bone samples in the 0- to 4-year-age group was reported as 0.31 $\mu\mu c/g$ Ca, after dividing results from rib samples by 2 and those from vertebra by 4 to obtain an average for the total skeleton. This adjustment to obtain a skeletal average was predicated on Sr^{85} distribution studies in adult cancer patients given a single injection. The data in Table III suggest that adjustment of rib and vertebra results to obtain a skeletal average may not be entirely valid for children, in which a major portion of the skeletal calcium was laid down during the period of Sr^{90} environmental contamination. The adjustment, however, in the case of adult bone samples (in which the majority of the Sr^{90} was laid down during a contamination period relatively short compared to the age of the individual) might be justified. These adjusted values, in the case of adult bone, might be a measure of new bone formation by exchange plus skeletal remodeling during the period of contamination. On the basis of these data, it should be possible to postulate an internally consistent model for Sr^{90} deposition in the skeleton of persons of all ages, taking into consideration the rate of increase of environmental contamination and the rate of calcium deposition by skeletal growth and bone remodeling plus exchange as a function of age. Figure 5, derived from data given by Mitchell (25), shows the total deposition of skeletal calcium in males as a function of age and their rate of skeletal accretion in per cent of calcium deposited per year. This figure also shows the rate of increase in integrated Sr^{90} fallout in the Chicago milk-shed area from 1953 through 1957 and suggests a doubling time of about one year (3).

Using the rate of skeletal accretion (Table V, Fig. 5), increase in integrated fallout and Kulp's average Sr^{90} bone values for the various age groups, it should be possible to calculate the maximum average equilibrium level of Sr^{90} as of the fall of 1955. The specific activity of the calcium in the bones of children should depend on the fraction of the skeletal calcium laid down by all mechanisms during the period of environmental contamination, and the specific activity of the bone calcium of adults should depend on the per cent of skeletal calcium equilibrated during the same period by new bone formation through bone remodeling plus exchange.

---

[8] The assumption of 2½ in. as the effective depth for Sr^{90} is subject to great uncertainty. Plowing will result in mixing to a depth of about 6 in., and no allowance is made for the possibility that some of the Sr^{90} may become unavailable to plants (as is some of the soil calcium) and over long periods of time some may be removed from the soil by leaching.

TABLE V.—*Yearly accretion of skeletal calcium in males*

Age (years)	Calcium in total skeleton [1] (g)	Increase per year (g)	Fraction of equilibrium Sr^{90}/Ca	Age (years)	Calcium in total skeleton [1] (g)	Increase per year (g)	Fraction of equilibrium Sr^{90}/Ca
0	28		[2] 0.50	13	624	85	0.23
1	100	72	0.79	14	715	91	0.22
2	147	47	0.59	15	806	91	0.21
3	179	82	0.42	16	894	88	0.19
4	201	22	0.29	17	973	79	0.16
5	219	18	0.20	18	1035	62	0.13
6	239	20	0.16	19	1073	38	0.09
7	264	26	0.16	20	1078	5	0.045
8	297	83	0.18	21	1078	0	0.019
9	341	44	0.20	22	1078	0	0.006
10	396	55	0.22	23	1078	0	0.001
11	463	67	0.23	24	1078	0	0.000
12	539	73	0.24				

[1] From reference (25).
[2] Assuming 50 percent fetal protection (26).

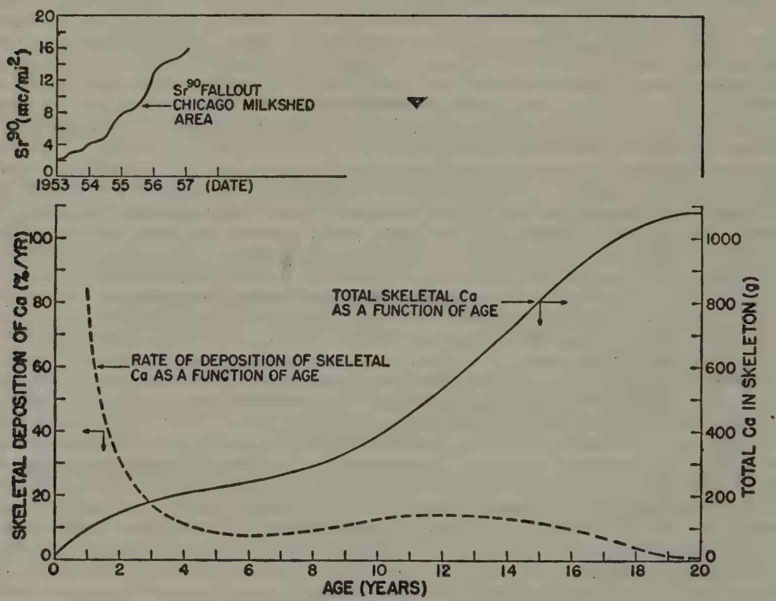


FIGURE 5. Rate of accretion of skeletal calcium in relation to rate of environmental contamination.

It is assumed that each yearly increment of skeletal growth will contain Sr^{90} at a concentration corresponding to the Sr^{90} build-up in the biosphere for that year. For a first approximation, the skeleton will be regarded as a unit and the Sr^{90} burden averaged over the entire skeleton. As better data are available, it will be profiable to consider the individual bones separately, both in terms of their Sr^{90} burden and their radiosensitivity.

Calculated values for the apparent fraction of equilibrium Sr^{90}/Ca ratio as a function of age, based on skeletal growth rate alone and a yearly doubling time of the Sr^{90} level, are given in the last column of Table V and are shown by the solid curve of Fig. 6. The method of calculation is best explained by an example. For an eight-year-old skeleton (Table V) in 1956 the last 33 g of calcium contain an equilibrium concentration C of strontium, and thus a total amount of strontium equal to 33 times C. The previous year's deposition of 26 g would have been formed with a concentration C/2 of strontium and would con-

tribute 13 C. The fifth and fourth years' growth would incorporate 20 C/4 and 18 C/8 units of strontium, respectively. The total strontium in the skeleton at age eight is, therefore, (33+13+5+2.5) C, or 53 C. (A four-year cut-off is used since large-scale testing began in 1952. Actually, the series is converging so rapidly that the cut-off has little effect.) If the entire skeleton had been in equilibrium with the Sr^{90} level of the environment in the eighth year, it would have contained 297 C units of Sr^{90}. The fraction of equilibrium is, therefore, 53 C/297 C, or 0.18.

The points in Fig. 6 represent Kulp's unadjusted values for subjects under 20 years of age and his adjusted data for adults normalized to the 0- to 4-year age group as representing 59 per cent of equilibrium Sr^{90} concentration.

At age 24 (4 years beyond the age at which skeletal growth stops) these data show that about 10 per cent of the skeletal calcium was involved in bone remodeling plus exchange during the period of environmental contamination. The data further suggest that the amount of calcium involved decreases with age, which is in keeping qualitatively with classic concepts of bone physiology. If a similar fraction of the skeletal calcium of growing subjects is involved in exchange plus remodeling, then the Sr^{90} levels in children would be proportionally higher than the curve based on skeletal calcium accretion alone. This indeed appears to be the case and indicates that the major factors have been considered in constructing the model.

If the fraction of remodeling and exchange for children's bones is similar to that observed for adults, then the accretion curve below age 20 should be raised proportionately, to give the over-all apparent fraction of equilibrium Sr^{90}/Ca ratio as a function of age (dashed line, Fig. 6). This curve permits the use of adequate bone data from any age group to predict the average maximum equilibrium Sr^{90} bone level and indicates an average maximum equilibrium level of 0.9 $\mu\mu c/g$ Ca at the end of 1955.

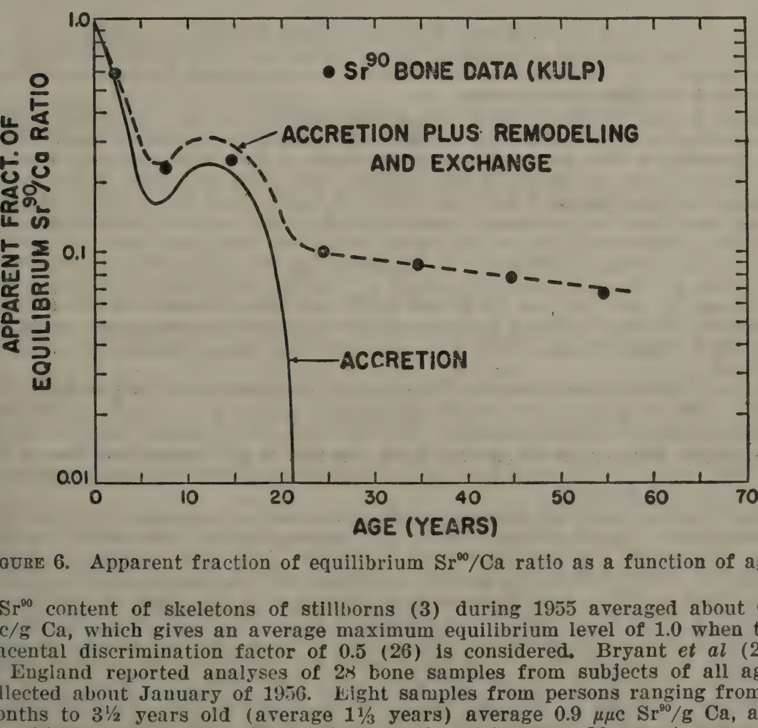


FIGURE 6. Apparent fraction of equilibrium Sr^{90}/Ca ratio as a function of age.

Sr^{90} content of skeletons of stillborns (3) during 1955 averaged about 0.5 $\mu\mu c/g$ Ca, which gives an average maximum equilibrium level of 1.0 when the placental discrimination factor of 0.5 (26) is considered. Bryant *et al* (27) in England reported analyses of 28 bone samples from subjects of all ages collected about January of 1956. Eight samples from persons ranging from 3 months to 3½ years old (average 1⅓ years) average 0.9 $\mu\mu c$ Sr^{90}/g Ca, and 11 subjects ranging from 20 to 65 years of age (average 36 years) averaged 0.07 $\mu\mu c$ Sr^{90}/g Ca. after dividing all rib results by 2. The predicted average maximum Sr^{90} equilibrium level about January 1956, based on these age groups, is 1.0 and 0.9 $\mu\mu c/g$, Ca, respectively.

Since the Sr^{90} environmental contamination level continued to rise during 1956, the predicted average maximum Sr^{90} equilibrium level of new bone as of the first of 1957 is about 1.8 $\mu\mu c$/g Ca, which agrees quite favorably with the value of 1.7 for October 1956 derived from ecological discrimination factors (Table IV). Extrapolation to 1975, assuming no more weapons tests, gives a maximum average equilibrium level in young children of 2.6 $\mu\mu c$/g Ca. If it is assumed that 1.8 $\mu\mu c$ Sr^{90}/g Ca represents present equilibrium conditions for the area between 60° N and 10° N latitude, values for the upper midwestern and eastern United States and the rest of the world can be calculated from the present and predicted average maximum levels of Sr^{90} surface deposition given in Table II. Results calculated in this way are compared in Table VI with young children's equilibrium values (Table IV) derived from ecological discrimination factors.

TABLE VI.—*Comparison of average maximum Sr^{90} equilibrium levels in skeletons of young children, derived from bone equilibrium and ecological discrimination considerations*

Area	Predicted maximum 1957		Predicted maximum 1975	
	Bone data ($\mu\mu c$/g Ca)	Ecological data ($\mu\mu c$/g Ca)	Bone data ($\mu\mu c$/g Ca)	Ecological data ($\mu\mu c$/g Ca)
Upper Midwestern and Eastern United States	2.5	2.4	3.2	3.1
60° N. to 10° N. Latitude	1.8	1.7	2.6	2.5
Rest of world	0.4	0.4	1.1	1.1
World average	0.9	0.9	1.7	1.6

These data show good agreement between the two methods of estimation.

Other observers have estimated average maximum Sr^{90} equilibrium levels in bone usually by methods that involved surface deposition levels and various ecological discrimination factors.

Libby first estimated the maximum equilibrium level in the United States and Europe in about 1970 at about 11 $\mu\mu c$/g Ca (2). This estimate was based on the assumption that bone calcium would reach a Sr^{90} level approximately 70 per cent of that of available soil calcium. He later estimated a maximum average in the 1970's of 4 to 10 $\mu\mu c$/g Ca (3) on the basis that calcium of bone would come into equilibrium at 10 per cent of the Sr^{90} content of available soil calcium. More recently he has estimated the average maximum bone equilibrium level in the spring of 1957 at 1.7 to 3.9 $\mu\mu c$ Sr^{90}/g Ca. The latter estimate was based on an over-all discrimination ratio of 1/13 to 1/30 for surface deposition levels of 25 mc Sr^{90}/mi^2 between 10°N and 60°N latitude. Because of the rate of decay of Sr^{90}, he concluded that the equilibrium bone concentration would be very little higher in the 1970's when the maximum soil contamination level is reached.

Kulp (24) estimated the world-wide average maximum equilibrium Sr^{90} bone level at 0.6 $\mu\mu c$/g Ca in the fall of 1955 and at 1.3 $\mu\mu c$/g Ca in about 1970, assuming no more weapons tests after the 1956 series. His estimates for the United States bone value were 0.9 $\mu\mu c$/g Ca for the fall of 1955 and about 2$\mu\mu c$/g Ca by 1970. All estimates were based on Sr^{90} surface deposition levels and ecological discrimination factors.

Eisenbud (4) made an estimate of the maximum average bone level on the assumption that bone calcium would come into equilibrium with the Sr^{90} in calcium from milk. In the summer of 1955, New York milk contained 2.5 $\mu\mu c$ Sr^{90}/g Ca, corresponding to a surface deposition level in the area of 6.5 mc/mi^2. Since he expected a maximum surface deposition in this area of about 21.5 mc/mi^2 in about 1970 when the stratospheric inventory of Sr^{90} is deposited, he estimated an average maximum bone level of 8.3 $\mu\mu c$/g Ca. Admittedly, his estimate was pessimistic in that it ignored the possibility of transient high Sr^{90} values in milk resulting from cows eating fresh fallout on the surface of plants and assumed no discrimination between strontium and calcium in going from milk to bone. Introduction of the milk-to-bone discrimination factor of 0.5 lowers his estimate to 4.1 $\mu\mu c$/g Ca.

Sr^{90} data on milk samples might be used to give a general estimate of the average maximum equilibrium level in bone. Chicago (2), United Kingdom

(27), New York (4), and Turkish milk (2) samples during the fall of 1955 and spring of 1956 averaged about 2.5$\mu\mu c$ Sr^{90}/g Ca. Ignoring all factors other than the discrimination factor (DF_3) of 0.5 in going from milk to bone and the one-year doubling rate for environmental contamination, the average maximum Sr^{90} equilibrium bone level in the fall of 1956 would be 2.5 $\mu\mu c$/g Ca. Extrapolated to 1975, the value would be 3.5 $\mu\mu c$/g Ca.

The various estimates of average maximum Sr^{90} equilibrium levels show a trend toward general agreement with those based on the most recent available bone data and the latest opinions regarding ecological discrimination factors (Table VI).

The most troublesome feature of the above considerations is that all values are average maximum equilibrium levels of Sr^{90} and make no allowance for local concentrations of fallout due to meteorological factors, variations in available soil calcium, dietary patterns and habits, nutritional state of segments of the population, etc. Frequency distribution patterns have been reported for stable strontium (11), natural radium (28) and Cs^{137} (7) in man. All these nuclides show essentially normal distributions with standard deviations of about 35 per cent, which suggests that the range ($\pm$ 3σ) of Sr^{90} equilibrium bone levels as of the first of 1957 (based on an average of 1.8 $\mu\mu c$/g Ca) should lie between about 0.3 and 4 $\mu\mu c$/g Ca.

On the basis of the above distribution patterns, Libby (10) has stated (at steady state among people *living in a given locality*) only one person in about 700 will have more than twice the average Sr^{90} burden, and the chances of anyone having as much as three times the average will be about one in 20 million. Presently the Sr^{90} measurements of bone samples from subjects of all ages show a much greater scatter than indicated by a standard deviation of 35 percent. The greater scatter of the observed values is due largely to the fact that samples came from many localities and (because of the relatively short period of environmental contamination and the age dependence of Sr^{90} deposition) represent varying degrees of equilibrium conditions. As stated by Libby, the spread may be expected to decrease as equilibrium is approached, and a study of the distribution of all bone data when normalized to equilibrium according to the upper curve of Fig. 6 is underway.

Local meteorological conditions will result in increased intensity of fallout in certain localities. The worst possible situation that could come about would be for these "hot spots" to coincide with localities of low available soil calcium in which the population grew up and lived in provincial isolation. Libby (10) has considered this problem in view of the averaging which occurs in food distribution systems and has postulated that a factor of 5 encompasses the total variation due to all factors, including soil calcium deficiencies.

6. SIGNIFICANCE OF SR^{90} LEVELS IN THE POPULATION

A. Basis of Maximum Permissible Levels of Radiation

Consideration of the basis of maximum permissible levels of radiation is essential to the evaluation of the potential hazard of Sr^{90} contamination to the general population. Human experience through diagnostic and therapeutic use of X and gamma rays and extensive animal experimentation with all types of radiation have demonstrated conclusively the production of deleterious biological effects. These effects are manifest as early physiologic aging resulting in life shortening, mutations, irregularities in hematopoietic function (some of which result in increased incidence of leukemia), and specific organ or tissue changes such as cataracts of the lens of the eye and tumors of bone.

Damage from ionizing radiations may occur from radioactive isotopes deposited in the tissues and organs, as well as from radiations originating from an external source.

Occupational Exposure.—Maximum permissible levels for most radioisotopes as applied to occupational exposure are calculated on the premise that no critical organ or tissue will receive an average dose rate greater than 300 mrem per week (the maximum permissible dose rate for external whole body radiation), assuming uniform distribution of the isotope throughout the tissue or organ. It is recognized that calculations based on average dose or uniform distribution in a critical organ or tissue may lead to considerable error, since some radioisotopes are unevenly deposited. Although the average dose rate to a critical organ may be 300 mrem per week, some portions of the organ may receive considerably less than the average and others correspondingly more. This error, however, is be-

lieved to be offset at least in part by the fact that 300 mrem is considered the acceptable weekly exposure to the entire body or the blood-forming organs, and therefore may be overly conservative when applied to a small element of tissue.

The maximum permissible levels of radionuclides that localize in bone (i. e., Sr–90) are determined by direct comparison with the 0.1 μc maximum permissible burden for radium. Limited human experience has indicated conclusively that small amounts of radium fixed in the skeleton will produce osteoporosis, necrosis, and sarcoma (29). Bone changes in radium dial painters and persons who received radium therapeutically provide the basis for the value of 0.1 μc. The maximum permissible levels of other bone-seeking radioisotopes are established on the premise that the amount fixed in the bone will not result in greater probability of biological effect than that produced by 0.1 μc of fixed radium.

Derivations of formulae for the calculation of maximum permissible levels, their parameters and pertinent information on individual nuclides are given in the Handbooks of the International (8) and the National (30) Commissions on Radiological Protection. These handbooks provide the only official sources of maximum permissible levels for the various radionuclides from the standpoint of internal absorbed dose. All values presently published in the handbooks refer to continuous occupational exposure.

Nonoccupational Exposure.—Maximum permissible levels for nonoccupational exposure or exposure of a large segment of the general population were established by taking arbitrarily one-tenth of the value for working personnel (8, 30, 31).

The rationale behind a lower value for the general population is based on the numbers involved in the two groups at risk and the increased heterogeneity of the general population over that of the select working group. The latter group is composed of supposedly healthy workers (over 20 years of age), while the general population group may contain children, pregnant women, the undernourished, the sick and the old. On the assumption that frequency of response to radiation stress follows a Gaussian distribution (Fig. 7), the probability of injury of a few individuals from a specified dose increases with increase in size and heterogeneity of the group at risk.

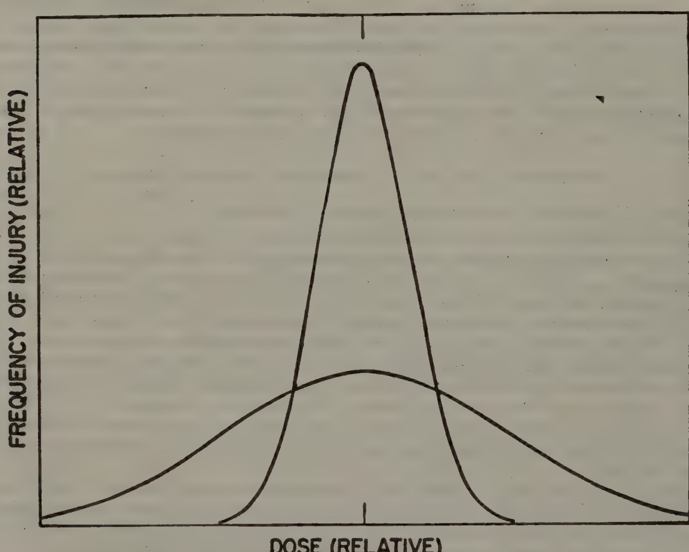


FIG. 7.—Effect of increased heterogeneity of the population on Gaussian distribution of frequency of injury as a function of dose.

The maximum permissible level of Sr^{90} for workers is set at 1 μc in the total adult skeleton, and the recommended level for the general population is set at 0.1 μc[4] (9, 32, 33). The permissible levels of radiation exposure (including that from Sr^{90}) is predicated on the assumption that chronic and/or delayed effects of radiation are threshold phenomena (Fig. 8). That is to say, there is a threshold dose below which effect rapidly becomes insignificant and above which effect increases expenentially over a limited dose range. If this is indeed the case, 100 $\mu\mu c$ Sr^{90}/g Ca must be looked upon as a true maximum permissible level and not an average value for the general population.

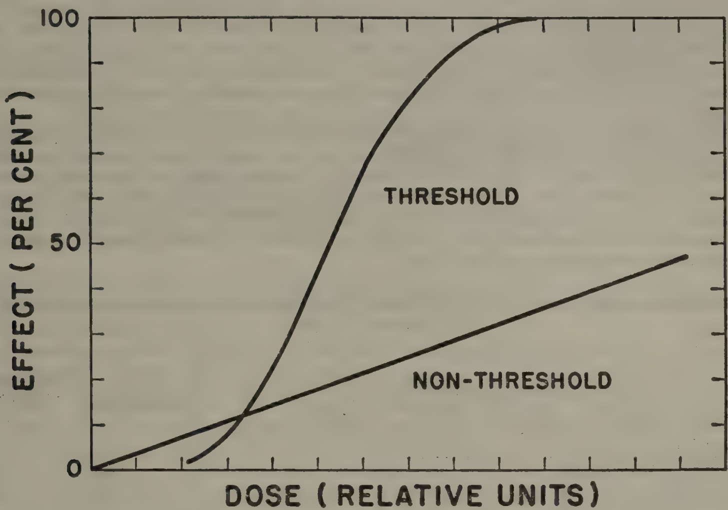


Figure 8.—Threshold versus nonthreshold effect in relation to increasing radiation dose.

Threshold versus Nonthreshold Response.—At the present time it is impossible to say whether leukemogenic and sarcogenic responses to chronic radiation dosage are threshold or nonthreshold relationships. Argument for a linear relationship between incidence of leukemia and radiation dose was presented recently by Lewis (34). His argument was based on all major sources of human data and included a consideration of the Japanese atomic bomb survivors, the British cases of X-ray treated spondylitis patients, X-ray treated cases of thymic enlargement, practicing radiologists and spontaneous incidence of leukemia in Brooklyn, New York. Radiation as a carcinogenic agent has been discussed at length by Brues (35), who stated that the relation between radiation dose and carcinogenic effect is not easy to find and a critical experiment has yet to be done which will clearly indicate, even in a single instance, what the relation is over more than a small range of dosages. While admitting that it is not known, he proposes that a threshold relationship between radiation dose and tumor incidence does exist (36).

Genetic response to external radiation indeed appears to be linear and a given increment of dose produces a corresponding equal increment of effect, regardless of position on the dosage scale (Fig. 8). If it is assumed that all chronic effects of radiation are linear, it would seem more reasonable to establish permissible levels for the general population on the basis of probability of risk averaged over the entire group. Present incidence of bone sarcoma and of leukemia averaged over the entire population is about 2 and 6 per 100,000, respectively. About 10 percent of the natural incidence of leukemia (34) (and perhaps of bone sarcoma) may be attributable to natural radiation background. If this is true, doubling the natural background dose to the bone might be expected to increase the inci-

[4] There is about 1 kg of calcium in the adult human skeleton; therefore, the MPL of Sr^{90} in the general population is equivalent to 0.1 μc Sr^{90}/kg Ca=100 mμc/kg Ca=100 $\mu\mu c$/g Ca=100 Sunshine Units (2).

dences of bone tumors and leukemia to 2.2 and 6.6 per 100,000, respectively. Such a small increase distributed through the general population may be undetectable, and 100$\mu\mu c$ Sr^{90}/g Ca (which would about double the background skeletal dose) may be regarded by some as an acceptable average maximum equilibrium for the general population.

B. Hazard from Present and Predicted Sr^{90} Levels

The significance of the general hazard of present and predicted levels of Sr^{90} in bone can be evaluated only in relation to human experience, which is indeed inadequate. Bone sarcoma has resulted from a fixed skeletal burden of 3.6 μc of pure Ra^{226}, and nondeleterious bone changes have been observed in persons having only 0.4 μc for a period of 25 years (37). Necrosis and tumors of the bone have occurred also several years after large doses of X-ray (38), and consideration of human experience with leukemogenic effects of X and gamma radiation (9, 34, 39) suggests that about 80 rads may double the incidence of leukemia.

The only other human experience with which present and predicted levels of Sr^{90} may be compared is that arising from natural background radiation. Natural background dose to the bone (during a 70-year lifetime) may vary from about 8 to 38 rem (40). The major contribution to background variation is differences in the radium levels of soils and minerals. The average natural skeletal radiation dose rate was carefully evaluated by Dudley and Evans (41) and their data are shown in Table VII.

TABLE VII.—*Average natural background radiation dose rate to the skeleton (Dudley, Evans)*

Source of radiation	Skeletal dose rate (mrem/year)	Total dose to age 70 (rem)
K^{40} (internal)	8	0.56
Ra^{226} (internal)	12	0.84
MsTh (internal)	12	0.84
RaD (internal)	12	0.84
Cosmic rays (external)	30	2.1
Local gamma rays (external)	60	4.2
Total	134	9.4

Table VIII (after Brues (42)) gives a general summary of estimated skeletal radiation doses from accepted maximum permissible levels and from present and predicted Sr^{90} burdens in relation to human experience. The maximum permissible level of Sr^{90} (100 $\mu\mu c$/g Ca) is estimated to deliver about 8 rads[5] to the skeleton during a 70-year life-time. This is comparable to the average natural background dose to the bone for the same time period and a factor of ~4 below the maximum natural background dose to which small segments of the general population may be exposed as a result of differences in altitude and natural radium content of soils and minerals. It is a factor of 40 below the lowest skeletal dose which has produced minimal nondeleterious bone changes. These data suggest that the present average maximum Sr^{90} equilibrium level in children will result in a life-time radiation dose of approximately 2 per cent of the accepted maximum permissible level for the general population. The predicted average maximum level of Sr^{90} (from bone data) in about 1975, assuming no further weapons tests, corresponds to a skeletal radiation dose of about 2.6 per cent of the maximum permissible level with a spread ($\pm 3\sigma$) of about 0.5 to 6 per cent.

The biological significance of present and predicted Sr^{90} average maximum equilibrium levels and maximum permissible levels for occupational and non-occupational exposure is summarized in Table IX.

[5] Eight rads is the calculated dose assuming incorporation to age 20 and decay to age 70 with no more incorporation. If equilibrium were maintained, the calculated skeletal dose would be about 21 rads. Since some but not all of the skeleton undergoes remodeling plus exchange, somewhere between 8 and 21 rads is probably more correct.

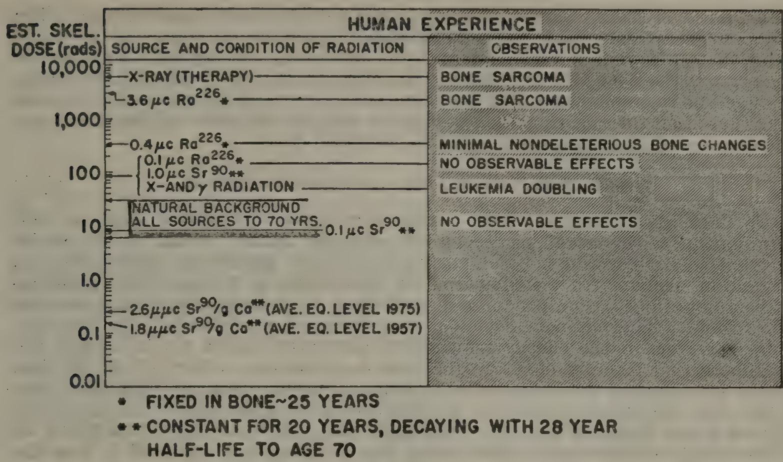


TABLE IX.—*Biological significance of present and predicted Sr^{90} average maximum equilibrium levels and maximum permissible levels for occupational and nonoccupational exposure*

Sr^{90} level	MPL nonoccupational exposure (100 $\mu\mu$ c/g)	MPL occupational exposure (1,000 $\mu\mu$ c/g)	Minimum bone changes	Minimum sarcoma dose	Leukemia doubling dose
Present (1.8 $\mu\mu$ c/g Ca)[1]	1/50	1/500	1/2,000	1/20,000	1/500
Predicted (2.6 $\mu\mu$ c/g Ca)[1]	1/40	1/400	1/1,400	1/14,000	1/360
100 $\mu\mu$ c/g (MPL nonoccupational exposure)	------------	1/10	1/40	1/400	1/10
1,000 $\mu\mu$ c/g (MPL occupational exposure)	10	------------	1/4	1/40	1

[1] Average maximum equilibrium level of Sr^{90}, probability slight that many will run more than 3 times average.

The most interesting comparison made in Table IX is that between Sr^{90} levels and the leukemia doubling dose, assuming a nonthreshold relation between incidence and radiation exposure. These data indicate that the predicted average maximum equilibrium level of Sr^{90}, assuming no more weapons tests after Operation Redwing (Fall 1956), is 1/360 of the leukemia doubling dose. Theoretically, this level is equivalent to an increase in leukemia incidence of 1.7 cases per 10 million population. The rate in some localized areas may be several times higher, but averaged over the world population of 2.6 billion, this would produce an increased lukemia burden of about 400 cases per year. If the entire world population is allowed to reach an average maximum Sr^{90} equilibrium level of 100 $\mu\mu$c/g Ca, the average increase in world's leukemia burden would be about 16,000 cases per year, or about 5 to 10 per cent (34).

The above discussion entails the assumption that Sr^{90} beta radiation induces leukemia of the bone marrow origin at the same rate (per unit of absorbed dose) as X and gamma rays. Much of the beta radiation from Sr^{90} will be absorbed in the bone and not reach the hematopoietic tissues at all. Experiments by Brues *et al* (43) suggest that Sr^{89} (half-life 55 days, $E\beta$=1.5 Mev.) administered to mice is relatively more spectacular as an osteosarcogenic agent than a leukemogenic agent. Furthermore, leukemia was not a significant finding in the radium dial painters (29, 44) or in the radium-injection cases (37).

Human data on radiation-induced osteogenic sarcoma are not adequate to provide a basis for a sarcoma doubling dose or for an estimation of the per cent of normal incidence attributable to natural background. If, however, the same assumptions used for leukemia are applied to osteogenic sarcoma (nonthreshold response, 10 per cent of normal incidence of 2 per 100,000 attributable to natural background and a doubling dose of 80 rads), the predicted average maximum Sr^{90}

equilibrium level from weapons already tested would produce an increase in the world's burden of osteogenic sarcoma of about 150 cases per year. If the world population is allowed to reach an average maximum Sr^{90} equilibrium level of 100 $\mu\mu c$/g Ca, the bone tumor incidence would be increased by about 5,000 cases.

It should be re-emphasized that the above considerations are extremely tenuous and are based on the questionable assumption that the incidence of leukemia and bona sarcoma bear a linear relationship to radiation dose.

7. SR^{90} LEVELS IN RELATION TO WEAPONS TESTING

Little data are available which permit correlation of present levels of Sr^{90} contamination with actual megatons of weapons tested. Total of all weapons tested to date would be of little value without additional information on the fraction derived from fission and on conditions of firing which influence relative amounts of fission products deposited as local, tropospheric and stratospheric fallout. Thermonuclear yield *per se* does not produce Sr^{90}; it does contribute, however, to the energy required to carry the fission products into the stratosphere and thereby effect world-wide distribution. Libby (2, 3, 10) has provided estimates of the megaton equivalents of fission products that have been injected into the stratosphere and deposited over various regions of the earth. Based on these values, Kulp (24) estimated that present levels (Fall 1956) of environmental contamination (including the stratospheric reservoir) was the result of injection of products from about 50 MT of fission yield.

By simple proportionality he estimated biospheric injection of Sr^{90} from 35,000 MT of fission would bring the average maximum Sr^{90} equilibrium bone level of the world's population up to 100 $\mu\mu c$/g Ca (the MPL for occupational exposure). He used 1.3 $\mu\mu c$ Sr^{90}/g CA as the average maximum equilibrium level for the world population in 1970, assuming no more weapons tests. Kulp did not say that the average maximum level of the world population should be allowed to reach 1000 $\mu\mu c$ Sr^{90}/g Ca,[e] but apparently tried to show the relation between megatons of fission tested to date and a MPL familiar to all (45). Libby (2, 3) also has used the occupational MPL as a reference point in discussing the hazard to the world population. This practice has led to confusion of the public and criticism of the Atomic Energy Commission (46). The data in Table X (based on various estimates of 1970 equilibrium bone levels) show the estimated megaton equivalents fission yield that may be injected into the biosphere (all at once) to bring the Sr^{90} average maximum equilibrium bone values in the United States, the northern hemispheric fallout belt and the world up to the limits set for occupational and nonoccupational exposure. The table also shows the influence of various factors for nonuniformity of distribution and uptake on the estimates for the northern hemispheric fallout belt (in which the majority of the world's population is distributed) based on the average maximum level derived from bone data. *These data are presented primarily to emphasize the principal areas of uncertainty responsible for apparent disagreements among various authorities.*

---

[e] The Sr^{90} maximum permissible level accepted by the National and International Commissions on Radiological Protection as being applicable to large segments of the population is equivalent to 100 $\mu\mu c$/g Ca.

TABLE X.—*Sr^{90} levels in relation to megaton equivalents of fission products injected into the biosphere*

Source of equilibrium estimate and area	Estimated equilibrium 1970–75 ($\mu\mu c$ Sr^{90}/g Ca)	MT required to produce average MPL	
		(1,000 $\mu\mu c$/g Ca)	(100 $\mu\mu c$/g Ca)
United States (average):			
Libby (10), Ecological data	1.7–3.9	30,000–13,000	3,000–1,300
Kulp (24), Ecological data	2	25,000	2,500
This report, Ecological data	3.1	16,000	1,600
This report, Bone data	3.2	16,000	1,600
Eisenbud (4) [1], Milk data	4.1	12,000	1,200
This report, Milk data	3.5	14,000	1,400
Area 10° N. to 60° N. latitude (average):			
This report, Ecological data	2.5	20,000	2,000
This report, Bone data	2.6	20,000	2,000
World (average):			
Kulp (24), Ecological data	1.3	38,000	3,800
This report, Bone data	1.7	30,000	3,000
This report, Ecological data	1.6	30,000	3,000
Ave. bone data (10° N. to 60° N. latitude)	2.6	20,000	2,000
Ave. × ⅕ (for nonuniformity (10))		4,000	400
Ave. × $\frac{1}{10}$ (for nonuniformity) [2]		2,000	200

[1] Eisenbud's value corrected for discrimination factor of 0.5.
[2] Indicated by spread in current bone data from all ages (24).

Inspection of the data in Table X shows a variation of about 300 in the megaton equivalents of fission products that may be injected into the biosphere, depending on whether one wishes to be ultraconservative and use the highest equilibrium bone value, the nonoccupational MPL and the largest safety factor for nonuniformity, or use the occupational MPL applied to the world average maximum bone level with no safety factor for nonuniformity. The most important point to these data is that they show that the major portion of the variaion is associated with two factors, (1) the maximum permissible level for Sr^{90} as applied to the fallout problem, and (2) the factor for nonuniformity of Sr^{90} distribution and uptake.

The most important question regarding the potential hazard of world-wide fallout to the general population is its relation to future weapons testing. If there is an upper limit to the amount of Sr^{90} that can be tolerated in the bones of the population, then the number of megaton equivalents of fission products that can be contributed per year to the biosphere by all nations must be limited.

Theoretically, the total yearly injection rate should be that amount which, at equilibrium, will not result in a significant fraction of the population exceeding the limit of safety. If a constant yearly injection rate of 1, 10 or 100 MT of fission is adhered to, in about 100 years the amount of Sr^{90} added to the environment will come into equilibrium with the rate of Sr^{90} decay, and continuation of weapons testing at that rate will result in no further increase in the average maximum equilibrium level in the bones of the population. At that time the average equilibrium bone level will be directly proportional to the yearly injection rate, i. e., if 10 or 100 megaton equivalents are injected per year, the average equilibrium bone level will be 10 and 100 times higher, respectively, than it will be if only 1 megaton equivalent is injected.

Only three unclassified reports concerning implications of future biospheric fission product injection rates have appeared. Campbell (5) mathematically related surface deposition to a constant stratospheric injection rate. His equation suggests Sr^{90} surface deposition levels may reach 30 times present values with continuation of the present rate of biospheric contamination for 100 years. His approach, however, makes no allowance for the tropospheric deposition rate. Libby (49) has developed an expression relating Sr^{90} surface deposition to a constant test rate, using a calculated ratio of rates of stratospheric to tropospheric injection of 4 to 7. Libby's calculation is based on a measured value of 30 mc Sr^{90}/mi^2 for the present cumulated fallout in the northeastern United States (one of the more highly contaminated spots in the world) and the assumption that the stratospheric reservoir presently contains the Sr^{90} from 24 MT of fision. Libby first estimated that the maximum equilibrium Sr^{90} deposition level should approach 8 times the present level (10). On re-

calculation of the ratio he obtained a value of 11 (49).[7] Using a build-up factor of 8 and assuming an ecological discrimination factor of 20 to 80 against Sr^{90} in going from soil to human bone, he estimated an average maximum bone equilibrium level of 5 to 20 $\mu\mu c$ Sr^{90}/g Ca for the population of the United States when equilibrium is reached. If the present rate is continued for 28 years, the average maximum level would reach only one-half of the above values (10). The general consensus of opinion (23) of the various specialists in the field of bone and mineral metabolism is that the discrimination factors used above are too high and that a factor of 10 to 20 is more realistic.

Stewart, Crooks and Fisher (48) estimated the concentration of Sr^{90} on the ground in the United Kingdom as 4.5 mc/km^2 on January 1, 1956, and the mean deposition rate as 2.3 mc/km^2/year. From these data the ground concentration in the U. K. as of January 1, 1957, would be about 17 mc/mi^2. They also estimated that an equilibrium value of about 500 mc/mi^2 (200 mc/km^2) of Sr^{90} may be reached in the U. K. in about 100 years if the present rate of biospheric contamination is continued. These data show a build-up factor of about 30 over present levels.

Predicted average maximum Sr^{90} bone levels, assuming continuation of the present biospheric contamination rate for about 100 years, are given in Table XI. These values were calculated from present (Fall 1956) bone equilibrium levels derived either from analyses or from ecological considerations assuming an ecological discrimination factor of 10 against Sr^{90} and Libby's (49) build-up factor of 11 for continued testing. The one British value mentioned above is given for comparison.

TABLE XI.—*Average maximum Sr^{90} bone equilibrium levels assuming continuation of past 5-year injection rate for 100 years*

Basis of estimate and area	Average maximum equilibrium bone level	
	Fall 1956 ($\mu\mu c$/g Ca)	In 100 year [1] ($\mu\mu c$/g Ca)
United States:		
Libby (10), ecological data	1.7–3.9	[2] 19–43
Kulp (24), ecological data	1.4	15
This report, ecological data	3.6	40
This report, bone data	2.5	28
This report, milk data	2.5	28
Eisenbud, milk data	4.1	45
Area 60° N. to 10° N. latitude:		
This report, ecological data	2.6	29
This report, bone data	1.8	20
Stewart, et al. (48), present soil data United Kingdom [3]	3.1	34
Stewart, et al. (48), United Kingdom soil in 100 years	----------	[4] 90
World average:		
This report, ecological data	1.3	14
This report, bone data	.9	10
Kulp (24), ecological data (1970)	1.3	14

[1] Based on, or 5MT of fission yield per year assuming testing for infinite time, an equilibrium level, with continued testing, of 11 times the present level.
[2] Libby's estimate (10) was 5 to 20 $\mu\mu c$/g Ca.
[3] Assuming an ecological discrimination factor of 10 against Sr^{90}.
[4] This value corresponds to a buildup factor of about 30.

The discrepancy between the British value and the others is immediately apparent. No details as to the basis of their estimate were given but it is possible that no consideration was given to the ratio of rates of stratospheric and tropospheric injection, since it compares favorably with the value that would be predicted from the derivations made by Campbell (5).

The data in Table XI (excluding the British value) suggest that, continued biospheric contamination at the present rate for 100 years might result in average maximum equilibrium bone levels of about 30, 25 and 12 $\mu\mu c$ Sr^{90}/g Ca for the United States, the area between 60°N–10°N latitude and the world, respectively.[8]

[7] R. K. Zeigler of LASL Theoretical Division has confirmed the calculation using Libby's assumptions.
[8] If Machta's concept of uneven stratospheric fallout is indeed the case, the average Sr 90 values for the United States and the area between 60° N–10° N latitude may be increased by about a factor of 2.

The upper limit that might be expected in the United States, assuming a factor of 5 as adequate to make allowances for non-homogeneities of Sr^{90} deposition and uptake, would approach about 150$\mu\mu c$ Sr^{90}/g Ca, or 150 percent of the accepted maximum permissible level. After 30 years of testing, average maximum equilibrium bone levels may approach one-half of the above values, which may result in an upper limit for the United States of about 75 percent of the maximum allowable level. Assuming the average yearly injection of fission products during the past five years equal to about 10MT of fission yield, it would seem that the testing of 10MT of fission per year by all nations for 30 years should probably be considered the upper limit, or 5MT of fission yield per year assuming testing for infinite time. If these are the limits of acceptable injection rate, international agreement not to exceed these levels seems desirable. Present levels of Sr^{90} contamination are due almost entirely to tests held by only two nations. Present and predicted future Sr^{90} levels, even if weapons tests are continued at the present rate for a few years, does not seem dangerous. However, indiscriminate testing of high-fission yield weapons by many nations could result in serious levels of worldwide contamination.

SUMMARY

What does the accompanying mass of technical data mean with regard to the controversy over cessation or continuation of nuclear weapons tests? Nowhere in this report has a recommendation been made either to stop or to continue testing. Such a recommendation requires a careful weighing of the importance of the nation's nuclear weapons capability in averting a nuclear war, against the probability that a few people might get leukemia or bone sarcoma or manifest a genetic abnormality who otherwise might not have done so. Therefore, the decision to stop or continue tests requires a value judgment involving knowledge of the potential seriousness of present and future threats to the national security, and whether they should or should no be stopped on the basis of moral and humanitarian principles is not readily amenable to solution by the scientific method.

The purpose of this report is to evaluate, as factually as possible from existing data, the potential hazard of Sr^{90} fallout. Evaluation of existing data supports the following general conclusions:

(1) Radioactive isotopes deposited in the bone in sufficient quantity will produce serious consequences, including bone cancer and leukemia. Present Sr^{90} levels in the bones of the population are quite low. The present average maximum equilibrium Sr^{90} radiation dose to the bones of young children is greater than that for adults and is about 2 per cent of the average dose received from unavoidable natural background radiation contributed by cosmic rays and by radium, thorium, uranium, etc., in the environment. It is about 2 per cent of the maximum permissible level adopted by the National and International Commissions on Radiological Protection as acceptable for large segments of the general population. The present Sr^{90} radiation dose to adults, averaged over the total skeleton, is about one-tenth of that to children. Because of nonuniformity of fallout and individual variations in uptake and deposition of Sr^{90} in bone, a very small number of people may accumulate a skeletal dose that will be about five times the average, and an equal number will accumulate only about one-fifth the average. Since in the stratosphere there is still some Sr^{90} from past weapons tests, the average radiation dose may continue to rise until about 1975 even if no more weapons tests are held. At that time the equilibrium level may be 3 to 4 per cent of the average natural background.

(2) If Sr^{90} contamination from weapons testing by all nations continues at the same rate as has occurred during the past five years (about 10 megaton equivalents of TNT fission yield per year), equilibrium will be reached in about 100 years. At equilibrium the amount of Sr^{90} which will disappear each year from our environment due to radioactive decay will just about equal the amount that is being produced each year. At this time continuing weapons tests will not result in any further increase in Sr^{90} in the bones of the population. Assuming a buildup factor of 11 for equilibrium levels, the average Sr^{90} radiation dose to the bones of the population of the United States is predicted to be about 30 percent of the average radiation dose from natural background, or about 30 percent of the maximum permissible level adopted by the National and International Commissions. Since a factor of 5 may be necessary to allow for nonuniformities in fallout and bone uptake, the Sr^{90} radiation dose to a few individuals may approach 150 per cent of the recommended maximum level as an upper limit. If strato-

spheric fallout is not as uniform as predicted by the Libby model, the above values may be somewhat higher. Thirty years of testing will result in an average Sr^{90} level of about one-half of the equilibrium value, which may result in a few people approaching 75 percent of the recommended maximum permissible radiation dose. On this basis, limitation of biospheric contamination by all nations to about 10 megaton equivalents of fission yield per year for 30 years, or 5 megaton equivalents per year, indefinitely, might be desirable.

(3) The existing data support the conclusion that the present rate of biospheric Sr^{90} contamination, if continued for 20 to 30 years, will not produce average maximum population bone levels that will exceed the maximum permissible levels accepted by the National and International Commissions on Radiological Protection. The data also show that many nations cannot test high fission yield weapons indiscriminately and indefinitely without running the risk of seriously exceeding these recommended levels. For this reason, international agreement to limit testing might be desirable while negotiating for agreement to stop testing altogether.

(4) The data presently available definitely show that the greatest question concerning world-wide Sr^{90} contamination concerns the decision as to the *ACCEPTABLE* maximum permissible body dose for the general population in terms of the individual and in terms of world health. The answer to this question involves moral and humanitarian principles, as well as scientific uncertainties as to the biological consequences.

Some deductions as to the worst biological consequences of Sr^{90} fallout (which excludes the genetics question) can be made by accepting two rather pessimistic assumptions, neither of which has been proved, but both of which seem conservative. The natural yearly incidence of leukemia in the United States is about six cases per 100,000 population, and the incidence of bone tumors is about two cases per 100,000. Therefore, normally there are about 10,000 cases of leukemia in the United States per year and about 3,000 cases of bone tumors. This was the natural incidence even before any radioactive fallout had occurred; so atomic bombs have had nothing to do with it. The first assumption is that any amount of radiation has a small chance of producing tumors and leukemia. That is, the assumption is made that there is no *absolutely safe* radiation dose and any amount is theoretically bad. This assumption is open to serious question and is one of the major points of disagreement among scientists. If all radiation is bad, the natural background radiation which we cannot avoid must be responsible for a fraction of the six leukemia cases and the two bone cancer cases per 100,000 that occur in the population. The second assumption is that about 10 percent of the normal incidence of bone tumors and leukemia is due to natural background radiation. There is some scientific evidence from X and gamma radiation in support of this assumption as applied to incidence of leukemia, but none for Sr^{90} radiation. There is no evidence to support its application to bone cancer. We do know, however, that there are things in our environment other than radiation which will cause bone cancer; so it certainly would not be right to attribute 100 per cent of the bone cancer incidence to natural background.

On the basis of the assumptions given above, the incidence of leukemia in the United States might increase from 10,000 cases per year to about 10,030 and the incidence of bone cancer might increase from about 3,000 to about 3,010. This suggests a total increase of 40 cases out of a population of 165 million people as the maximum biological consequence of Sr^{90} fallout from weapons tested to date *by all nations.* If the present rate of biospheric Sr^{90} contamination continues for 30 years, the biological consequence of Sr^{90} fallout may be a total increase in the United States population of about 250 cases per year of these two diseases. There is a good chance that this prediction may overestimate the population risk. The assumption that even the smallest amount of Sr^{90} deposited in bone carries a small probability of harm is seriously questioned, and there is a possibility that a threshold below which no leukemia and bone cancer will be produced does exist. Animal experiments with radioactive strontium definitely indicate that Sr^{90} may not be as bad as anticipated for producing leukemia because the beta radiation from the Sr^{90} gets absorbed in the hard bone and does not radiate the bone marrow where leukemia begins. These same experiments do suggest that production of bone tumors by Sr^{90} is about as expected on the basis of human experience with radium.

(5) The present data also indicate that much more research on both the physical and biological factors of fallout must be done if weapons tests are to be continued. This research is necessary to narrow the limits of error and uncertainty in existing data and to permit predictions to be based on facts instead of on what appears to be reasonable assumptions. More research on the biological and

medical effects of radiation and radioactive materials is essential, even to the future of the power reactor program which also produces Sr^{90} and other fission products.

Although present knowledge of the biological effects of radiation and radioactive materials is not all it should be to allow plunging ahead recklessly and without worry into all aspects of nuclear technology, it is adequate to dispel an attitude of gloom and doom. Radiation is not the only potential hazard man is facing as part of the price of living in a highly developed society nor is it the insurmountable one.

REFERENCES

1. W. F. Libby, Radioactive Fallout and Radioactive Strontium, Science *123*, No. 3199, 657–660 (April 1956).
2. W. F. Libby, Radioactive Strontium Fallout, Proc. Nat. Acad. Sci. *42*, No. 6, 365–390 (June 1956). (See p. 1468.)
3. W. F. Libby, Current Research Findings on Radioactive Fallout, Proc. Nat. Acad. Sci. *42*, 945–962 (December 1956). (See p. 1494.)
4. M. Eisenbud, Global Distribution of Radioactivity from Nuclear Detonations, with Special Reference to Strontium-90, presented at the Washington Academy of Sciences meeting, Washington, D. C. (November 15, 1956).
5. C. I. Campbell, Radiostrontium Fallout from Continuing Nuclear Tests, Science *124*, No. 3227, 894 (November 1956). (See p. 1338.)
6. L. D. Marinelli, Gamma-Ray Activity of Contemporary Man, Science *124*, 122 (July 1956).
7. E. C. Anderson, R. L. Schuch, W. R. Fisher and W. H. Langham, Radioactivity of People and Foods, Science *125*, No. 3261, 1273–1278 (June 28, 1957). (See p. 752.)
8. *Recommendations of the International Commission on Radiological Protection*, Supplement No. 6, British Journal of Radiology, (December 1, 1954).
9. The Hazards to Man of Nuclear and Allied Radiations, British Medical Research Council, Her Majesty's Stationery Office, London (June 1956), (See p. 1539.)
10. W. F. Libby, Radioactive Fallout, presented before the Spring Meeting of The American Physicial Society, Washington, D. C. (April 26, 1957). (See p. 1519.)
11. K. K. Turekian and J. L. Kulp, Strontium Content of Human Bones, Science *124*, 405–406 (August 31, 1956). (See p. 706.)
12. K. K. Turekian and J. L. Kulp, The Geochemistry of Strontium, Geochim. et Cosmochim. Acta *10*, 245–296 (1956).
13. R. M. Hodges, N. S. MacDonald, R. Nusbaum, R. Stearns, F. Ezmirlian, P. Spain and C. McArthur, The Strontium Content of Human Bones, J. Biol. Chem. *185*, 519–524 (1950).
14. E. M. Sowden and S. R. Stitch, Trace Elements in Human Tissue. Part 2. Estimation of the Concentrations of Stable Strontium and Barium in Human Bone, AERE/MRC/R 2030 (1956).
15. G. V. Alexander, Analytical Data presented at the Bio-Medical Program Directors meeting at UCLA (April 29, 1957).
16. G. V. Alexander, R. E. Nusbaum and N. S. MacDonald, The Relative Retention of Strontium and Calcium in Bone Tissue, Jour. Biol. Chem. *218*, No. 2, 911–919 (1956).
17. R. Menzel, personal communication to J. L. Kulp, Reference 12, Science *125*, No. 3241, 219–225 (February 8, 1957).
18. K. Larsen, UCLA-380 (November 6, 1956).
19. H. I. Bowen and J. A. Dymond, Uptake of Ca and Sr by Plants and Soils from Nutrient Solutions, Jour. Expt. Botany 7, 264–272 (1956).
20. C. L. Comar, quoted in Committee Report—Deposition and Retention of Ingested Strontium 90 in the Skeleton, Washington, D. C. (April 23, 1957). Official Use Only.
21. D. Laszlo, quoted in Committee Report—Deposition and Retention of Ingested Strontium 90 in the Skeleton, Washington, D. C. (April 23, 1957). Official Use Only.
22. C. L. Comar, R. H. Wasserman and M. M. Nold, Strontium-Calcium Discrimination Factors in the Rat, Proc. Soc. Expt. Biol. Med *92*, 859–863 (1956).
23. Committee Report—Deposition and Retention of Ingested Strontium 90 in the Skeleton, Washington, D. C. (April 23, 1957). Official Use Only.
24. J. L. Kulp, W. R. Eckelmann and A. R. Schulert, Strontium–90 in Man, Science *125*, No. 3241, 219–225 (February 8, 1957). (See p. 694.)

25. H. H. Mitchell, T. S. Hamilton, F. R. Steggerda and H. W. Bean, J. Biol. Chem. *158*, 625 (1945).
26. C. L. Comar, I. B. Whitney and F. W. Lengemann, Proc. Soc. Exp. Biol. Med. *88*, 232 (1955).
27. R. J. Bryant, A. C. Chamberlain, A. Morgan and G. S. Spicer, *Radiostrontium Fallout in Biological Materials in Britain*, AERE/HP/R 2056, Harwell, Berks, (1956). (See p. 1709.)
28. R. F. Palmer and F. B. Queen, Hanford Atomic Energy Works Report, HW–31242 (1956).
29. J. C. Aub, R. D. Evans, L. H. Hempelmann and H. S. Martland, The Late Effects of Internally-Deposited Radioactive Materials in Man, Medicine *31*, No. 3, 221–329 (1952).
30. *Maximum Permissible Amounts of Radioisotopes in the Human Body and Maximum Permissible Concentrations in Air and Water*, National Bureau of Standards Handbook 52 (1953).
31. *Permissible Dose from External Sources of Iionizing Radiation*, National Bureau of Standards Handbook 59 (1954).
32. *The Biological Effects of Atomic Radiation. Summary Reports.* National Academy of Sciences-National Research Council (1956). (See p. 1827.)
33. *Pathologic Effects of Atomic Radiation*, National Academy of Sciences-National Research Council Publication 452 (1956).
34. E. B. Lewis, Leukemia and Ionizing Radiation, Science *125*, No. 3255, 965–972 (May 17, 1957). (See p. 962.)
35. A. M. Brues, Radiation as a Carcinogenic Agent, Rad. Res. *3*, No. 3, (November 1955).
36. A. M. Brues, Commentary on the Modes of Radiation Injury, International Conference on the Peaceful Uses of Atomic Energy (June 23, 1955).
37. W. B. Looney, R. J. Hasterlik, A. M. Brues and E. Skirmont, A Clinical Investigation of the Chronic Effects of Radium Salts Administered Therapeutically (1915–1931), Am. Jour. Roentgenology *73*, 1006–1037 (January–June 1955).
38. W. G. Cahan, H. Q. Woodward, N. L. Higinbotham and F. W. Stewart, Sarcoma Arising in Irradiated Bone, Report of Eleven Cases, Cancer *1*, 3–29 (1948).
39. W. M. Court Brown and J. D. Abbat, The Incidence of Leukemia in Ankylosing Spondylitis Treated with X-Rays, Lancet *268*, 1283–1285 (1955).
40. F. W. Spiers, The Hazards to Man of Nuclear and Allied Radiations, Her Majesty's Stationery Office (June 1956). (See p. 1671.)
41. R. A. Dudley and R. D. Evans, Radiation Dose to Man from Natural Sources, submitted to Joint Committee on Atomic Energy, Congress of the United States (May 20, 1957). (See p. 1236.)
42. Committee Report—Operation Sunshine, Washington, D. C. (December 19–20, 1956). Official Use Only.
43. A. M. Brues, Biological Hazards and Toxicity of Radioactive Isotopes, Jour. Clin. Invest. *28*, 1286–1296 (1949).
44. W. B. Looney, Late Effects (Twenty-Five to Forty Years) of the Early Medical and Industrial Use of Radioactive Materials. Their Relation to the More Accurate Establshment of Maximum Permissible Amounts of Radioactive Elements in the Body. Part II, Jour. Bone and Joint Surgery *38–A*, No. 1, 175–218 (January 1956).
45. J. L. Kulp, W. R. Eckelmann and A. R. Schulert, Strontium-90 in Man, Science *125*, No. 3254, 934 (1957). (See p. 705.)
46. R. E. Lapp, Strontium-90 in Man, Science *125*, No. 3254, 933–934 (1957). (See p. 704.)
47. L. Machta, The Nature of Radioactive Fallout and Its Effect on Man, presented at the Open Hearings of the Joint Committee on Atomic Energy, Washington, D. C. (May 27–June 7, 1957). (See p. 141.)
48. N. G. Stewart, R. N. Crooks and E. M. R. Fisher, The Radiological Dose to Persons in the U. K. Due to Debris from Nuclear Test Explosions prior to January 1956, AERE/HP/R 2017 (1956). (See p. 1689.)
49. W. P. Libby, Letter to Hal Hollister (June 7, 1957). (See p. 1345.)

The committee will stand adjourned.

(Whereupon, at 12:15 p. m., Thursday, June 6, 1957, the committee adjourned, to reconvene at 10 a. m., tomorrow, Friday, June 7, 1957, in the old Supreme Court chamber, the Capitol.)

THE NATURE OF RADIOACTIVE FALLOUT AND ITS EFFECTS ON MAN

FRIDAY, JUNE 7, 1957

CONGRESS OF THE UNITED STATES,
SPECIAL SUBCOMMITTEE ON RADIATION
OF THE JOINT COMMITTEE ON ATOMIC ENERGY,
Washington, D. C.

The special subcommittee met, pursuant to recess, at 10 a. m., in room P-63, the Capitol, Hon. Chet Holifield (chairman of the subcommittee) presiding.

Present: Representatives Holifield, Durham (chairman of the Joint Committee), Cole, Price, Van Zandt; Senators Anderson and Jackson.

Also present: Professional staff members: James T. Ramey, executive director; George E. Brown, Jr., Hal Hollister, staff technical adviser, and Paul C. Tompkins, consultant.

Representative HOLIFIELD. The committee will come to order.

The Chair would like to explain something which occurred yesterday for the benefit particularly of the press.

Dr. Libby in response to a request of mine on a previous day handed in a short statement regarding the scientific reasons for testing weapons. Shortly after this, one copy which was handed to me was turned in, I was called to another meeting, and I had to stay in it all morning, because it was an executive session. Senator Anderson took over the chair. There was some confusion between Senator Anderson and me, and neither one of us read the statement of Dr. Libby at that time or before adjournment, I should say. As there were no other copies handed out, there were some reporters, apparently, who noticed it and asked to see it. They read it before the papers were taken to the committee room. I have asked the staff to prepare some copies which have been handed out this morning, and I am going to ask, since it is a short statement—and it was not accepted officially—Dr. Dunham to read this into the record at this time.

STATEMENT OF DR. WILLARD F. LIBBY, UNITED STATES ATOMIC ENERGY COMMISSIONER, READ BY DR. CHARLES L. DUNHAM

Dr. DUNHAM (reading):

I have been asked by the committee to explain briefly in my statement for insertion in the record why I believe nuclear tests must continue.

For the survival of our Nation and that of the free world, heavy reliance is placed on the United States' defensive and deterrent capabilities. These capabilities are inextricably bound to our nuclear warheads and to the weapons systems which would carry these warheads.

We are today in a period of radical transition in the weapons systems available and becoming available. A very few years ago the possibility of a surprise nuclear attack upon the United States and the other free nations was small. Our own weapons could be readied carefully and the carrying vehicles could take time in delivering the warheads to destination.

Now, however, with the gains made by nations in delivery systems the technical possibility of surprise attack against us has increased. This then is forcing a change in our defensive systems. We require new weapons of smaller size, capable of withstanding extreme conditions, instantly ready and tailored to give the desired effect. These characteristics are particularly important and stringent in the case of weapons for defense of our Nation and our forces against possible enemy attack. Generally at present, each new weapon-delivery system requires a new or modified warhead design.

Naturally, in view of the reliance we must place on our nuclear-weapons systems, new or modified weapons and design changes must be tested if we are to rely on them in the emergency. Unfortunately, there is no substitute for testing to determine the reliability of a weapon, conventional or atomic. To cut off testing, therefore, means the cutting off of the introduction of improved nuclear-weapons systems. Though there can be very limited extrapolation of present information without testing, cessation of tests would, to all intents and purposes, end shortly our developmental work. It would mean the cutting off of attempts to achieve further improved designs—designs which would lessen still further radioactive contamination from detonations, designs to make most efficient use of materials, designs which could function under the extremes that they would be called upon to face. It would mean that systems being developed aand urgently needed for our defense would be without the most effective warheads.

As you know, one cannot enumerate in an unclassified statement and without divulging to the world the status of our nuclear armaments the known defense and deterrent weapon systems which we would forego by stopping tests. From past test series, we have found, sometimes unexpectedly, means of increasing the efficient use of materials, reducing the size and complexity of warheads, increasing their deliverability and yield, and reducing the radioactive contamination from our larger yield devices. The committee is familiar with these past developments and with the fact that additional systems now scheduled could not be brought into being without further tests. In a public statement, I can only emphasize that our weapons development would be crippled by a cessation of tests.

Naturally, should we interrupt our testing and others do not so do, we could find ourselves shortly with systems whose nuclear warheads were not adequate for the intended purposes. With the rapidity with which weapons systems are now changing we might find ourselves in such a position in a relatively short period of time.

As I stated earlier, therefore, cessation or interruption of testing is in itself a form of disarmament. Such a step should only be taken, therefore, as a part of a comprehensive disarmament plan. It should only be taken when there are means in existence of assuring that other nations similarly will cease their testing and hence their development of new systems.

We must continue our efforts to find an assured way of disarmament, as part of which all would stop testing of nuclear weapons. In the meantime, we as a Nation should continue to limit our test shots to those essential to our development program for the weapons so vital—in the absence of safeguarded disarmament—to assuring the survival of the free world. We should continue by all possible means our efforts to reduce the amount of residual contamination from these fully justified shots.

Representative HOLIFIELD. The request of the Chair was that Dr. Libby give the scientific reasons for the continued testing of weapons. While there are some scientific reasons in the statement, there are also statements of policy and conclusions on Dr. Libby's part. In view of the fact that Dr. Libby is not here, I have no questions on the statement. Do the other members of the committee have any questions?

Chairman DURHAM. No.

Representative COLE. Except to point out, as I am sure the Chair will agree, that Dr. Libby could not discuss the scientific considera-

tions and justifications of continuing weapons tests without disclosing vital security information.

Representative HOLIFIELD. I certainly agree with that statement.

Dr. Dunham, I believe you have some documents to insert in the record.

Dr. DUNHAM. Yes; I have two statements prepared by Dr. Libby, advance copies of which the committee already has, but these are up-dated material on radioactive fallout on soils and the radioactivity of rain, and the other statement has to do with natural occurrence of radioactivities.

I would like to on Dr. Libby's behalf introduce these in the record.

Representative HOLIFIELD. I do not believe the members have had a chance to see these statements. Did you present the copies this morning?

Dr. DUNHAM. I believe Mr. Hollister has copies of these. Yes; they were brought up this morning.

Representative HOLIFIELD. For the present we will accept them without knowing exactly what they are.

Dr. DUNHAM. I would also like to take the opportunity at this time to introduce a statement prepared by Dr. Harry Wexler, of the Weather Bureau, entitled, "Radioactive Fallout in the Stratosphere," which is essentially a discussion of research approaches in meteorology to solving some of the fallout problems.

Chairman DURHAM. Mr. Chairman, this is a very interesting statement. It will not take but a few minutes to read it. Would you like to read it?

Dr. DUNHAM. I will be very happy to read it if the committee so desires.

STATEMENT OF DR. HARRY WEXLER,[1] DIRECTOR OF METEOROLOGICAL RESEARCH, UNITED STATES WEATHER BUREAU (READ BY DR. CHARLES L. DUNHAM)

Since the large nuclear explosions deposit a considerable portion of their radioactive debris in the stratosphere, these questions arise: What happens to this radioactivity? Does it mix uniformly over the world after a year or two? Is there much vertical mixing upward and downward? Are there important

[1] Chief scientist of the USNC–IGY Antarctic Program and is responsible for its entire scientific effort to be undertaken in 1957 and 1958 in Antarctica. He is a member of the USNC–IGY Antarctic Committee, the USNC–IGY Technical Panel on Meteorology, the ad hoc Arctic Committee and the ad hoc Equatorial Committee. He is a member of the United States National Committee for the International Geophysical Year and an alternate member of its Executive Committee. Received his B. S. degree in mathematics from Harvard University in 1932. He did graduate work at the Massachusetts Institute of Technology from 1932 to 1934 and from 1937 to 1938 and obtained his Sc. D. in meteorology in 1939. During the intervening period, 1934 to 1937, he worked at the United States Weather Bureau in Chicago, Ill., and Washington, D. C. He was appointed assistant professor of meteorology at the Institute of Meteorology of the University of Chicago in 1940 and left this position in late 1941 to return to defense work at the Weather Bureau in Washington. In November 1942, he joined the Army Air Force's Weather Service as a captain and was appointed senior instructor in meteorology to the Army Air Force Aviation Cadet School in Grand Rapids, Mich. While there he served as a member of the wartime University Meteorological Committee established to assist the military services in matters related to meteorological training.

In late 1943, Dr. Wexler was transferred to the Weather Division of the Army Air Forces in Washington, where he served as research executive and initiated and fostered a program of research in weather which later developed into the large Air Force program in geophysics. In September 1944, with Colonel Floyd Wood as pilot, made the first penetration of an Atlantic hurricane, which because of its unusual intensity became known as the Great Atlantic Hurricane. In 1946 Dr. Wexler became Chief of the United States Weather Bureau's Science Services Division. In 1955 he was named Director of Meteoro-

seasonal and geographic effects? Are there channels connecting the stratosphere to the troposphere through which large quantities of radioactvity can flow downward with the air and ultimately reach the precipitation layers and thus be rained out?

Dr. Machta has presented testimony pointing strongly in the direction of nonuniformity of stratospheric fallout which is corroborated by analysis of strontim 90 in soil samples collected by Dr. Alexander. This is a surprising result to most meteorologists who would believe that after a year or two, airborne contaminants released in the stratosphere should be uniformly mixed over the world. Admittedly, there has been little evidence to go on; for example, Krakatao volcanic ash apparently spread uniformly over the world within 3 to 6 months as best as could be judged from fragmentary optical and solar radiation effects available in 1883.

In recent years, observations of the atmospheric content of ozone, most of which is found in the lower stratosphere, show decided geographic and seasonal nonuniformity over the world, although the interpretation of this gas in terms of mixing is complicated by geographic and time variations in solar ultraviolet radiation, air temperature, and large-scale vertical air motions.

Against this background of conflicting evidence, one thing stands out. In view of great quantities of radioactive debris now present in the stratosphere and the possibility that as the years go on, new and larger amounts may be placed there, we must learn much more about the lateral and vertical transport and mixing mechanisms in the stratosphere. In addition, further understanding of atmospheric removal processes is needed. An outline of proposed research and development is presented below.

1. METEOROLOGICAL OBSERVATIONS IN THE STRATOSPHERE

Only in recent years have meteorological balloons penetrated systematically into the lowest layers of the stratosphere—but their average height (60,000 feet) is well below the top of the large thermonuclear bomb mushroom clouds (100,000 feet or higher). With increased effort during the International Geophysical Year, the average height may approach closer to 100,000 feet, but there will be large areas, particularly in regions of low temperatures, where the balloons become brittle and burst before rising appreciably in the stratosphere or even reaching the stratosphere.

Recommendation: Development of a relatively inexpensive, lightweight, solid propellant, frangible cased rocket, capable of carrying meteorological instruments to 200,000 feet and dropping them by parachute which can be followed by radar. This offers the only hope of establishing a widespread network of meteorological observations in the important 100,000–200,000 foot layer to observe stratospheric winds, temperatures and densities, so that the higher yield atomic clouds can be tracked.

2. AIR SAMPLING AND ASSAYING IN THE STRATOSPHERE

Although the availability of stratospheric meteorological observations will aid in determining the bulk transport of clouds of radioactive debris, they will not usually be sufficient to help estimate the degree of mixing between the

---

Footnote continued from preceding page.

logical Research. The Weather Bureau has made Dr. Wexler available to the USNC–IGY for scientific direction of the Antarctic program.

In 1945 the Institute of Aeronautical Sciences presented Dr. Wexler the Losey Award in recognition of his outstanding contributions to the science of meteorology as applied to aeronautics.

A member of the Advisory Committee on Reactor Safeguards for the Atomic Energy Commission; Chairman, Committee on the Meteorological Aspects of the Effects of Atomic Radiation of the National Academy of Sciences; a member of the Meteorological Operations Subcommittee of the National Advisory Committee for Aeronautics. He is a councillor of the American Meteorological Society, a fellow of the American Academy of Arts and Sciences, a member of the Royal Meteorological Society and the Washington Academy of Sciences. He is a member of the American Geophysical Union. In August 1955, Dr. Wexler was a member of the United States delegation to the Atoms for Peace Conference in Geneva, Switzerland.

As an internationally recognized authority on meteorology, Dr. Waxler has published some 50 papers in scientific journals treating such subjects as the radiative cooling of the air, polar anticyclones, atmospheric turbidity, structure of hurricanes, and upper atmosphere temperatures and dynamic connections with the lower atmosphere.

In November 1956 was the recipient of the Department of the Air Force Exceptional Service Award in recognition of distinguished patriotic service. (Submitted by U. S. Department of Commerce.)

radioactive clouds and their environments. The existing limited program of sampling the stratosphere for natural and artificial constituents has thrown some light on the incompleteness of mixing in the stratosphere. Strontium 90, carbon 14, water vapor, and ozone have been sampled or assayed directly, and the few observations available, especially of the vertical profile of water vapor and ozone, reveal a marked stratification in the lower stratosphere, indicating very weak vertical mixing—at least during the few times soundings were made. There are not sufficient observations geographically or seasonally to arrive at similar conclusions regarding the degree of lateral mixing.

Recommendations: An intensified program of air sampling and analyzing to a height of 150,000 feet by aid of large "sky-hook" balloons should be inaugurated over large areas to determine the lateral and vertical mixing efficiency of the lower stratosphere and exchange mechanisms with the troposphere.

3. PREDICTION OF STRATOSPHERIC TRANSPORT AND MIXING

After an adequate program of stratospheric observations has been inaugurated so that one has a reasonably accurate picture of the present state of the stratosphere and its distribution of trace elements, then the question of the future state arises.

The few day-to-day weather analyses that have been carried on in the lower stratosphere suggest strongly that the flow patterns are so different from those existing below that inference or extrapolation from lower level happenings or predictions are of little or no value. The electric computer, with its capacity for analyzing large quantities of data quickly, has proved its value in forecasting important changes in flow pattern in the troposphere such as the change from a basically zonal (west and east) flow to largely meridional (north and south) flow. Such changes, occurring very rapidly, can have an important effect on the distribution of airborne material in the troposphere. It is suspected that there may be similar large scale changes in flow patterns in the stratosphere.

Recommendations: A special research unit should be established to plot and analyze daily stratospheric charts, using all the data described above, and having access to a modern high speed automatic computer to develop prediction techniques for stratosphere transport, mixing, and exchange with the troposphere.

4. REMOVAL PROCESSES

The hazard from strontium 90 stems from ingestion. The radioactive particles must, therefore, be removed from the atmosphere onto the ground or foliage. Evidence suggests precipitation as the prime removal process, although not the exclusive one. Impaction on obstacles and, to a lesser extent, gravitational settling are other known mechanisms.

The details of the precipitation scavenging process are a matter of speculation. Empirical correlations with fallout are inadequate for predictive purposes under different conditions.

The removal of small particles over water bodies is especially important since water surface constitutes almost three-fourths of the earth's surface.

Recommendations: The study of removal processes by impaction, scavenging, etc., should be intensified. Ocean samples should be analyzed for strontium 90 and cesium 137 in order to determine whether the oceanic fallout pattern follows that over land.

Representative HOLIFIELD. Are there any questions on this statement?

Dr. Dunham, does the AEC have any comments on this statement?

Dr. DUNHAM. No, it seems like a very sound recommendation.

Chairman DURHAM. You have made some fine recommendations here. Are we equipped to carry out the recommendations, as far as you know?

Dr. DUNHAM. Maybe Dr. Machta, who is here, would like to speak to that point. I gather his item 1 would require some development work. I am sure Dr. Machta is better prepared to answer that question.

Representative HOLIFIELD. Dr. Machta, would you like to come forward?

Chairman DURHAM. Have you read Dr. Wexler's statement?

Dr. MACHTA. Yes, I have.

Chairman DURHAM. Do you concur in his recommendations?

Dr. MACHTA. Yes, sir, I do.

Chairman DURHAM. Is any agency of the Government equipped to carry out his recommendations at the present time?

Dr. MACHTA. Yes, I think the Weather Bureau, if asked to do so, would be equipped to do it.

Chairman DURHAM. You are qualified to do it?

Dr. MACHTA. Yes, sir. We have a high-speed computer which is used for our everyday forecasting which would be devoted to this purpose, and we have in mind certain other projects which Dr. Wexler indicated.

Chairman DURHAM. You think it is important that we proceed to do this type of testing?

Dr. MACHTA. Yes, sir; I do.

Representative HOLIFIELD. This statement, as I read it, has to do with nonuniformity of stratospheric fallout. Does that pertain to the matter while it is in the stratosphere or does it pertain to the nonuniformity of its descent to earth?

Dr. MACHTA. The statement includes both aspects. In making a prediction, one must know where it is in the atmosphere, and how it would be removed.

Representative HOLIFIELD. At the present time the research you recommend is to find out the conditions in the upper stratosphere?

Dr. MACHTA. This primarily, but secondly the removal processes. For example, does it, as we really suspect happens, come down primarily in rainfall and factors of this sort?

Representative HOLIFIELD. It goes to sustain the theory of nonuniformity of deposit on the earth's crust, does it not?

Dr. MACHTA. I do not believe I would say that. What we want to do is to conduct more research to confirm the suspicions we have.

Representative HOLIFIELD. His suspicions, if you want to call them that, are along that line?

Dr. MACHTA. Yes, sir.

Chairman DURHAM. Is any of this type of sampling now being done?

Dr. MACHTA. Yes. During the International Geophysical Year, a very much more extensive program of ozone sampling in the upper atmosphere is to be conducted. We believe this will throw great light on the exchange in the stratosphere and the exchange between the stratosphere and troposphere. In addition, the AEC is undertaking a very good sampling program of strontium 90 and other fission products in the stratosphere which would be very important.

Chairman DURHAM. How about the oceanic testing?

Dr. MACHTA. According to my information, the Woods Hole Oceanographic Institute is sampling the Atlantic Ocean to determine the content of strontium 90. Their preliminary report will be out very shortly.

Representative HOLIFIELD. Before we proceed further, it has been called to my attention that we have Dr. Waterman, the Director of

the National Science Foundation, present this morning to partake in the panel discussion. We are glad to have you here, sir.

Dr. WATERMAN. Thank you.

Representative HOLIFIELD. Dr. Dunham, yesterday Senator Richard L. Neuberger released a press release which reads as follows:

> Senator Richard L. Neuberger today urged that the Atomic Energy Commission make public a report on strontium 90 effects submitted to the AEC by an advisory committee.
>
> The Oregon Senator charged in a Senate speech that the report, requested by the Commission over 2 years ago, has not yet been issued in final form due to "official reluctance."
>
> Dr. H. Bentley Glass, professor of biology at Johns Hopkins University, indicated Tuesday that release of the study prepared by the Advisory Committee on Biology and Medicine has been delayed by the AEC. Glass is a member of the advisory group.
>
> "The American public has a right to know of the dangers inherent in exposure to radioactive materials," Neuberger declared. "Suppression of this report on the part of the Atomic Energy Commission represents a betrayal of public trust."
>
> "This subject is one of vital concern to all Americans," the Senator said. "It affects not only the present population of the United States, but future generations as well. Any attempt to shield the facts from public scrutiny is inexcusable."
>
> The 44-year-old Oregon Senator, sponsor of a bill to establish a National Radiation Health Institute, said that the AEC owes the public an explanation as to why the report was not previously released. "Full disclosure of unclassified information regarding radiation exposure is vital to establishment of informed public opinion and the rational discussion of this issue which looms paramount in the minds of many Americans" he said.

In view of the fact that this release was made during these hearings, and in view of the fact that there has been no evidence presented to the committee that there has been such a study or report, I will ask you at this time to comment on this in order that the Atomic Energy Commission may have an opportunity to state its views.

Dr. DUNHAM. Thank you very much, Mr. Holifield.

First I would like to make it perfectly clear that there is no such report. The Commission has never asked that the Advisory Committee on Biology and Medicine make a report.

On the other hand, as I indicated the other day in testimony, during the last 2 years there has not been a meeting of that committee at which the matter of the fallout studies was not discussed, and a brief bringing up to date of the commitee on the facts given by a member of my staff. The committee has considered this matter very, very carefully. They have as yet prepared no statement on the subject.

Representative HOLIFIELD. There has been a study made of this subject?

Dr. DUNHAM. Not by the committee as such. They have reviewed the status of the material being developed at each of their meetings each year. When on 1 or 2 occasions I neglected to put this item on the agenda, one or another of the Commissioners called my attention to it, and asked that it be put on the agenda, bringing the committee up to date on this information.

Representative HOLIFIELD. Has this committee been presented with the results of your studies on strontium 90?

Dr. DUNHAM. Yes. They have been presented, as I say, at every meeting, and they have been brought up to date on the new material as it developed.

Representative HOLIFIELD. I am speaking of the congressional committee. Has this committee been presented with all of the pertinent information in regard to strontium 90 which is in possession of the AEC?

Dr. DUNHAM. I believe this is so. With the material introduced in the record by Dr. Libby and material presented by other people who have appeared before you, I think this constitutes the present body of knowledge of the Atomic Energy Commission on the contamination of the environment by strontium 90.

Representative HOLIFIELD. Is there an area of knowledge on the subject of strontium 90 which is in the classified area and which has been or will be presented to the committee in executive session?

Dr. DUNHAM. The only material pertinent to this question which is classified is that to do with actual fission yield of specific devices. This material, I believe, has been in the hands of the committee. You are brought up to date at each series of tests as to what these fission yields are.

Representative HOLIFIELD. So there has been no withholding of your overall calculations as to the amount that is in the stratosphere, and which has been deposited, according to your measurements, in various places throughout the earth's crust?

Dr. DUNHAM. That is right. There has been no withholding at all.

Representative HOLIFIELD. That information, to the best of our present ability to measure, has been presented not only on the continental United States, but other measurements in other lands?

Dr. DUNHAM. Yes. We have presented you with all the material we have developed.

Representative HOLIFIELD. Are there any further questions on this point?

Chairman DURHAM. Then it is a fact, Doctor, that all the information has been made public through the present hearings and other methods?

Dr. DUNHAM. That is correct, sir.

Representative HOLIFIELD. Dr. Dunham, we will now let you read your statement.

STATEMENT OF DR. CHARLES L. DUNHAM, DIRECTOR, DIVISION OF BIOLOGY AND MEDICINE, ATOMIC ENERGY COMMISSION

Dr. DUNHAM. Thank you. I would like first to say what I am going to do is to review briefly the present program of research of the Division of Biology and Medicine which relates to fallout. I understand the committee has invited representatives of other Government agencies who are making very important contributions to the general information on this subject to present their programs. Therefore, I will let them speak for themselves.

I have with me today, Dr. Shields Warren, from the New England Deaconess Hospital. I would like to call upon him, after I have finished, to speak from the standpoint of a university scientist, plus his great background in this particular area. Also, Dr. Austin Brues, who is the Director of the Division of Medicine and Biology of the Argonne National Laboratory, to speak to you from the standpoint of a director of such a program at one of our national laboratories.

That part of the research program of the Division of Biology and Medicine which relates to fallout falls into four major categories:

1. Collection and analysis of samples to determine distribution of strontium 90 in the atmosphere and the biosphere.
2. Further refinement of our knowledge of the radiotoxicity of strontium 90 and cesium 137 and other radionucleides and of the long-term effects of external gamma radiation.
3. Research into methods of treating and ameliorating radiation injury.
4. Civil effects test programs at weapons tests.

With the exception of the activities of the Health and Safety Laboratory in New York, all work is done by contract either at national laboratories or at private laboratories, both university and commercial, or with Government laboratories by transfer of funds to Naval Radiological Defense Laboratory, Naval Research Laboratory, the Air Force, and so forth.

With the exception of experimental studies done at weapons tests, none of this work is classified even at the time it is done. In fact, we have made deliberate efforts to see that this is so, so as to avoid delays in making the information available. That work undertaken at weapons tests is now carefully planned so that insofar as possible the first preliminary reports are written in unclassified form and the final complete reports whenever possible are unclassified or unclassified versions are written simultaneously.

I. COLLECTION AND ANALYSIS OF SAMPLES

The gummed-paper network of the Health and Safety Laboratory now comprises 94 stations in the United States and 75 in foreign countries. Stainless-steel pot collections are being made at 7 stations in the United States and in the following countries: Hawaii (which is not a country), Chile, French West Africa, Austria, Union of South Africa, Thailand, South Rhodesia, Peru, Pakistan, Kenya, Japan (2), Colombia, and Brazil. This network is to be expanded as cooperative arrangements are developed with other countries.

Soil sampling on a worldwide basis is under Dr. Lyle Alexander, United States Department of Agriculture, and collections in 17 countries were made in 1955 and in 39 countries in 1956 and annually or more frequently collections are made in various parts of the United States. These are supplemented by soil collections in the New York area by the Lamont Laboratories of Columbia University. In the future, we hope to be able to undertake simultaneous soil sampling in North and South America on an annual basis.

Sampling of the Pacific Ocean in relation to weapons tests is extensive, and is supplemented by excellent Japanese sampling and analysis, with frequent exchanges of data.

The ocean is large, and we cannot sample the whole ocean, but we do endeavor to follow the radioactive material as it moves along with the current. In cooperation with such organizations as the NORPAC and the Japanese, we have been able to get quite a lot of information on the movement of these water masses.

Followup studies on the Rongelap Atoll are annual, and include analysis of soils, edible plants, animals, and seafood. Both the Naval Radiological Defense Laboratory and the University of Washington

School of Applied Fisheries, and the biological laboratories established by the AEC at Eniwetok have taken part in this with assistance from the Walter Reed Medical Center. The medical followup on the people of the Rongelap and Uterik Atolls is under the direction of a group at Brookhaven National Laboratory with assistance from the National Institutes of Health, Walter Reed Medical Center, and the Department of the Navy.

The human bone sampling program is largely carried out by the Lamont Laboratories in New York City with supplementary collections by the Argonne Cancer Research Hospital and other groups in this country. Analyses are carried out by commercial laboratories with careful cross checks by Lamont Laboratories and by the Health and Safety Laboratory of AEC for analytical accuracy.

Representative HOLIFIELD. I understand that Dr. Schulert is here to talk about the bone samples.

Dr. DUNHAM. That is fine.

Stratospheric sampling: Techniques are being developed to make possible the monitoring of radioactive fission products in the stratosphere. Such measurements would provide important information on quantities of weapons debris reaching the stratosphere, the distribution and retention of such materials in the stratosphere, and their release to the lower atmosphere. In experiments now being conducted with the Department of Defense, balloons were used to carry sampling equipment to altitudes of 50,000 to 90,000 feet, where radioactive particles were filtered from a defined volume of air. Balloons were being launched at Minneapolis, Minn., San Angelo, Tex., and at France Air Force Base in the Panama Canal Zone.

Radiochemical analyses of the samples are presently being made on a pilot scale by the Commission's Health and Safety Laboratory, New York, until arrangements can be made with commercial laboratories to perform this work. Results of these studies will be useful in planning a worldwide network for the stratospheric monitoring of long-lived radioistotopes.

In addition, we have work at the Midway Laboratories in Chicago in attempting to develop a more adequate method of sampling. The present sampling devices are filter devices and, although we believe they collect 25 percent of the material present in the stratosphere, we want to be able to be sure we are actually getting all of the material from a given volume of air.

The milkshed sampling program includes regular sampling in the New York and Chicago milksheds, with less frequent sampling by the Health and Safety Laboratory and the Los Alamos Scientific Laboratory of other milk supplies.

Food sampling in the United States is related to milksheds and the location of United States agricultural experiment stations and carried out largely under Dr. Alexander in the United States Department of Agriculture.

Representative COLE. May I interrupt here, Dr. Dunham, to ask you to explain to what extent you conduct continued sampling of milk in the area of the Nevada tests?

Dr. DUNHAM. We have some samples of that.

Representative COLE. The reason for the question is that, presumably, the fallout would be greater in the area near to the Nevada test.

Dr. DUNHAM. Yes, sir.

Representative COLE. Some people may feel and I am curious why you select the Chicago and New York milksheds for your milk tests.

Dr. DUNHAM. Those are the two largest milksheds, of course. As to the milk within hundreds of miles of the Nevada test site, much of that is imported. Some is developed locally. But the Los Alamos Scientific Laboratory has been sampling milk in communities there.

Representative COLE. That answers the question. The Los Alamos Laboratory is the agency responsible for sampling the milk in the area of the Nevada test.

Dr. DUNHAM. That is correct, sir.

Representative COLE. That is, I assume, a reasonably continuous sampling process?

Dr. DUNHAM. That is correct. We are in the process of initiating a worldwide food sampling program with the assistance of the Interdepartmental Committee on Nutrition, aimed at checking especially the food constituting the principal source of calcium in the diet. Analyses will be by the Health and Safety Laboratory or contracted out.

There are two ways in which the information thus developed reaches the public: One, as unclassified Atomic Energy Commission reports, second, the publication in the scientific literature. The material often appears first as an unclassified AEC report and later is published in a scientific journal. This is in part because of backlogs of accepted papers in the better scientific journals. This material is also disseminated to the United Nations Scientific Committee on Radiation Effects as soon as it has become available in printed form to United States scientists. It is our hope that many of the worldwide collections and analyses can be done in the countries of origin. The AEC Health and Safety Laboratory is training scientists from a number of countries in these techniques. Pending their taking over, we are getting excellent cooperation from the countries which have expressed an interest in these matters. Meanwhile we are analyzing any samples submitted from foreign countries.

Representative VAN ZANDT. At this point, Dr. Dunham, is it not true that spokesmen from the AEC are constantly making speeches and copies of the speeches are given a pretty good coverage, and they contain firsthand information taken from the files of the AEC?

Dr. DUNHAM. That is one of the reasons these speeches are given. By the time you get the reports accepted and printed by the scientific journals, much time has elapsed. Many of these speeches are done deliberately to get the material out as fast as it is available.

Representative VAN ZANDT. I might say I read these speeches religiously and as a result I feel I am being kept up to date in regard to the developments in this field.

Representative COLE. Let me say in response to that that if my good friend, Mr. Van Zandt, reads all the speeches coming to his desk emanating from the Atomic Energy Commission, he does not do anything else.

Representative VAN ZANDT. I am speaking about radiation hazards.

Representative COLE. Because they come to my desk, and I admit that I do not read them all. They come too fast.

Representative VAN ZANDT. I agree with my colleague from New York. I am especially interested in the radiation hazard.

Dr. DUNHAM. I know you have a special interest in that, Congressman.

The worldwide and national bone sampling program will be stepped up as more sources of material can be found.

II. RADIOTOXICITY STUDIES

The radiotoxicity studies include extensive small animal studies at the University of Rochester atomic energy project, Argonne National Laboratory and Los Alamos Scientific Laboratory. Lifetime studies in dogs at the University of Utah to compare in mature animals radium, plutonium, mesothorium, and strontium 90 have been underway for 6 years. The University of California, Davis Campus, is just commencing a womb-to-tomb strontium 90 experiment in dogs. That means the mothers are fed strontium 90, and as soon as the pups are born, they are given strontium 90 throughout their whole lifetime. That is the way that experiment is planned.

Representative COLE. Doctor, would you tell me why dogs are selected for this experiment, rather than any other animals?

Dr. DUNHAM. Yes. We have quite a lot of information on mice and rats. But these substances we are talking about here are bone seekers. A rat bone or a mouse bone never really ceases to grow. Furthermore, they are very short-lived animals. It is a matter of a year or 2 or 3 at the most. Dogs are chosen because they are longer lived and, secondly, because their bone development and maturation follow very closely the human pattern. So when they are mature and the epiphyses have closed the bone structure of those dogs resemble exactly what we know to exist in the radium dial workers and the people who were given radium as treatment which are the basis of our information on radiotoxicity of internal emitters in man.

Representative COLE. What is mesothorium?

Dr. DUNHAM. Mesothorium is one of the products in the decay scheme of the thorium series. It is the material which Dr. Looney mentioned as being in the radium dial paint, and which has confused some of the data there because the early work was done sort of like somebody's cook. They might put more or less mesothorium in the mixture. Dr. Evans' group at Cambridge has worked very hard and now I believe has developed a method which he feels is satisfactory so he can work back on the living patients and estimate how much of the thorium series was introduced with the radium. This has been a very difficult problem.

Three more large-scale dog studies are planned with strontium 90, cesium 137, and mixed fission products.

Studies on the discrimination between strontium and calcium in animals and humans are underway at the Oak Ridge Institute of Nuclear Studies, University of Rochester, University of California at Los Angeles atomic energy project, Sloan-Kettering Research Institute, and Montefiore Hospital in New York. Additional work will be undertaken by the Argonne National Laboratory and the Argonne Cancer Research Hospital as low-level counting facilities become available at Argonne National Laboratory.

III. TREATMENT OF WHOLE BODY RADIATION INJURY AND METHODS OF AMELIORATING RADIATION EFFECT

Currently there are three approaches to a more specific treatment for whole-body injury:

1. Prophylaxis: This approach is useful only if given prior to exposure. In other words, it would have no practical value in the event of an atomic catastrophe except if a person knew he had to go in and take two or three hundred roentgens, he would be able to take some prophylactic measure to reduce his injury.

Representative VAN ZANDT. Dr. Dunham,, would it be possible to innoculate military personnel with this preventive, whose duties kept them in close proximity to reactors on board naval vessels?

Dr. DUNHAM. What we have been working with now, which shows promise, you have to take a few hours before exposure. It is questionable whether a person could take it indefinitely so I don't think it would be useful there. But if a person had to go in in the event of an accident and had to take 300 roentgens to save a person's life, this would be very useful.

Representative VAN ZANDT. What would be the life of this preventive?

Dr. DUNHAM. It can be taken in experimental animals. This is the point I want to make very definitely. This can be taken for a matter of several days. You have to begin and give a good-sized dose within a few hours prior to beginning of the exposure.

Chairman DURHAM. What is the combination of the bromide?

Dr. DUNHAM. That means that this long name here is a salt just like sodium chloride. The chloride is what makes it the salt.

Chairman DURHAM. I could not tell from that whether it was a combination of 3 or 4 other salts or not.

Dr. DUNHAM. It is the bromide of this compound.

Representative COLE. Is there reasonable reason for encouragement in this field of pretreatment?

Dr. DUNHAM. Yes. We found this particular material, which I abbreviate AET, developed at the Oak Ridge National Lab to be quite effective in essentially doubling the resistance of rats, mice, and monkeys. The only question is what is the toxicity of the material in man? We found in dogs they do not tolerate it. You cannot give enough to protect the dog and similarly the rabbit. This will have to be determined and some studies are being done. Some work is being done using this material at the National Institues of Health and other hospitals, not in the doses required to protect, but in the treatment of what is called radiation sickness, the nausea and vomiting that goes with radiation therapy, so we can begin to get a feel of how sensitive human beings are to this material.

We don't know at this point. It may be like one of the new drugs for TB. You can cure monkeys because they are 30 times less sensitive than humans to the drug. But you can't give enough to the human to totally wipe out the disease.

Representative VANZANDT. How much of the dose could the body of of the animal absorb if this preventive was applied beforehand?

Dr. DUNHAM. I don't recall the exact doses for any specific species of animals. It is a matter of a few hundred milligrams. I don't recall the exact dose.

AET (S-B-aminoethylisothiouronium bromide) pretreatment developed at Oak Ridge National Laboratory holds great promise. Its toxicity in humans has yet to be fully evaluated. In experimental animals it will roughly double resistance.

2. Treatment which is effective if given up to 48 hours after exposure begins: (*a*) Bone marrow transplants have proven very effective in a variety of mamalian species—mice, rats, and monkeys. Intensive efforts to establish the value of this treatment in humans suffering from aplastic anemia are currently underway. There are several hospitals in this country which are attempting to do this in humans who have had their bone marrow injured by radiotherapy which was necessary to treat a cancerous condition. This has only been going on for a few months. I think we will have a pretty good feel for this 6 months from now as to whether this is a feasible approach.

Representative VAN ZANDT. Dr. Dunham, some years ago at Los Alamos there was an accident that involved Dr. Graves and another physicist.

Dr. DUNHAM. Yes.

Representative VAN ZANDT. The other physicist died as a result of the dose.

Dr. DUNHAM. That is correct.

Representative VAN ZANDT. How much knowledge did the Commission gain as a result of that accident which caused the death of the physicist?

Dr. DUNHAM. I would say that the report which has been published in the Annals of Internal Medicine of both accidents—there were 2, 1 a little earlier in the game, and 1 about 1945—constitutes the most carefully documented, from a scientific standpoint, group of cases of whole body radiation exposure, complicated by skin burns, that exists anywhere.

Representative VAN ZANDT. Do you recall the name of the physicist?

Dr. DUNHAM. It was Dr. Sloton who died in one accident. The other one was an Armenian name. Dr. Sloton is the one I think you are thinking of.

Representative VAN ZANDT. How long did he live after the accident?

Dr. DUNHAM. About 3 or 4 weeks.

Representative VAN ZANDT. Can you describe the body reaction to the exposure?

Dr. DUNHAM. What essentially happened is that he had his hands quite close to the criticality experiment that got out of control, so he received probably 60,000 to 80,000, or better, rep equivalent to the skin and forearms. He received a very extensive burn. It was almost as though you immersed his hands in a tub of boiling water and held them there for some time. In addition there was a complete cessation of the formation of all the blood constituents. That was the whole body effect of the mixed gamma and neutron irradiation.

Representative VAN ZANDT. Was he unconscious immediately after the accident?

Dr. DUNHAM. I don't recall. I don't think anybody was unconscious immediately after the accident.

Representative VAN ZANDT. I understand there was ample time to study the effects?

Mr. DUNHAM. These were done by Dr. Louis Hempelmann, who is now at Rochester. I might say that this matter of bone-marrow

transplants was originally developed by Dr. Lorenz, the late Dr. Lorenz, at the National Cancer Institute, who was working during the Manhattan District days very closely with Dr. Leon Jacobson at the Argonne National Laboratory.

(*b*) Another approach is to concentrate an active substance believed to be present in pregnant cow's blood. This is a very elusive material and all attempts to concentrate it to date have failed. It is fragile and very difficult to keep in its original state. There is no question that there is something in pregnant cow's blood which does convey a certain degree of protection after injury has occurred.

III. REMOVAL OF RADIONUCLIDES FROM THE BODY

As to the removal of radionuclides from the body, and this is of prime interest with respect to fallout and strontium 90, whereas we now have two very valuable drugs, EDTA (ethylenediamine-tetraacetic acid) and zirconium citrate which will divert plutonium in the blood stream away from bone and into the urine. No known preparation will do this for strontium. We have nothing which will selectively remove important amounts of either plutonium or strontium once it is deposited in the bone. Work in this field has been discouraging; nevertheless scientists at the Los Alamos Scientific Laboratory and at the Montefiore Hospital in New York are working hard at the problem.

We have a few projects studying skin burns induced by beta radiation as well as the followup studies on the Sandstone skin burns, and the skin burns in the Rongelapese. There are also a series of studies dealing with the problem of inhalation of radioactive materials. In addition to all this we have a large effort in the genetic effects of radiation and in the biochemical effects of radiation which will provide basic knowledge of the mechanisms of injury which in turn should permit a more efficient approach to the problem of combating radiation effects.

IV. CIVIL DEFENSE ASPECTS

As to the methods of coping with fallout, they fall into two groups.

1. Adequate shelter during maximum gamma radiation hazards: The Division of Biology and Medicine has, since 1952, taken advantage of each Nevada test series to develop information on shelter design. This work is done in cooperation with the Federal Civil Defense Administration and at Operation Plumbob the FCDA is supporting practically all of the work on shelters.

2. Decontamination: This is relatively easy with ordinary detergents and water for small objects and structures. For larger areas it is an economic problem. One approach is to fix with asphalt spray, then bulldoze off. For very large farm areas this becomes very difficult and one must rely on time for decay and deep plowing for more uniform mixing, thus diluting available strontium-90. Small areas of low-calcium soil can be treated with calcium to reduce the strontium-90–calcium ratio. Milk can be treated similarly up to a point, or one can simply remove all calcium and strontium by ion exchange resins and add uncontaminated calcium. Continuing the initial studies at Upshot-Knothole and at Operation Teapot, Operation Plumbob will develop further information on contamination of soils, crops,

and foods, shielding attenuation factors for various structures and structural configurations, and evaluation of decontamination procedures under field conditions.

Mr. Chairman, at this point I would like to ask permission to introduce into the record a summary of the Operation Plumbob civil effects test group project summaries, dated 1957. I would like also, with your permission, to introduce into the record a letter which I wrote you on May 28 outlining the shelter program at Operation Plumbob.

Representative HOLIFIELD. I am glad you are presenting that. I had intended to ask you to, or have Mr. Corsbie do so.

(The material referred to follows:)

MAY 28, 1957.

Hon. CHET HOLIFIELD,
Chairman of the Special Subcommittee on Radiation of the Joint Committee on Atomic Energy,
Washington, D. C.

DEAR MR. HOLIFIELD: This is in reply to your request of May 27, 1957, for information on the number of shelters, and sponsors thereof, being tested in the current weapons-test series.

The program of the civil effects test group includes studies on the blast effect on prototype shelters and test structures, the latter being tested principally to obtain engineering design data. There are 3 reinforced home-type shelters for a few persons; 1 mass shelter capable of holding large numbers of people and serving the dual function of a parking garage; 3 concrete dome structures for obtaining engineering design data; 1 industry-financed prototype vault for records storage; 1 industry-financed windowless reinforced clay-masonry structure; 9 shelters similar to design and capacity of the German structures; and 3 prototype entranceways to test blast-resistant doors, ventilation equipment, et cetera, for France.

All of the above structures are being tested under the sponsorship of the Federal Civil Defense Administration and, except for the industry-sponsored and foreign shelters, are being financed by FCDA.

In addition, the AEC is continuing some blast biology studies, using two shelters which were included in the Teapot series of 1955. These were tested at pressures of approximately 100 pounds per square inch. They are being used to supply further data on biologically acceptable criteria for open shelters.

Also, I am enclosing copy of Mr. Corsbie's remarks prepared for the press briefing in Nevada, which explains in more detail the civil effects test program.

Sincerely yours,

C. L. DUNHAM, M. D.,
Director, Division of Biology and Medicine.

[Preseries briefing, May 1957]

NEVADA TEST ORGANIZATION

OFFICE OF TEST INFORMATION

Las Vegas, Nev.

REMARKS BY ROBERT L. CORSBIE, DIRECTOR, CIVIL EFFECTS TEST GROUP

Organizationally, the Civil Effects Test Group is one of six scientific and technical units reporting to the test director and is a counterpart of the Military Effects Group. It is principally sponsored by AEC and FCDA. Other Government agencies, some private industrial groups, and two foreign nations have projects in this program. The foreign nation and industry projects are proposed and sponsored by FCDA. There are financial arrangements between the participants and the Civil Defense Administration.

The scientific and technical content comprises 10 programs, 54 projects, about 200 shot participations, and requires at NTS a peak population of around 400 scientific and staff personnel over the operational period of several months.

All projects are reviewed by the appropriate scientific and technical test screening and planning committees before acceptance for field testing, and are coordinated with the military effects tests.

The continuing need for effects information parallels and keeps pace with new developments in weapons. The civil effects program stems from this continuing need for up-to-date information on the weapons effects given by a family of nuclear weapons. Our weapons development tests afford an opportunity to augment laboratory experiments with new and useful knowledge from nuclear detonations. Continental tests afford unusually good opportunities to verify in the field various theoretical concepts and laboratory programs which are directed toward complete knowledge of effects on man.

The six general areas of study in the Plumbbob program are as follows:

1. Fallout radiation
2. Prompt-gamma and prompt-neutron radiation
3. Blast effects on structures
4. Blast biology studies
5. Radiological countermeasures and training
6. Instrumentation and supporting services

FALLOUT STUDIES

The fallout studies for Plumbbob represent a continuation of work begun by the Atomic Energy Project of UCLA in the study of the fallout from the Trinity test in 1945. The main purposes of these studies include:

(*a*) Learning to control the availability of radioactive fallout to plants, animals and man;

(*b*) Defining accurately the limits of environmental radiation that can be safely tolerated. Such studies are indispensable also for establishing safety criteria for weapons testing program.

PROMPT-GAMMA AND PROMPT-NEUTRON RADIATION STUDIES

It is necessary to study prompt-neutron and prompt-gamma radiation to obtain data on shielding necessary for shelter design. Better understanding of biological effects of radiation is possible through the advances in gamma and neutron dosimetry. Radiation dosimetry now available makes it possible to measure radiation doses in the field with an accuracy which equals or exceeds measurements in the laboratory. Laboratory and field experiments relating to radiation effects on animals have pointed up the necessity for a better-defined relation between biological effects on animals and on humans. This has reemphasized the value and foresightedness of the medical studies through the Atomic Bomb Casualty Commission that have been underway in Japan since 1946. The ABCC files contain clinical records of more than 4,000 well-documented cases of survivors. These afford data which would be more meaningful to all radiation medicine in the world if we knew the varying doses that individual cases received under the known shielding conditions and distances that prevailed.

During Operation Plumbbob we will initiate studies that are expected to establish the angular distribution of radiations at several distances in air so that total effect on persons inside structures can be evaluated. These basic data will permit the next phase of a long-range program to begin. This will be the determination of the attenuation and scattering by structural and other shielding materials, including terrain. Around 65 percent of the survivors whose cases are adequately documented were shielded in light wood houses of unique construction and geometry, and it is therefore necessary and important that these materials and geometrical configurations be studied to evaluate the shielding afforded to individuals in such structures.

The results of the Plumbbob studies, plus subsequent pilot and laboratory experiments, will permit detailed planning for a third phase of the overall program, the end-product of which is expected to establish the individual doses in terms of gamma and neutron radiation for the medical records developed by ABCC through 10 years, study of survivors. The benefits that could result from this are enormous. Obtaining new data on doses, and through use of the ABCC medical records, their subsequent effects may well provide information that will permit the administration of improved treatment of radiation effects on man. Two of the major laboratories of the AEC are cooperating on this program. They are Oak Ridge National Laboratory and Los Alamos Scientific Laboratory. In addition, the Division of Biology and Medicine of the AEC is being assisted in

this program by the Brookhaven National Laboratory, the Air Force School of Aviation Medicine, the Army Surgeon General's Office, and the Naval Medical Research Institute.

BLAST EFFECTS ON STRUCTURES

Studies of blast effects on shelters and structures are sponsored principally by FCDA. Included are reinforced-concrete home-type shelters for a few persons and so-called mass shelters, capable of holding large numbers of people as well as performing dual functions, such as serving as a parking garage in ordinary times. Also, there are some industry-sponsored items; namely, a protective vault for records, and a windowless reinforced-clay masonry structure. Three reinforced-concrete dome structures are being subjected to overpressures in several ranges to obtain engineering design data for use in future mass shelter designs. Through a United States architect-engineering firm and FCDA, France and West Germany are testing a number of shelters of their design. This represents the first time other nations have included structures or otherwise participated in the Civil Effects Test Group. In addition, a variety of valves, devices, and equipment will be tested for use as shelter components.

BLAST BIOLOGY

The present state of knowledge makes mandatory further studies relating to blast biology. These investigations are being carried out by the Lovelace Foundation and are directed toward obtaining more information on the primary, secondary, and tertiary effects of blast. They are a continuation of the work begun during 1953–55, where together with other valuable data for the first time a means was devised of obtaining usable information on numbers and types of missiles (flying bricks, timber, glass, etc.) per unit area and on the penetrability of glass and masonry fragments and other small missiles likely to be produced in an urban area that has been subjected to a nuclear blast. It is expected that the studies during plumbbob will provide equally valuable information on the problems associated with biomedical effects of static pressures and dynamic pressures sufficiently strong to translate bodies the size and weight of a man from a state of rest to a state of motion.

COUNTERMEASURES AND TRAINING

One of the most important new programs that will be initiated during Plumbbob is work by the Naval Radiological Defense Laboratory on countermeasures against fallout radiation. The proof-testing of radiological shelters and typical buildings is expected to produce data useful in practical applications and guidance for planning a long-range program on methods of survival and continuing occupation of areas that have been subjected to heavy radioactive fallout. This program is designed to provide confirmation and applicability of laboratory theories and methods of decontamination to the large-scale recovery of areas contaminated by radioactivity and in addition to develop data on scaling from low yield to megaton detonations.

Our program includes several training exercises and an offsite radiological defense project of especial importance to civil defense. In addition, other projects will include the field testing of aerial monitoring equipment, and the indoctrination and training of radiological defense personnel drawn generally from State and community civil defense organizations. The field testing of commercially produced radiation detection instrument is included in these projects.

INSTRUMENTATION AND SUPPORTING SERVICES

The instrumentation and supporting services concerned with radiation, blast, thermal, technical photography, are self-explanatory and are designed to give necessary measurements and records to project authors for use in reaching conclusions and in the preparation of technical reports. To supplement and make these physical measurements more meaningful, biological materials, and dosimeters are used to provide data for correlating exposures with effects and extrapolating the findings to probable physical effects on human beings. This requires for Plumbbob several species of animals such as mice, guinea pigs, primates (monkeys), dogs and swine—all of which you will recognize as routinely used in biomedical research.

SUMMARY

It is emphasized that probably the most significant aspect of the Civil Effects Test Program, Operation Plumbbob, is the coordination of continuing laboratory research and less frequent test activities in planning projects to provide information essential to an adequate understanding of nuclear effects on life in all its phases. This coupling provides a continuous flow of basic data usable in immediate practical application and in planning of future research into the means of national self-protection, individual survival, and accommodations of medical practice to the atomic era.

In any case, peace or war, we are already well into the atomic age. Learning to live with the byproducts of nuclear reactions is now necessary and urgent. In addition to the necessity for developing military strength, the weapons testing programs furnish a unique opportunity for providing indispensable information to this end. Additionally, opportunity is provided for the training of key personnel in the theoretical and practical aspects of dealing with environmental radiation in the great variety of situations in which it occurs.

Dr. DUNHAM. He is so busy at the tests. I have also the remarks he made at a press conference at the beginning of Operation Plumbob, which again outlines the civil-effects program.

Representative HOLIFIELD. I think it is very important that the American people know that these tests in Nevada have a much greater effect than just the testing of weapons. There is the testing of materials, the learning of how to protect from radiation, the effect on animals and all of these things which go toward furthering our knowledge of radiations involved in these Nevada tests.

Dr. DUNHAM. That is very true. It not only gives us basic knowledge for all radiation problems, but as long as there is a possible threat of nuclear warfare, it gives us information that is absolutely vital for the defense of the country.

Representative HOLIFIELD. I think the listing of the research projects which you have made in this speech and also the listing of contracts, which are another part of the record, will assure the American people that there is a deep concern and a very wide range of studies in these fields, and that we are not being careless or indifferent regardless of differences of opinion as to what the effects mean. We are at least trying to scientifically get these effects.

Dr. DUNHAM. Yes.

Representative VAN ZANDT. Dr. Dunham, in connection with the use of water for the purpose of attacking the radiation hazard, is that possible? What I have in mind is a pressurized water hose being used to wash off the contamination.

Dr. DUNHAM. That is no question you can hose the fallout material off. Certainly the type of material which came down in the Marshall Islands, which was visible large particles, one could get rid of a great deal by ordinary pressure.

Representative VAN ZANDT. What about the ordinary sprinkler?

Dr. DUNHAM. You have to watch that if you hose it off the roof and sides of the building, and it collects at the edge of the building, you may actually build up more radiation intensity in the building than if you left it alone. One actually has to move it some distance. This becomes quite a problem sometimes.

Representative VAN ZANDT. I understand this practice has been very effective aboard ship.

Dr. DUNHAM. Very effective. I think NRDL has done extensive studies.

Representative HOLIFIELD. I think that distinction between a ship and land is very important because as you say the removal from the walls or roofs of the building means that you are removing it into the soil where people walk.

Mr. DUNHAM. That is right.

Representative HOLIFIELD. Previous testimony from, I believe, Dr. Alexander from the Agriculture Department, was to the effect that it was very difficult to leach this material from the soil.

Dr. DUNHAM. That is correct. It takes years.

As I see it, the areas of greatest immediate need for information are:

1. Better predictability of the properties of nearby strontium 90 fallout: By that I mean whether it is soluble or relatively insoluble, and there is evidence that the near-in fallout is less soluble, and would be less available to plants than the fine particles—better predictability of the properties of nearby strontium 90 fallout, that having tropospheric dissemination, and that getting into the stratosphere for each type of weapon and for each circumstance of burst (ground, low air, high air, etc.). This is very difficult and can only be learned at weapons tests.

2. Amount and distribution of strontium 90 and other fission products in the stratosphere and more precise estimates of holding time in the stratosphere: We will know much more about the first of these in a year's time.

As Dr. Libby indicated, we will know considerable more about that in a year's time as a result of our balloon sampling.

3. Strontium 90 toxicity: In 10 to 15 years we will have experimental data in dogs which will firm up the maximum permissible body burden of strontium 90 for all ages and will have determined whether strontium 90 is ever leukemogenic in dogs and presumably in man, and will have gone a long way to settling whether or not the bone tumor effects of strontium 90 have a threshold.

4. True doubling dose for mutation rate in human germ cells; this will take many years to accomplish, if ever.

5. Tolerable mutation rate for the human race: I use the word "tolerable" advisedly. This again will take many years and will never be exact because of human social patterns.

6. Leukemogenesis and effects on life span by low level external radiation exposures: A study on the latter in several thousand mice at Argonne National Laboratory will be completed in 2 to 3 years. A small scale study in dogs has been going at the University of Rochester for 6 years. This has another 8 to 10 years to go. Related to this is an intensified effort to get more acurate estimates of the true exposures of the Japanese irradiated at Hiroshima and Nagasaki. We attack this from two standpoints. One is to get more exact information about the exact location of the survivors, the type of structures, by aerial photography, and by studies now going on in Nevada attempting to get a much better idea as to what the shielding properties of different materials are, housing materials and the like, to the various types of mixtures of neutrons and gamma rays characteristic of the two weapons detonated in Japan.

I have transmitted separately a more detailed summary statement of the Atomic Energy Commission's biomedical program related to biological hazards of radiation.

(The detailed summary referred to follows:)

U. S. ATOMIC ENERGY COMMISSION, DIVISION OF BIOLOGY AND MEDICINE

Summary—Studies and research projects on fallout problems and related research on the biological hazards of radiation

	Scientific man-years	Amount (in thousands)
1. Sampling and analysis of radioactive fallout, including fission product toxicity and transport:		
(a) Sampling and analysis of radioactive fallout (Sunshine project)	253	[1] $1,193
(b) Research on the biological hazards of radioactive fallout (Sunshine project)		[1] 4,424
2. Effects of radiation on humans, mammals, and other organisms (exclusive of genetic studies)	449	9,168
3. Treatment and methods of ameliorating radiation effects	60	1,338
4. Genetic effects of radiation:		
(a) Studies of human genetics and of genetic effects of radiation on human cells and tissue culture	12	154
(b) Experimental studies of the genetic effects of radiation on species other than man	71	1,314
5. Biochemical and microbiological studies of radiation effects	110	1,472
6. Environmental studies	4	52
7. Dosimetry research: The development of improved methods of measuring radiation	47	1,026
Total	1,006	20,141

[1] A breakdown of these totals appears in the following pages.

In addition to the research programs recapitulated above as concerned with fallout problems and related research on the biological hazards of radiation, the AEC's Division of Biology and Medicine supports a sizable program of research involving the utilization and better understanding of nuclear energies.

This portion of the program involves cancer research and other atoms for peace uses; radioisotopes in medicine; improvement of crops in agriculture; and many other projects less closely related to the fallout problem.

[In thousands of dollars]

Installation	Radiation effects on biological systems (1)	Combating radiation detrimental effects (2)	Beneficial applications (3)	Biomed problems (4)	Dosimetry and instrumentation (5)	Total
1. Argonne Cancer Research Hospital			1,240			1,240
2. Argonne National Laboratory			440		180	620
3. Brookhaven National Laboratory	300		1,197		110	1,607
4. University of California Radiation Laboratory	137	170	1,117		73	1,497
5. University of California Medical School			235			235
6. University of California at Los Angeles	120	182	140	225	50	717
7. GE Co., Hanford Works					286	286
8. ORINS			667			667
9. University of Rochester			115	88	41	244
10. Knolls Atomic Power Laboratory				88		88
11. University of Tennessee			50			50
12. Health and Safety Laboratory, New York				538		538
Total	557	352	5,201	939	740	7,789
13. University and other institution laboratory research projects						3,668
Total, other biological and medical research projects						11,457
Total AEC biology and medicine research program for fiscal year 1957						31,598

Sunshine Project: Studies and Research Projects on Fallout Problems and Related Research on the Biological Hazards of Radiation

1. Sampling and analysis of radioactive fallout, including fission product toxicity and transport

(A) SAMPLING AND ANALYSIS OF RADIOACTIVE FALLOUT

Institution	Title	Investigator	Number of scientists	Number of supporting personnel	Fiscal year 1957 project amount
Agriculture, U. S. Department of, Soil Conservation Service.	Collection and Preparation of Samples of Soils, Plants and Animals for Calcium and Strontium Analyses.	T. Alexander	3	3	$29,000
Armour Research Foundation of the Illinois Institute of Technology.	Efficiency of Scavenging Devices Used in Determining Fallout.	J. Rosinski	6	1	25,000
California, University of, Scripps Institute of Oceanography.	Proposal for Use of Nuclear Tools in Oceanographic Research.	H. E. Suess and H. Craig	4	1	37,000
California, University of, College of Agriculture	Study of the Decontamination of Soils Containing Radioactive Elements and Salts.	R. Overstreet	6	2	17,000
Chicago, University of, Chicago Midway Laboratories.	Study of High Altitude Sampling Techniques	R. Hogness	4	4	47,000
Columbia University, Lamont Geological Observatory.	Distribution of Certain Fission Product Activities	J. L. Kulp	5	8	105,000
Commerce, U. S. Department of, Weather Bureau.	I. Transport of Bomb Debris, and II. A-Bombs and Weather.	H. Wexler	3	3	43,000
General Mills, Inc.	Texas Balloon Flight Operation	H. Demorest	1	1	41,000
	Upper Atmospheric Monitoring Program	J. Gravelle	5	10	221,000
Health, Education, and Welfare, Department of, Public Health Service.	Establishment of a Radiation Surveillance Network	J. G. Terrill, Jr.	2	2	38,000
Interior, U. S. Department of, U. S. Geological Survey.	Airborne Monitoring Program				6,000
Isotopes, Inc.	Radiostrontium Analysis of High Altitude Filter Paper Samples.	H. L. Volchok	1	1	6,000
	Radiostrontium Analysis	do	3	3	52,000
Navy, U. S. Department of, Naval Research Laboratory.	Radioactivity Monitoring Program	I. H. Blifford, Jr.	3	3	20,000
Nuclear Science and Engineering Corp.	Radiostrontium Analysis of High Altitude Filter Paper Samples.	R. A. Brightsen	1	1	18,000
	Radiostrontium Analysis	do	3	3	52,000
California, University of at Los Angeles (atomic energy project).	Factors Influencing the Biological Fate and Persistence of Radioactive Fallout—Operation TEAPOT.	K. Larson, R. G. Lindberg, and J. W. Neel.	21	40	125,000
	Phenomenology of Fallout at Near Distance (within 300 miles of Nevada site).	do			
	Radiochemical Analysis of Certain Marine Samples	do			
	Studies to Document the Occurrence of Radioactive Debris Resulting from Weapons Testing Programs.	do			

	Radio-Ecological Survey of Areas Adjacent to NTS and in Selected Areas up to 600 Miles Distance.	do			
	Environmental Decay Studies	do			
	Study of Strontium and Calcium Relationship in Bone, Plant and Soil—Sampling and Analyses.	R. E. Nusbaum	6	7	39,000
	Correlation of Radiation Intensity from Fallout Material and Weapons Yield.	L. Baurmash	10	11	55,000
	Study of Solubility and Radiostrontium Content of Soils from Various Distances from Ground Zero to Determine Variations Due to Fractionation of Radioisotopes.	do			
	Study of Physical Characteristics of Fallout Material	do			
	Phenomenology of Fallout—TEAPOT SERIES	do			
	Study of the Fluctuation of Radioactivity Levels at Selected Sites in California.	do			
	Fluctuations in Radioactivity Levels in Known Contaminated Areas Adjacent to NTS.	do			
	Studies to Improve Methods of Sampling Radioactive Particulate Materials.	do			
	Studies on Biophysical Aspects of Fallout Phenomenology During NTS Test Series—Proposed in 1957 and 1958.	do			
	Theoretical Studies and Preparation of Summaries on Fallout Using Data Accumulated During Past Test Series.	do			
General Electric Co., Hanford operations office	Pacific Sample Analysis	D. L. Reid	3	3	10,000
Health and Safety Laboratory, New York operations office.	Operation of Gummed Film Network, Collection and Counting of Samples; Other Measurements.	I. B. Whitney	1	1	46,000
	Recording and Analysis of Data from Gummed Network	A. E. Brandt	2	2	35,000
	Collection and Analysis of Samples for Specific Radioisotopes, Uptake of Sr-90.	J. H. Harley and E. P. Hardy, Jr.	5	6	53,000
	Analysis of Marine Samples from the Pacific, Including Tuna, Water, Plankton, Coral.	J. H. Harley	3	3	21,000
	Analysis of Field Samples from Operation Redwing and Related Activity.	R. T. Graveson	1	1	4,000
	Analytical Staff Assistance, etc	I. B. Whitney	1	1	20,000
	Basic Developmental Studies in Trace Radiochemistry	J. H. Harley and G. A. Welford.	3	3	22,000
	Special Studies in Evaluation of Hazards Resulting from Radioactive Fallout at AEC and Contractor Installations.	A. J. Breslin	1	1	6,000
Subtotal, sampling and analysis of radioactive fallout.			107	125	1,193,000

SUNSHINE PROJECT: STUDIES AND RESEARCH PROJECTS ON FALLOUT PROBLEMS AND RELATED RESEARCH ON THE BIOLOGICAL HAZARDS OF RADIATION—Continued

1. Sampling and analysis of radioactive fallout, including fission product toxicity and transport—Continued

(B) RESEARCH ON THE BIOLOGICAL HAZARDS OF RADIOACTIVE FALLOUT

Institution	Title	Investigator	Number of scientists	Number of supporting personnel	Fiscal year 1957 project amount
Agriculture, U. S. Department of, Soil and Water Conservation Branch.	Accumulation and Movement of Fission Products in Soils and Plants.	R. F. Reitemeier	10	3	$80,000
Arizona, University of	Utilization of Phosphorus from Biological Material and Uptake of Strontium by Various Type Crops.	W. H. Fuller and W. T. McGeorge.	2	------------	5,000
California, University of, agricultural experiment station.	Effects of Sr-90 Administered During the Growth of the Dog.	A. C. Anderson	2	2	29,000
California, University of	Study of Internal or Metabolic Factors and the External or Environmental Factors Affecting Ion Absorption by Plants.	L. Jacobson and R. Overstreet.	3	3	10,000
Columbia University, Lamont Geological Observatory.	Studies of Circulation of the Deeper Oceanic Water	M. Ewing	3	4	50,000
Emory University	Long-Range Effects of Radiation on Natural Populations and Communities of the Granite Outcrops.	R. B. Platt	2	3	10,000
Hawaii, University of	Radioisotope Uptake in Marine Organisms with Special Reference to the Passage of Such Isotopes a sare Liberated from Atomic Weapons Through Food Chains Leading to Organisms Utilized as Food by Man.	R. W. Hiatt	3	1	35,000
Idaho State College	Development of Analytical Methods for the Determination of Small Amounts of Strontium, Uranium and Fluoride.	A. E. Taylor	2	2	15,000
Interior, U. S. Department of, Fish and Wildlife Service.	Accumulation of Fission Products by Marine Fish and Shellfish.	W. A. Chipman	5	11	43,000
Jefferson Medical College of Philadelphia	Effects of Radioactive Particulates in Lung Tissues	H. Brieger	3	3	22,000
Johns Hopkins University	Investigation of the Mechanism of Bone Deposition and Related Physiological Studies.	J. E. Howard	4	7	23,000
Kansas, University of	Study of Deposition and Excretion of Bone-Seeking Radioisotopes.	F. E. Hoecker	2	3	20,000
Little, Arthur D., Inc	Studies on the Effects of Natural and Artificial Radioactivity on the Electrical Properties of the Atmosphere.	B. Vonnegut	5	------------	25,000
Marquette University School of Medicine	The Pathological Effects of Radioactive Isotopes of Calcium and Strontium on Bone and Soft Tissue.	J. F. Kuzma	2	2	16,000
Massachusetts General Hospital	Effects of Radioactive Iodine on Biology of the Thyroid Gland.	O. Cope and J. B. Stanbury	5	2	13,000
	The Metabolism of Calcium and Strontium as Disclosed by Tracer Studies on Patients with Thyroid and Related Diseases.	J. B. Stanbury	3	2	20,000

Massachusetts Institute of Technology	Radium, and Mesothorium Poisoning, and Dosimetry and Instrumentation Techniques in Applied Radioactivity.	R. D. Evans	4	5	176,00
Miami, University of	A Study of Factors Affecting the Selective Uptake and Retention of Zn–65 by the Dorsolateral Prostate of the Rat, and a Study of the Role of Zinc in the Physiology of the Prostate.	S. A. Gunn	2	1	11,000
Michigan State University	I. The Absorption and Utilization of Radioactive Minerals Applied to the Leaves of Plants; II. The Absorption and Utilization of Ruthenium by Plants; III. The Leaching of Nutrients from Leaves of Plants.	H. B. Tukey	2	1	25,000
Minnesota, University of	Effects of Radioactive Substances on Plant Pathogens and Other Microorganisms.	J. J. Christensen and E. C. Stakman.	3	7	23,000
Montefiore Hospital for Chronic Diseases	A Study of the Distribution and Excretion of Lanthanum and the Rare Earth Elements.	D. Laszlo	6	3	25,000
	Dynamics of Strontium Distribution in the Body	do	4	3	22,000
Navy, U. S. Department of, Naval Radiological Defense Laboratory.	Radiological Countermeasures System Operations Training and Evaluation.	P. Tompkins	15	17	59,000
	Fallout Distribution and Radiation Contours—Low Yield Detonation.	do	------	------	------
New Mexico Highlands University	Selective Denuding Action of Atomic Radiation on Vegetation of Some Nevada Sites and Gross Changes in the Surviving Flora.	L. M. Shields	2	------	13,000
New York, State University of, Research Foundation.	Effects of Irradiation on the Calcifying Mechanism of Epiphyseal Cartilage.	A. Hirschman	2	------	10,000
New York University, Bellevue Medical Center	The Distribution and Persistence of Radioactive Aerosols in the Lungs of Animals.	B. Altshuler	5	2	19,000
	Tissue Reactions to Intrapulmonary Radiation	M. Kuschner	1	3	15,000
	Aerosol Retention Studies	N. Nelson	5	3	20,000
North Carolina, University of	The Double Isotope Effect of Ca–45 and Sr–90 on the Pattern of Distribution in the Body, Particularly in Bone.	C. D. VanCleave	2	2	10,000
Ohio Agricultural Experiment Station	Detailed Characterization of Soil and Vegetation on Selected Sites to Serve as Basis for Future Evaluation of Effects of Radioactive Contamination.	N. Holowaychuk	3	2	30,000
Pittsburgh, University of	Hazard from Inhaled Radioactive Particulate Matter	H. Cember	3	4	20,000
Presbyterian and St. Luke's Hospital	Mobilization of Radioactive Emitters from Bone	R. D. Ray	2	1	15,000
Rochester, University of	Dynamic Aspects of Skeletal Metabolism	A. R. Terepka	4	2	17,000
Tennessee, University of	The Metabolism of Alkaline Earth Metals by Bone	F. W. Lengemann	1	4	9,000
	Vegetation Studies Related to Disposal of Radioactive Wastes.	R. E. Shanks	4	1	17,000
	Studies in Iodide Metabolism	L. Van Middlesworth	5	2	19,000
Utah State University	Effect of Radioactive Elements and Radiation on Ewes Maintained on Different Levels of Nutrition.	L. E. Harris	3	3	10,000
Utah, University of	Toxicity Studies of Plutonium and Other Radioactive Substances in Animals.	T. F. Dougherty	10	27	250,000
Western Reserve University	A Study of the Physiological Function and Histological Changes of Thyroids Irradiated with Radioactive Iodine.	B. M. Dobyns	2	2	3,000
Woods Hole Oceanographic Institution	Studies on the Background Radiation and Flow of Deep Ocean Currents.	A. B. Arons	2	2	9,000

SUNSHINE PROJECT: STUDIES AND RESEARCH PROJECTS ON FALLOUT PROBLEMS AND RELATED RESEARCH ON THE BIOLOGICAL HAZARDS OF RADIATION—Continued

1. Sampling and analysis of radioactive fallout, including fission product toxicity and transport—Continued

(B) RESEARCH ON THE BIOLOGICAL HAZARDS OF RADIOACTIVE FALLOUT

Institution	Title	Investigator	Number of scientists	Number of supporting personnel	Fiscal year 1957 project amount
Argonne Cancer Research Hospital	Metabolism of Bone Seeking Radio-Elements in Human Beings.	R. J. Hasterlik	2	2	32,000
Argonne National Laboratory	Toxicity of Radiostrontium	M. P. Finkel			
	Study of Sr-90 Metabolism in Laboratory Animals	W. E. Kisielski			
	Toxicity of Calcium 45	M. P. Finkel			
	Toxicity of Sr-90, Ca-45, Sr-89, Ra-226, etc	do			
	Isotope Toxicology and Pathology, Morphologic Aspects of Radiation Carcinogenesis.	H. Lisco	6	7	
	Study of Radiation Damage in Bone	L. A. Speckman, T. W. Speckman, and W. P. Norris.			
	Dynamics of the Metabolism of the Alkaline Earths in Laboratory Animals and Men.	W. P. Norris	8	9	346,000
	Radioelement Metabolism in Humans	A. E. Stehney and L. D. Marinelli.	3	3	
	Toxicity and Metabolism of Radioactive Ruthenium (Ru-106).	A. M. Brues and H. Walton	2	1	
	Natural Environmental Radium Levels and Bone Tumors	H. Auerbach	1	1	
	Radium Levels in Bone Tumor Patients	H. Auerbach and W. P. Norris.	2	1	
	Determination of Permissible Dosage of Isotopes	A. M. Brues	1	1	
	Meteorology Studies	H. Moses and H. A. Schultz	5	6	
Brookhaven National Laboratory	Meteorological Research	M. Smith and M. Fox	2	2	65,000
California, University of, at Los Angeles (atomic energy project).	Evaluation of Acute Inhalation Hazard from Radioactive Fallout Materials.	G. Taplin	3	3	
	Sr-90 Poisoning and Research for Therapeutic Measures	N. MacDonald	6	7	
	Fixation and Extractability of Fission Products Contaminating Various Soils and Clays.	E. Romney			
	Effect of Organic Matter on Availability of Fission Products to Crop Plants.	do			
	Effects of Calcium Carrier and Stable Strontium in Plant Uptake Radiostrontium.	do			
	Effects of Cation Balance, Root Temperature, Killing of Roots and Photoperiod on Calcium, Phosphorus and Strontium Uptake by Barley Plants.	do			
	Isolation and Identification of Biochemical Substances in Plant Tissues Labeled with Radioactive Fission Products.	do	13	14	518,000
	Effects of Complementary Ions, Texture and Ph on Plant	do			

	Uptake of Radiostrontium.				
	Chemistry of Fission Products in Plant Tissue	do			
	Effects of Ionizing Radiations from Fission Products on Certain Metabolic Systems in Plants.	do			
	Determination of the Maximum Levels of Fission Product Uptake by Crop Plants Grown on Agricultural Soils.	do			
	Digestibility of Fission Product Elements in the Diet	R. Monroe	6	7	
	Comparative Dynamics of Strontium and Calcium	do			
California, University of, Radiation Laboratory	Radiobiological Studies	J. G. Hamilton	12	13	165,000
	Internal Irradiation and Hematological Response	J. F. Lawrence, et al	4	4	46,000
General Electric Co., Hanford operations office	Absorption of Radioelements into Plants	F. P. Hungate	4	4	60,000
	Fission Product Absorption and Metabolism	R. C. Thompson	4	4	89,00
	Biological Effects of Iodine-131	L. K. Bustad	3	3	100,000
	Effects of Process Effluents on Aquatic Organisms	R. F. Foster	4	4	128,000
	Plutonium Absorption and Metabolism	R. C. Thompson	4	4	94,000
	Toxicity and Metabolism of Inhaled Radioactive Particles	R. W. Wager	7	8	110,000
	Ground Waste Investigations	D. W. Pearce	1	1	48,000
	Soil Chemistry	do	3	3	47,000
	Soil Physics	do	2	2	71,000
	Meteorological Transport and Diffusion	do	3	3	59,000
	Particle Translocation	do	4	4	51,000
Los Alamos Scientific Laboratory	Cesium-137 Studies (and Strontium)	W. Langham	5	6	100,000
Oak Ridge Institute of Nuclear Studies	Metabolism of Radiostrontium in Humans and Experimental Animals.	C. L. Comar	1	1	25,000
Rochester, University of (atomic energy project)	Biological Effects of Internally Deposited Radioactive Isotopes in Rats and Monkeys.	L. Tuttle and R. Baxter	2	2	35,000
	Biological Effects of Inhaled Radioactive Materials	J. Stannard	13	14	190,000
	Mechanism of Bone Deposition, Mobilization and Transport of Radioelements.	W. F. Neuman	18	20	117,000
	Chronic Effects of Sr-90 in the Monkey	L. Tuttle, R. Baxter, and M. Goldman.	3	3	36,000
	Dosimetry of Fission Product Fallout Fields	A. H. Dahl	2	2	21,000
	Plutonium Inhalation Field Study	J. Stannard	3	3	35,000
	Fate of Radium and Its Daughter Products in Living Organisms.	J. Hursh	1	1	15,000
	Aerosol Problems	H. Hodge and K. Lauterbach.	2	1	
	Toxicological Studies of Elements and Compounds of Interest to the AEC.	H. Hodge and E. Maynard	2	1	318,000
	Effects of Heavy Metals on Cells and Tissues	A Rothstein	1	1	
	Biological Transport Mechanisms for Particulates	R. Gosselin	1	1	
	Pharmacology and Toxicology of Thorium Isotopes	J. Stannard	1	1	20,000
	Biological Effects of Internally Deposited Radioactive Isotopes in Rats and Monkeys.	L. Tuttle and R. Baxter	2	1	30,000
Tennessee, University of	Fission Product Metabolism in Animals	J. A. Ewing	1	1	94,000
Washington, University of	Evaluation of Radioactive Contamination in Western Pacific—Radiological Study of Rongelap Atoll, etc.	L. R. Donaldson	14	15	111,000
Subtotal, research on the biological hazards of radioactive fallout.			335	340	4,424,000
Total, sunshine project			442	465	5,617,000

2. Effects of radiation on humans, mammals, and other organisms (exclusive of genetic studies), $9,168,000

Institution	Title	Investigator
California, University of	The Effect of Radiation on Work Capacity and Longevity of the Dog.	A. C. Andersen, G. H. Hart.
Cedars of Lebanon Hospital	Chemical Studies on Connective Tissues of Animals Aged Prematurely by Irradiation (Assessment of Biochemical Age).	H. Sobel.
Chicago, University of	Radiosensitivity of the Lymphocytes	P. P. H. DeBruyn.
	Bacteriological Aspects of Radiation Sickness	C. P. Miller.
	A Study of the Effect on Gastric Tissues of Irradiation Therapy in Peptic Ulcer.	W. D. Palmer.
	The Physiological Factors Involved in Antibody Synthesis and in the Modification of the Immune Process by X-Irradiation.	W. H. Taliaferro.
	The Effect of Whole Body Ionizing Radiation on the Quality of Antibody.	D. W. Talmage.
Children's Medical Center	The Effect of Irradiation on Induction of Pituitary Tumors.	J. Furth.
Columbia University	Effect of Ionizing Radiation on Nerve Tissue	D. Nachmansohn.
Emory University	Effect of Radiation on Learned Behavior, Problem-Solving Ability and Neural Mechanisms of Monkeys.	A. J. Riopelle.
Florida, University of	Effect of Radiation on the Uptake of Large Organic Molecules by the Liver and Spleen of the Mouse.	F. E. Ray, M. F. Argus.
George Washington University.	The Dose-Incidence Relationship of Beta Radiation Induced Skin Cancer in the Rat.	L. K. Alpert.
Harvard University	The Effect of Ionizing Radiations on Peripheral Nerve.	E. L. Gasteiger.
	Radiation Effects on the Lung	S. Warren.
Illinois, University of	A reevaluation of Radiation Injury (B-rays) of the Skin by a Direct Method Approach.	S. R. Rosenthal.
Iowa, State University of	A Quantitative and Morphologic Study of Radiation Induced Cataracts.	T. C. Evans and P. J. Leinfelder.
Johns Hopkins University	The Mechanism of the Activation of Latent Epidemic Typhus Infections in the Laboratory Animals and in Humans by X-ray.	W. H. Price.
Kansas, University of	Immunological Study of Radiation-Induced Damage to Biological Systems.	C. A. Leone.
Kresge Eye Institute	Effects of Neutrons and Other Radiations on the Ocular Lens.	V. E. Kinsey.
Marquette University	Temperature Prevailing During Exposure as a Modifying Factor in the Dose-Response Relationship of X-rayed Mammalian Skin.	J. P. O'Brien.
Massachusetts General Hospital.	A Biochemical Study of the Effects of Radiation on Cells.	P. C. Zamecnik, I. T. Nathanson.
Miami, University of	A Quantitative Study of the Effects of Radiation on the Blood Capillaries of Normal Animals.	E. L. Chambers.
Michigan, University of	Effects of Irradiation on the Localizing Response (to Antigen) of Different Tissues in Immunity.	A. C. Curtis.
Syracuse University	Effects of X-Radiation Upon the Renal-Endocrine System.	W. R. Boss.
Minnesota, University of	Effects of Ionizing Radiation Upon Tissue Metabolism.	W. D. Armstrong, W. O. Caster.
	9Toxic Effects of Irradiation	J. F. Marvin, F. J. Lewis.
Nebraska, University of	Effects of Cranial X-Irradiation on Psychological Processes in Rats.	W. J. Arnold.
Nevada, University of	Range Livestock Production Adjacent to Nevada Proving Grounds.	V. R. Bohman.
New England Deaconess Hospital.	The Effects of Ionizing Radiation on the Developing Mammalian Nervous System.	S. P. Hicks.
	Acute and Chronic Radiation Injury	S. Warren.
New York University	Histochemical Studies of Metabolic Alterations in Rats Receiving Lethal and Sublethal Doses of Radiation, with Emphasis on Terminal Vascular Bed.	B. W. Zweifach, B. P. Sonnenblick.
New York University, Bellevue Medical Center.	Study of the Biological Effects of Ionizing Radiation (Alpha and Beta) on Human Skin.	M. B. Sulzberger.
Nuclear Science and Engineering Corp.	A Toxic Substance Produced by Irradiation	Abraham Edelmann.
Oregon, University of, Medical School.	Studies of Hemic Effects of Radioisotopes, X-rays and of Adrenocortical Hormones.	E. E. Osgood, A. J. Seaman.
Pennsylvania, University of	Changes in the Capillary Fragility and the Colloidal Properties of Blood Following Irradiation.	L. V. Heilbrunn.
Pittsburgh, University of	The Study of the Effects of Radiation on the Immune Response.	F. J. Dixon.
Rochester, University of	Individual Response to Ionizing Radiation in Animals and Patients.	L. H. Hemplemann.

2. Effects of radiation on humans, mammals, and other organisims (exclusive of genetic studies), $9,168,000—Continued

Institution	Title	Investigator
Stanford University	Marine Biological Survey of Western Pacific, with Special Emphasis in the Palau Island (Survey Area Includes New Guinea Region, Philippine, Caroline and Mariannas Islands).	R. R. Harry, Jr.
	Biological and Medical Investigations with the 70 Mev. Linear Electron Accelerator.	H. S. Kaplan, E. L. Ginzton.
Tennessee, University of	The Response of the Reticulo-Endothelial System to X-irradiation.	N. R. DiLuzio.
	A Study of the Effects of Cobalt-60 Gamma Irradiation on Infection and Immunity.	W. M. Hale.
Texas Technological College	The Effects on Rat Behavior of Developmental Aberrations Induced by Ionizing Radiation in utero.	S. J. Kaplan.
Texas, University of, M. D. Anderson Hospital and Tumor Institute.	Physical and Radiobiological Investigations with 22-Mev. X-rays and Electrons, as Compared with Cobalt 60 and 250-kilovolt X-rays.	W. K. Sinclair.
Tufts College	Study of the Effects of Radiation on Growth	D. Rapport.
Vanderbilt University	Fetal Irradiation and the Patterns of Behavior Development.	G. W. Meier.
Western Reserve University	Investigations of the Biological Effects of Internally Deposited Radioisotopes and Related Radio-Biology Studies.	H. L. Friedell.
Wisconsin, University of	The Effect of Various Forms of Irradiation of the Brain on Learned and Unlearned Behavior of Monkeys and Chimpanzees.	H. F. Harlow.
Yerkes Laboratory of Primate Biology, Inc.	Behavioral Effects of Ionizing Radiation on Chimpanzees of Various Ages.	H. W. Nissen.
Argonne Cancer Research Hospital.	Biological Studies with High Energy Sources	E. L. Simmons.
Argonne National Laboratory	Hemolysin Formation in X-Irradiated Rabbits	B. N. Jaroslow.
	Variations in the Haemagglutination, Electrophoretic and Serological Properties in the Sera of Monkeys Exposted to Low-level Gamma Irradiation.	C. W. Leone. G. A. Sacher.
	Radiation Effects on Immunity to Ascites Tumors.	A. M. Brues. A. N. Stroud.
	Effect of Continuous Irradiation on Tumors	A. M. Brues.
	Continuous Radiation Effects on Cell Division in Tissue Cultures.	A. M. Brues, A. N. Stroud.
	Cell Division Effects in Radiated *Paramecium*	E. L. Powers.
	Beta Irradiation of Skin From Point Sources	A. J. Finkel.
	Biological Effects of Cosmic Rays	A. M. Brues, H. Walton.
	Organ Weight Changes in Irradiated Mice	A. M. Brues, A. N. Stroud.
	Studies on the Effects of Ionizing Radiation on Connective Tissue.	F. Wasserman.
	Primate Radiobiological Program	R. J. Flynn.
	Effects of X-Irradiation on Developing Embryos of the Grasshopper.	T. N. Tahmisian.
	The Effect of X-Irradiation on the Oscillation of Developing Egg Nuclei in Grasshoppers.	Do.
	Theory of Radiation injury and Lethality	G. Sacher, D. Grahn.
	Gamma-ray Toxicity: Lethality	D. Grahn, G. Sacher.
	Gamma-ray Toxicity: Histology, Hematology, Pathology.	G. Sacher, D. Grahn.
	Radiation Effects on Reproduction in the Female Mouse.	M. H. Sanderson, S. P. Stearner.
	Effect and Dose-Exposure-Time Relation of X and Gamma Radiations on Avian Species Other Than the Chicken.	S. P. Stearner, M. H. Sanderson.
	Effect of Dose and Exposure time of Co-60; Gamma Rays on Mortality of Young Chicks.	Do.
	Departure from Additivity when Mixtures of Fission Neutrons and Co-60 Gamma Rays are Administered to Mice.	H. H. Vogel, Jr., J. W. Clark.
	Effects on Mice of Acute Irradiation with Fission Neutrons and Co-60 Gamma Rays.	Do.
	"Recovery" After Irradiation of Mice with Fission Neutrons or Gamma Rays.	Do.
	Chronic Irradiation of Mice by Fission Neutrons	Do.
	Relative Biological Effectiveness (RBE) of Fission Neutrons and Co-60 Gamma Rays.	Do.
	Survival of the Chick Embryo as a Standard Radiobiological Test.	H. H. Vogel, Jr.
	Late Effects of Acute Irradiation with Fission Neutrons and Gamma Rays.	H. H. Vogel, Jr., et al.
	Effect of Natural and X-ray Induced Aging on Susceptibility to Chemical Carcinogens.	H. Ducoff, H. Lisco.

2. Effects of radiation on humans, mammals, and other organisims (exclusive of genetic studies), $9,168,000—Continued

Institution	Title	Investigator
Brookhaven National Laboratory.	Production of Radiation of Desired Character	L. E. Farr.
	Control of Radiation Distribution in Mammalian Organisms.	Do.
	Effects of Radiation	Do.
	Effects of Radiation on Aging in Mice	H. J. Curtis.
	Effects of Radiation on Animal Metabolism	L Nims.
	The Acute Effects of Radiation in Mammals	H. Quastler.
California, University of, at Los Angeles (Atomic Energy project).	Radiation Biology	T. Hennessy.
California, University of, Radiation Laboratory.	Biological Effects of Radiation	C. Tobias, J. Born.
General Electric Co., Hanford operations office.	Beta Irradiation of Skin	L. K. Bustad.
	Relative Biological Effectiveness	F. P. Hungate.
	Radiosensitivity of the Gastrointestinal Tracts	R. C. Thompson.
Oak Ridge National Laboratory.	Radiation Effects on Biological Systems	A. Hollaender.
Rochester, University of (Atomic Energy project).	Studies on the Hemorrhagic State and the Metabolism of Animals Exposed to Ionizing Radiation.	L. Tuttle, L. Miller.
	Study of the Morphological and Physiological Alterations of the White Cells Using Normal and Irradiated Animals.	M. Ingram.
	Studies of the Endocrine Imbalances in the Irradiated Animal.	S. Glasser. F. Brayer.
	Effects of X-Irradiation on the Aging Process in the Rat.	J. B. Hursh.
	Acute Radiation Effects on Whole Body X-Irradiation in Animals.	L. Steadman.
	Effect of X-Irradiation on Spermatogenesis in Dogs.	J. Hursh, G. Casarett.
	Clinical and Biochemical Studies of the Irradiated Dog.	W. Mason.
	Drosophila Melanogaster as a Tool in Radiobiologic and Toxicologic Investigation.	L. Tuttle. R. Baxter.
Tennessee, University of	Radiation Effects on Reproductive Functions in Farm Animals, Sperm Physiology.	J. A. Ewing.
	External Radiation Studies With Large Animals	Do.
National Academy of Sciences Atomic Bomb Casualty Commission.	Studies of the Radiation Effects of the Atom Bomb on the Survivors of Hiroshima and Nagasaki, Japan.	
Columbia University	Biological Action of Ionizing Radiation. Instrumentation for Research.	G. Failla.
Stephen F. Austin State College.	The effect of Radiation on a Natural Population of *Peromyscus gossypinus* (field mouse).	W. M. McCarley.
Los Alamos Scientific Laboratory.	Bio-Medical Problems in Atomic Energy Operations.	
Civil effects test program	Radiological Countermeasures	P. C. Tompkins.
	Biological Assessment of Blast Effects	C. S. White.
	Physical Response to Blast Loadings	L. J. Vortman.
	Radio-Ecological Aspects of Nuclear Fallout	K. H. Larson.
	Instrumentation and Dosimetry	R. L. Corsbie.

3. Treatment and methods of ameliorating radiation effects, $1,338,000

Institution	Title	Investigator
Buffalo, University of	Immunological Factors in Bone Marrow Transplants.	E. Witebsky, M. L. Bloom.
Children's Medical Center, Boston, Mass.	The Nature of Bleeding in Pancytopenia With Special Regard for Thrombocytopenia and the Vascular Defect.	S. Farber.
Jefferson Medical College of Philadelphia.	Transplantation of Preserved Marrow Between Animals and From One Human to Another.	L. M. Tocantins.
Mary Imogene Bassett Hospital.	The Collection, Storage and Use of Human Bone Marrow.	J. W. Ferrebee, E. D. Thomas.
Massachusetts General Hospital.	The Collection, Storage and Use of Human Cadaver Marrow.	B. Castleman, J. W. Ferrebee.
New England Center Hospital	Bone Marrow Research Project	W. Dameshek.
	Physiopathology of Platelets and Development of Platelet Substitutes.	Do.
New York, Research Foundation of State University of.	Experimental Transfusion of Bone Marrow Into Rabbits After Total Body Irradiation	J. H. Ferguson, M. F. Hilfinger.
Parke, Davis & Co	Factors Elaborated by Animal Tissues Which Stimulate Rate of Regeneration of Hemotopoietic Organs of Animals Exposed to Total Body Irradiation With Gamma Rays.	J. K. Weston.
Southwest Foundation for Research and Education.	An Investigation of the Production and the Possible Isolation of Substances Capable of Stimulating Recovery From Radiation by Utilizing Techniques of in vitro Maintenance of Spleen and Other Organs.	N. T. Werthessen.
Tennessee, University of	Physiology of Water and Ionic Balance in Monkeys Subjected to Whole Body Radiation.	R. R. Overman.
Do	Protective Action of Bone Marrow Perfusates and AET in Irradiated Monkeys.	Do.
Yale University	Biologic Implications of Isologous and Heterologous Bone Marrow Repopulation in Irradiated Animals.	J. W. Hollingsworth.
Argonne Cancer Research Hospital.	Radiation Recovery Factor	E. Goldwasser.
	Differential Reduction in Radiosensitivity	L. O. Jacobson.
Argonne National Laboratory	Radiation Protection and Therapy	H. H. Vogel, Jr., J. W. Clark.
	Radioelement Metabolism in Humans; Mechanisms of Spleen Protection.	H. S. Ducoff.
	Protective Factor in Plasma Protein	A. M. Brues, A. N. Stroud.
	Protective Effect of Non-Irradiated Protoplasm in Supralethally X-Irradiated Protozoan Animals.	E. W. Daniels.
	Role of Hydrogen Peroxide and Catalase in Radiation Lethality.	R. N. Feinstein.
	Phenothiazine Derivatives and Other Substances as possible Protective Agents Against the Lethality of Ionizing Radiation.	Do.
	Protective Mechanisms in Radiation Injury	H. M. Pratt.
	Therapy in Radioelement Poisoning	J. Schubert, M. White.
	Radiation Protection of Chicks by Means of Vasoconstrictor Drugs.	J. P. Stearner, M. H. Anderson.
Brookhaven National Laboratory.	Combating Radiation Detrimental Effects	H. Curtis.
Oak Ridge National Laboratory.	Radiation Protection, Living Cells	A. Hollaender.
	Mammalian Radiation Recovery	C. Congdon.
Rochester, University of (Atomic Energy project).	Studies on Therapy of the Radiation Syndrome With Attempted Control of Infection, Hemorrhage and Nutritional and Humoral Imbalances by Standard Medical Procedures.	S. Michaelson.
	Clinical Treatment of Radiation Injury. Tolerance of Animals to Repeated Sublethal Radiation Dosages. Study of Recovery Pattern Following Lethal Dosage of Ionizing Radiation.	J. Howland.
	Effect of Varied Nutritional States on the Survival and Recovery Process of the Irradiated Rat with Particular Interest on Aging, Anemia, and Partial Body Exposures.	T. Noonan.

4. Genetic effects of radiation

(a) STUDIES OF HUMAN GENETICS AND OF GENETIC EFFECTS OF RADIATION ON HUMAN CELLS AND TISSUE CULTURE, $154,000

Institution	Title	Investigator
Johns Hopkins University	The Effects of Ionizing Radiations on Gene and Chromosome Mutation Rates in Normal Human Cells in Tissue Culture.	H. B. Glass.
Long Island Biological Association.	The Study of Spontaneous and Induced Genetic Changes in Mammalian Cells Grown in Tissue Cultures.	M. Demerec and B. P. Kaufmann.
Michigan, University of	Development of Information Concerning (1) Human Mutation Rates; (2) The Accumulation of Deleterious Recessive Genes in Human Populations; and (3) The Manner of Action of Selective Factors on Both Contemporary and Primitive Human Populations.	J. V. Neel.
	Study of Consanguineous Marriage in Japan	Do.
Yale University	Radiation Effects on Mammalian Chromosomes in Tissue Cultures.	N. H. Giles.

(b) EXPERIMENTAL STUDIES OF THE GENETIC EFFECTS OF RADIATION ON SPECIES OTHER THAN MAN, $1,314,000

Columbia University	Studies of Mutations in Populations of Wild House Mice.	L. C. Dunn.
Iowa State College of Agriculture and Mechanic Arts.	Quantitative Study of Lifetime Sickness and Mortality and Progeny Effects Resulting From Exposure of Animals to Penetrating Irradiation.	J. W. Gowen and J. Stadler.
Oregon, University of	Investigations in Population Genetics and Ecology.	D. L. Jameson.
Roscoe B. Jackson Memorial Laboratory.	Quantitative Population Genetics of Mice Under Irradiation.	E. L. Green.
	Attempt to Delineate Inborn Anemias in Mice	E. S. Russell.
Texas, University of	Direct and Indirect Effects of Radiation on Genetic Developmental Systems of Vertebrates.	W. F. Blair.
Amherst College	Genetic Effects of Acute and Chronic Low Level Irradiation with Cobalt 60.	H. H. Plough.
Arkansas, University of	Developmental-Genetic Study of the Effects of X-ray Irradiation in *Drosophila virilis* and *Bufo valliceps*.	F. E. Clayton.
Brown University	Radiation Effects on the Cytoplasm of Habrobracon eggs.	W. Kenworthy.
Chicago, University of	The Genetic Functioning of Heterochromatin	W. K. Baker.
Columbia University	The Population Genetics of Species of Drosophila.	T. Dobzhansky.
Delaware, University of	The Relation of Genome Number to Radiosensitivity in Habrobracon.	A. M. Clark.
Indiana University Foundation.	The Influence of Radiation in Altering the Incidence of Mutations in Drosophila.	H. J. Muller.
Johns Hopkins University	The Action of Radiation and Other Mutagenic Agents—(1) In Inducing Mutation in Drosophila Females; and (2) In Controlling the Action of a Specific Gene Responsible for Suppressing Uncontrolled Growth.	H. B. Glass.
Long Island Biological Association, Inc.	Adaptive Value of Experimental Populations Exposed to Radiations.	B. Wallace.
Mississippi, University of	Chromosome Breakage in Oocytes of *Drosophila*	D. R. Parker.
North Carolina State College of Agriculture and Engineering.	The Genetic and Developmental Effects of Ingested Radioactives in Habrobracon.	D. S. Grosch.
North Carolina, University of	A Study of Genetic Recombination as Influenced by Mutagenic and Nonmutagenic Environmental Agents.	M. Whittinghill.
Northwestern University	Studies on the Radiation Genetics of *Drosophila melanogaster* Females.	R. C. King.
Pennsylvania, University of	Mutation Rates in Mormoniella	P. W. Whiting.
Pittsburgh, University of	Genetic Potential of Certain Populations of *Drosophila persimilis* from the Sierra Nevada of California.	E. B. Spiess.
Texas, University of—M. D. Anderson Hospital and Tumor Institute.	The Effects of Radiations on the Genetic System of Organisms in Relation to Their Physiological and Biochemical Systems.	M. L. Alexander.

4. Genetic effects of radiation—Continued

(b) EXPERIMENTAL STUDIES OF THE GENETIC EFFECTS OF RADIATION ON SPECIES OTHER THAN MAN, $1,314,000—Continued

Institution	Title	Investigator
Texas, University of	Research on Direct and Indirect Effects of Radiations on the Genetic Systems of Organisms.	W. S. Stone.
Emory University	Study of the Influence of Oxygen Level and Temperature on the Effects of Ionizing Radiation.	A. V. Beatty.
Florida, University of Agricultural Experiment Station.	Recovery of Radiation Induced Micromutations in Oats by Recurrent Selections.	A. T. Wallace and F. H. Hull.
Harvard University—Bussey Institution.	The Biological Effect of Radiation; Effects of Irradiation on Chromosomes.	Karl Sax.
Johns Hopkins University	Modification by Supplementary Agents of the Rates of Induced Chromosome and Gene Changes.	C. P. Swanson.
Minnesota, University of	The Genetic Basis and Practical Significance of Mutations Induced in Oats and Barley with Ionizing Radiations.	R. S. Caldecott.
Purdue Research Foundation	Genetic Effects of Thermal Neutron Irradiation in Homozygous Tomatoes.	A. B. Burdick.
Washington, State College of	A Study of Factors that Govern Radiosensitivity in Plants.	R. A. Nilan.
Yale University	Investigations on the Cytogenetic Effects of Radiations.	N. H. Giles.
Argonne National Laboratory	Cytology with Special Reference to Radiation Effects in Animal, Plant and Human Nuclei.	B. R. Nebel.
	Genetic Resistance to X-Irradiation in Inbred Lines of Mice.	C. A. Leone and H. M. Patt.
	Genetics of Radiation Toxicity	D. Grahn and G. A. Sacher.
	Radiation—Induced Recessive Mutations in Mice.	H. M. Slatis.
Oak Ridge National Laboratory.	Genetic and Developmental Effects of Radiation on Mice.	W. Russell.
California, University of Radiation Laboratory.	Radiation and Mutation Rate	C. Stern.
Oak Ridge National Laboratory.	Genetic and Cytogenetic Effect of Radiations	A. Conger and R. Kimball.
Brookhaven National Laboratory.	Radiation Mutations in Plants	C. Konzak.

5. Biochemical and microbiological studies of radiation effects, $1,472,000

Institution	Title	Investigator
Arkansas Medical School, University of.	Studies on the Biochemical and Nutritional Aspects of X-Radiation Injury.	P. L. Day.
Arkansas, University of	The Utilization of Radioisotopes by Vertebrate Embryos.	P. M. Jonston.
Brown University	Penetration of the Gut Wall by Intestinal Bacteria After X-Irradiation.	M. H. Hatch.
California Institute of Technology.	The Genetic and Cytological Effects of High Energy Radiation.	G. W. Beadle.
Chicago, University of	Studies on the Mechanism of Action of Ionizing Radiations.	E. S. G. Barron.
Christian Brothers College	Uranium Complexes with Amino Acids and Peptides.	E. J. Doody.
Columbia University	Study of the Action of Radiation on Deoxypentose Nucleic Acids Having Biological (Transforming) Activity.	S. Zamenhof.
Cornell University	Cytological and Genetic Studies of Bacteria as Related to Effects of Radiation.	M. R. Zelle.
Duke University	The Effects of Ultraviolet Light and Gamma Rays on Cell Lipids and the Physiological Action of Irradiated Lipids.	K. M. Wilbur, F. Bernheim.
Florida, University of	Concentration of Mineral Elements in the Fetus and the Relationship to Placental Transfer of These Elements.	G. K. Davis, R. L. Shirley, A. Z. Palmer.
George Washington University.	Studies of the Effects of Radiation on the Biosynthesis and Degradation of Nucleoproteins and Its Modification by Various Agents.	P. K. Smith.
Hahnemann Medical College and Hospital.	The Biochemical Properties of Microsomes and the Effects of Radiation on Them.	J. S. Roth, H. J. Eichel.
Johns Hopkins University	A Study of Metabolism and Active Transport of Certain Divalent Metals in Tissues and in Isolated Mitochondria, with Special Attention to the Possible Role of Complexing Agents in These Processes.	L. S. Maynard.
	Biochemical Changes Resulting from Mutations Induced by X-rays, Ultaviolet, and Nitrogen Mustard.	W. D. McElroy.
Marine Biological Laboratory	Studies on the Physiology of Marine Organisms Using Radioisotopes.	P. B. Armstrong.
Michigan, University of	Effects of Radiation on the Intermediary Metabolism of Mammalian Skin.	I. A. Bernstein.
	Studies with an X-ray Monochromator and X-ray Irradiation Service Operation.	H. J. Gomberg.
	The Biochemical Effects of Radiation: The Effect of Ionizing Radiation on Nucleic Acid Metabolism.	R. L. Potter.
Notre Dame, University of	Mechanisms Involved in the Action of Radiations on Living Cells.	C. S. Bachofer.
Oklahoma, University of Research Institute.	The Cytology and Genetics of Radiation Resistance in Bacteria.	J. B. Clark.
Pennsylvania, University of	The Internal Organization of Normal and Phage-Infected Cells as Influenced by Radiation.	S. Mudd.
Philadelphia General Hospital	The Effect of X-ray Radiation of the Lipids of the Skin.	H. P. Schwarz.
Pittsburgh, University of	Study of the Correlation of Radiation Effects with Physical and Chemical Changes in Viruses.	M. A. Lauffer.
Reed College	The Effect of Ionizing Radiation on Biochemical Compounds.	A. F. Scott, A. H. Livermore.
Sloan Kettering Institute for Cancer Research.	Biological Effects of Radiation, and Related Bio-Chemical and Physical Studies.	C. P. Rhoads.
Southern California, University of.	Effect of Radiation on Intestinal Absorption and Metabolism of Fats and Carbohydrates.	R. B. Alfin-Slater, A. L. S. Cheng.
Southern Illinois University	The Effects of X-rays and Ultraviolet Radiation on the Multiple Manifestations of a Gene Together with Genetical Analysis of the Radiation Induced Variations and the Effects of Extracts from Unirradiated Cells on the Repair of the Genotypes of Irradiated Cells.	C. C. Lindegren.
St. Louis University	Study of the Relation of Rickettsial and Viral Infections to Radiation Injury.	D. Greiff.
Tennessee, University of	A Survey of the Effects of Radiation on Animals Parasitized with *Taenia pisiformis*, on Parasites of the Irradiated Animals, and on the Parasites per se.	A. W. Jones.
Texas, University of	The Genetic and Biochemical Effects of Radiation on Bacteria.	O. Wyss.
Vanderbilt University	Study of the Absorption and Metabolism of Lipids and Vitamins, and the Alterations Which Occur in Acute Radiation Injury.	W. J. Darby.

5. Biochemical and microbiological studies of radiation effects, $1,472,000—Con.

Institution	Title	Investigator
Virginia, Medical College of	An Investigation of Certain Tissue Protein Changes in Irradiated Animals.	H. G. Kupfer, N. F. Young.
Virginia, University of	A Study of the Kinetics and Reactivity of Cell Surface Components as Affected by Ionizing Radiation.	H. Jonas.
Western Biological Laboratories.	Further Studies of an Unidentified Factor in Liver which Prolongs Survival of Animals Administered Multiple Sublethal Doses of X-Irradiation.	B. H. Ershoff.
Worcester Foundation for Experimental Biology.	Investigation of the Effects of Radiation on the Biosynthesis and Metabolism of Adrenocortical Steroids.	G. Pincus.
Argonne Cancer Research Hospital.	Effects of Ionizing Radiation in Protein Metabolism in the Human Being and Experimental Animals.	R. J. Hasterlik, E. I. Pentz.
Argonne National Laboratory	Iron Uptake by the Bone Marrow of Irradiated and Control Rats.	H. H. Vogel, J. W. Clark.
	Radiation Effects on Dry Bacterial Spores	E. L. Powers, C. F. Ehret.
	Mutagenic Effects of C-14	N. Williams, N. S. Scully.
	Biochemical Studies of Effects of X-Irradiation	E. K. Bernstein.
	Dependence on Radiation Dose Rate of Oxidation or Reduction of DPN/DPNH in Living Systems.	T. N. Tahmisian.
	Cytochemical Studies of Nucleic Acids in Irradiated Tissue Cultures.	A. M. Brues, A. N. Stroud.
	Radiation Effects on the Mating Reaction in *Paramecium bursaria*.	C. F. Ehret.
	Radiosensitivity of S-adenosylmethionine-forming System in Animal Tissues.	P. D. Klein.
	Biochemical Changes in Irradiated Animals	J. F. Thomson.
Brookhaven National Laboratory.	Effects of Radiation on Enzymes	L. Augenstine.
	Biochemical Effects of Radiation in Mammals	F. Sherman.
	Effects of Radiation on Large Molecules	D. Fluke.
California, University of, at Los Angeles (Atomic Energy project).	Radiation Metabolism	L. Bennett.
	Tissue Transplant—Effects of Irradiation	B. M. Allen.
	Metabolic Radiobiology	O. Schjeide.
California, University of, Radiation Laboratory.	Distribution and Metabolism of Trace Elements	C. Tobias.
Oak Ridge National Laboratory.	Effects of Radiation on Paramecium	R. Kimball.
	Biophysics	J. Kirby-Smith.
Rochester, University of (Atomic Energy project).	Effects of Radiation on the Biosynthesis of Hemoglobin.	K. Salomon.
	The Effect of Various Radiations on Bacterial Systems.	G. Whipple.

6. Environmental studies, $52,000

Institution	Title	Investigator
South Carolina, University of	An Ecological Study of the Fishes of the Savannah River Drainage.	H. W. Freeman.
	An Ecological Study of the Flora and Fauna of the Savannah River Plant Area.	W. E. Hoy.
Texas A. & M. Research Foundation.	A Study of Some Factors Involved in the Disposal of Radioactive Wastes at Sea.	R. G. Bader.
Washington, University of	Determination of Relationships Between Temperature Lapse Rate, Wind Speed, and Wind Shear (Atmospheric Turbulence Study).	F. I. Badgley.

7. Dosimetry research: The development of improved methods of measuring radiation, $1,026,000

Institution	Title	Investigator
U. S. Army Corps of Engineers	Program of Research and Development on Scintillation Crystals.	N. F. Blackburn.
Commerce, U. S. Department of, National Bureau of Standards.	Radiation Monitoring Systems	L. Costrell.
Commerce, U. S. Department of, National Bureau of Standards.	Radiation Shielding Problems	H. O. Wykoff.
Columbia University	Attenuation of Scattered Cobalt 60 Radiation in Lead and Building Materials.	C. B. Braestrup.
	Evaluation of Aerosol Filters and Impactors Having Chemically Different Surfaces.	V. K. LaMer.
Levinthal Electronic Products, Inc.	Study of Scintillation and Other Related Properties of NaI Crystals.	W. J. Van Sciver.
Argonne National Laboratory	Dosimetry of Mixed Radiations: Fission Neutrons and Gamma Rays.	H. H. Vogel, Jr., J. W. Clark.
	Tissue Dose Distribution of Soft X-rays	H. Walton, G. A. Sacher.
	Radiation Dosimetry	L. D. Marinelli.
California, University of, at Los Angeles (Atomic Energy Project).	Chemical Dosimeter Development	G. Taplin.
	Scintillation Counter Development for Medical Use.	B. Cassen.
General Electric Co., Hanford area.	Analytical, Bio-assay and Counting Methods	D. W. Pearce.
	Special Studies and Monitoring Methods	Do.
	Gamma and Beta Ray Dosimetry	Do.
	Neutron Dosimetry	Do.
Knolls Atomic Power Laboratory, Schenectady.	Investigation of Fast Neutron Effects on the Electrical Properties of Semiconductors in the Energy Range From About 0.5 to 18.0 Mev.	T. M. Snyder.
	Development of an Electron Microscope Capable of Resolving Individual Atoms.	Do.
Oak Ridge National Laboratory.	Radiation Dosimetry	G. S. Hurst.
Rochester, University of	Measurement of Radiation Dose to Lungs From Radon and Thoron Degradation Products.	W. Bale, A. Dahl.

Dr. Dunham. I call your attention to the first page of that in which it says that there are only four people working on environmental studies. The reason that happened is that in the hurry to get this material to you, we failed to take cognizance of the fact that most of the environmental studies now going on are lumped under the first group, sampling and analysis of radioactive fallout. So that this figure here looks very small, but that does not mean that we are not doing extensive studies in Nevada, and elsewhere, but they are listed in item 9.

I would like to at this point call upon Dr. Shields Warren, with your permission, to say a few words about research in this area.

Representative Holifield. I was going to ask Dr. Warren and Dr. Brues and Colonel Bach and Dr. Shelton if they wanted to comment on your statement.

Dr. Warren, will you lead off?

Representative Cole. May I interrupt for a moment to express my personal admiration for the appearances Dr. Dunham has made throughout these hearings and particularly this morning. I have been tremendously impressed with his grasp of the problem and his knowledge of the details of it. I congratulate you on it. I think the Commission is very fortunate in having a person of your capacity as Director of the Division of Biology and Medicine. The public is fortunate in having you as a public servant. I compliment you in the highest terms. I am very much impressed, and I am amazed that a person of your talents is willing to sacrifice your life to work for the Government for the pittance that you get.

Dr. DUNHAM. Thank you very much, Mr. Cole.

Representative VAN ZANDT. I have a question of Dr. Dunham. For the benefit of the record, can you describe briefly what happened to the animals that were on the Marianas in the 1954 test? There were 45 or 47 of them, as I recall. Are you familiar with the study on those animals?

Dr. DUNHAM. In the Marianas?

Representative VAN ZANDT. On the Marshalls.

Dr. DUNHAM. You mean the animals on Rongelap. Some of them were recovered almost immediately and taken to the Naval Radiological Defense Laboratory where some were sacrified immediately to find the body burden and some were kept for a period of months to discover the excretion rates and more recently a few more animals were found at the recent survey and similar studies have been done at the Radiological Defense Laboratory.

Representative VAN ZANDT. Is it possible for you to prepare a short statement giving the history of those animals and make it a part of the record?

Dr. DUNHAM. I would be happy to do so.

(The information referred to follows:)

UNITED STATES ATOMIC ENERGY COMMISSION
Washington, D. C., June 28, 1957.

Mr. JAMES T. RAMEY,
Executive Director of the Joint Committee on Atomic Energy,
Congress of the United States, Washington 25, D. C.

DEAR MR. RAMEY: Enclosed is a statement giving the history of the animals on Rongelap which Representative Van Zandt requested us to furnish for the record during the fallout hearings on June 7.

Sincerely yours,

CHARLES L. DUNHAM, M.D.,
Director, Division of Biology and Medicine.

Animals (swine, ducks, chickens) present on Rongelap Island were permitted to continue to live on the island and then were collected at time intervals up to about 2 months after the fallout had occurred. They were then sacrificed serially up to 6 months after the time of detonation. Under these conditions it is estimated that intake of the fallout material may be higher from consuming foods having external contamination than through the soil-plant-animal cycle. It would be expected that the eating habits of animals would result in greater intake of such material than for humans. Yet after living in this environment during the 2 months of heaviest contamination there were no significant gross changes nor pathological changes which could be ascribed definitely to radiation at the time of the examination (sixth month). A summary of the data is contained in the tables 1 and 2 attached, taken from Some Effects of Ionizing Radiation on Human Beings.

Analyses of a rooster and some rats that lived for 2 years in that area before collection and sacrifice, yielded similar negative pathological results. Table 3 summarizes these data. It will be noted from this table that the internal deposition of strontium 90, the isotope of greatest concern, is less than the maximum permissible concentration for adult atomic energy workers (1,000 sunshine units). Yet during this period of time the external radiation dose out-of-doors was at least 400 roentgens. These data definitely indicate that for times immediately following a detonation the external gamma exposure would be the dominant hazard.

A full account of these data may be found in Some Effects of Ionizing Radiation on Human Beings, and the forthcoming publication Radiological Contamination of Certain Areas in the Pacific Ocean From Nuclear Tests. Both of these documents can be obtained from the Superintendent of Documents, United States Government Printing Office, Washington, D. C.

TABLE 1.—*Mortality and external radiation dose of animals from the living areas of Rongelap and Utirik*

Animals	Series A			Series B			Series C			Series D			Total		
	200 roentgens (day 8) [1]			330 roentgens (day 25) [1]			340 roentgens (day 33) [1]			340 roentgens (day 51–53) [1]					
	Total received	Dead	Sacrificed	Total received	Dead	Sacrificed	Total received	Dead	Sacrificed	Total received	Dead	Sacrificed	Total received	Dead	Sacrificed
Hens	6	[2] 1	[2] 1	----	----	----	20	[3] 2	[4] 2	11	[5] 5	----	37	8	3
Roosters	1	----	----	----	----	----	2	[6] 1	----	1	----	----	4	1	----
Chicks	----	----	----	----	----	----	9	9	----	----	----	----	9	9	----
Ducks	----	----	----	----	----	----	4	----	[7] 1	----	----	----	4	----	1
Pigs	1	----	[8] 1	7	----	[9] 4	----	----	----	[10] 3	----	----	11	----	5
Cat	1	----	----	----	----	----	----	----	----	----	----	----	1	----	----
Total	----	----	----	----	----	----	----	----	----	----	----	----	66	18	9

[1] Day of collection past detonation and external dose.
[2] 23 days past detonation.
[3] 42 and 43 days past detonation.
[4] 44 days past detonation.
[5] Nos. 96, 39, 35, 7, and 24 were dead 67, 74, 92, 99, and 130 days past detonation respectively.
[6] 49 days past detonation.
[7] 56 days past detonation.
[8] 45 days past detonation.
[9] Sow and Nos. 6, 24, and 25 were dead 33, 57, 82, and 82 days past detonation respectively.
[10] Animals from Utirik; all others from Rongelap (group IV area animals received 22 roentgens external dose).

TABLE 2.—*Radiochemical analysis of tissues and urine of pigs from Rongelap on 82d day postdetonation*

BETA ACTIVITY—D/M/TOTAL SAMPLE

Sample	Gross activity $\times 10^{-3}$	Sr^{90} $\times 10^{-3}$	Ba^{140} $\times 10^{-3}$	Total rare earth $\times 10^{-3}$
Pig #24 (25.8 kgm):				
Skeleton (total)	8, 890	5, 660	660	1, 010
Liver	31	0. 40	0. 33	6. 4
Colon and contents	12	5. 0	2. 4	3. 2
Lung (alveolar)	1. 5	0. 22	0. 20	0. 8
Stomach	1. 2	0. 22	1. 1	1. 3
Intestine (small)	2. 3	0. 62	0. 50	0. 51
Kidney	3. 3	0. 21	0. 42	0. 74
Remaining tissues	690			
Total	9, 630	5, 667	665	1, 020
Urine sample, 24 hours	13	8. 7	1. 2	1. 6
Pig #25 (22.7 kgm):				
Skeleton (total)	8, 600	5, 100	530	690
Liver	27	0. 53	0. 20	5. 5
Colon and contents	16	5. 0	3. 2	4. 9
Lung (alveolar)	1. 1	0. 26	0. 23	0. 33
Stomach	2. 0	0. 29	0. 13	0. 30
Intestine (small)	2. 6	0. 83	0. 88	0. 88
Kidney	3. 1	0. 14	0. 19	0. 52
Remaining tissues	220			
Total	8, 870	5, 107	534	702
Urine sample, 24 hours	6. 2	4. 4	0. 40	0. 54

SUMMARY

Gross beta activity	Skeleton	Total body	Urine (24 Hrs)
Sr^{90}	62. 0	58. 0	69. 0
Ba^{140}	6. 8	6. 5	7. 9
Rare earth	9. 7	9. 0	10. 5
	78. 5	73. 5	87. 4

All values corrected for decay.

TABLE 3.—*Analysis of rats and a rooster collected on island of Rongelap*

	Wet weight	d/m Sr-90/ sample	February 1956 [1]	
			Ca/sample (gm)	Sunshine units
Rats: carcass [2]	44. 7	642±23	0. 533	545±19
Do	62. 5	315±62	. 315	453±90
Do	32. 3	367±21	. 353	470±27
Rooster:				
Femur	[3] 26. 0	1, 210±39	5. 19	105±3
Tibia	41. 0	5, 702±119	9. 50	272±5

	Sr-90	July 1956 [4]	
		Calcium	Sunshine units
Rats: Bone	245±5 d/m/g/wet	171±9 mg/g wet	644

[1] Naval Radiological Defense Laboratory.
[2] Does not include head, femurs, tibiae, and viscera.
[3] Dry weight of 2 femur halves.
[4] Applied Fisheries Laboratory, University of Washington.

Senator ANDERSON. I would like to have you see a letter that a Senator from Nevada received, and I will read it aloud. You tell me how you would feel if you were the Senator from Wyoming. I will leave the doctor's name out until I have his permission to insert it in the record. I guarantee it is an authentic signature of a radiologist at Reno, Nev.

The May 28 nuclear tests here in Neveda were of considerable concern to me as a doctor and a radiologist. The fallout as measured on my radiation survey meters was anywhere from 3 to 6 times above the acceptable tolerance level (that is 2 milliroentgens per hour or 50 milliroentgens per day) for the first 48-hour period. The local newspapers quoted reassuring phrases from the AEC to the effect that the above intensity of fallout was negligible and without danger and that the halflife of the radiation sources was calculated to be 12 hours.

This last statement is erroneous. One of the rules which the AEC enforces among users of radioactive isotopes is that residues of such isotopes be kept stored under appropriate protected conditions for 10 half lives, which is a period of decay, at the end of which the radioactive material is considered impotent. Yet, I find that after 10 half-life periods here in Reno there is still a 1.2 milliroentgen per hour radiation present. Actually, if the original information were accurate, at the end of the 10 half-life period it should be hardly detectable by my survey meters. This can mean one of two things: Either the estimated half life was in error or we have been revisited by fallout material dependent upon wind changes.

It is quite true that 50 milliroentgens per day (or 2 milliroentgens per hour) is considered to be of tolerance level. However, we must consider that all radiation received by this population is on an accumulative basis; and if we are to be periodically exposed to this much radiation, the accumulation can mount up to levels, which in the light of our present medical knowledge, becomes quite significant and hazardous. Actually, no one knows at exactly what dose level significant genetic changes and malignant tumors may suddenly appear in the human. So, it behooves us to practice utmost caution at all times in this regard.

Politics, physics, and medicine seem to be primarily concerned with nuclear tests and observation. But, from where I sit, only the medical man seems most keenly concerned with the future possibilities of these radiation dangers. So, I wish to add my protest to the situation which exists here in Nevada. I want more assurance that there is justification for continued testing, and I wonder if testing grounds could not be removed to more remote, uninhabited areas of the world.

I am not going to worry about your comments on the latter two questions, but what about these 10 half lives and what his testing devices show?

Dr. DUNHAM. I don't know what testing devices he has used. Dr. Dunning, a biophysicist in my division, who spends a good deal of time at the tests working closely with the radiation safety group and the Public Health Service there, has handed me a note to the effect that a dose of 50 to 100 millirep is now estimated for anybody who would continue to live in Reno for the rest of his life.

Senator ANDERSON. That is the estimate. What does that do to you?

Dr. DUNHAM. I think there has been a great deal of testimony during the past 2 weeks on the problem of whether there is a threshold at very low dose rates. This certainly is a low dose.

Senator ANDERSON. Geneticists all seem fairly well agreed that there was no threshold.

Dr. DUNHAM. That is correct. I don't think there has been any question of the geneticists' concept that the genetic effects are all cumulative, and at any level, high or low.

Senator ANDERSON. Would the Senator who got this letter be justified in answering the doctor and say you just have to grin and bear it?

Dr. DUNHAM. I would like to put this somewhat into perspective. If this doctor were to fluoroscope a patient suspected of having an ulcer of the stomach or a cancer of the bowel, the dose to the gonads—here we are talking about genetic effects—would be somewhere in the vicinity of 20 to 30 or possibly more roentgens, not 50 to 100 milliroentgens.

(NOTE.—These figures, 20 to 30 roentgens, may be high. Pullman and Laughlin, in their preliminary edition of Section III: Gonadal Dose Produced by the Medical Use of X-rays of the Report of the Genetics Committee of the National Academy of Sciences Study of the Biological Effects of Atomic Radiation, give an estimate of the gonadal dose from a study of the colon as from 4 to 15. The gonadal dose from a study limited to the stomach only would be less than 1 roentgen. This presupposes modern X-ray and fluoroscopy equipment and the taking of adequate precautions.)

Senator ANDERSON. As Dr. Selove pointed out in that instance the doctor would weigh the possibilities for good along with the possibilities for damage. In this particular instance there is no possibility for good. As far as the individual is concerned, he just gets damaged.

Dr. DUNHAM. That is correct to the extent that he would be damaged. I think that is one of the purposes of these hearings, that is, to point out and give the committee information on what factors have to be weighed when we talk about testing nuclear devices. Is it worth it to the country as a whole.

Representative HOLIFIELD. That milliroentgen is a thousandth of a roentgen?

Dr. DUNHAM. Yes.

Representative HOLIFIELD. How would you express a millionth?

Dr. DUNHAM. A millionth would be a millimilliroentgen.

Representative HOLIFIELD. You do not get that low in measuring.

Dr. DUNHAM. I don't think one can measure it satisfactorily that low that way and one has to begin to talk about counts per minute.

Representative HOLIFIELD. Thousandths is about the lowest?

Dr. DUNHAM. That is correct. Certainly film badges don't get that low.

Representative COLE. If you did get that low, what name would you give it?

Dr. DUNHAM. A microrentgen.

Representative HOLIFIELD. That is the same as a microcurie?

Dr. WARREN. Yes.

Representative HOLIFIELD. Dr. Warren.

STATEMENT OF DR. SHIELDS WARREN, NEW ENGLAND DEACONESS HOSPITAL, BOSTON, MASS.

Dr. WARREN. Thank you very much, Mr. Chairman.

Representative HOLIFIELD. Dr. Brues, would you like to come forward, and Lieutenant Colonel Bach and Dr. Shelton, if you wish to have anything to say, will you please come forward? Is Dr. Schulert here?

Dr. SCHULERT. Yes, sir.

Representative HOLIFIELD. Dr. Crow's name is here, too. I do not know whether he is still with us or not. I understand that Dr. Shannon and Dr. Burney have separate statements, so we will not ask them to come forward at this time.

You may proceed, Dr. Warren.

Dr. WARREN. Thank you. I appreciate the kindness of the committee in permitting me to make a statement relative to the present research program of the Atomic Energy Commission in this field of radiation, and to make comments as to the program and its possible future trends.

First, I would like to restrict myself to the fields of biology and medicine, as I am not competent to venture beyond them. I have some hesitancy in making recommendations as a medical man even in the field of biology. However, I would like to say that the program of the Division of Biology and Medicine of the AEC, which you have just heard outlined so clearly by Dr. Dunham, has within the limits of budgetary approval and personnel, attained as much in the way of research results as could be expected from any program.

Initially in this program there was a very real limiting factor, the scarcity of competent scientists. Scientists are still scarce, but are being trained rapidly. One of the best ways to attract scientists is to have adequate opportunities for them to work in any field. As I said, Dr. Dunham has outlined for you the present program with regard to radioactive fallout and certain suggested additions to it. I heartily second his suggestions, but would go somewhat further since he has restricted his comments largely to the factors that are immediately referrable to radioactive fallout.

Much of our knowledge with regard to the effects of radioactive fallout must be obtained by advancing our knowledge of other types of radiation, since fallout occurs in such very small quantities as to produce recognizable changes only with the greatest rarity. Therefore, there must be much research done in the general field of pathological effects of radiation on the one hand, and the effects of radiation on heredity on the other.

To be more specific, I would recommend expansion of research on the effects of radiation on the cell, both in its resting and its dividing state, and expansion of the study of the effects of radiation on enzyme structure. Additional studies are desirable on the effect on both acute and chronic radiation on animals, both as to specific changes and as to more subtle changes, such as shortening of the life span.

Studies of human population genetics, a relatively new science, should be greatly expanded, and the present studies in mammalian genetics should be multiplied several times. These studies, together with those recommended by Dr. Dunham and others, should not be at the expense of the present research program of the Division of Biology and Medicine of AEC since those existing studies, I believe, are well chosen and significant. Rather, the research budget of the Division should be materially increased. I think this should be done in an orderly fashion. A sudden increase is not a practical thing to undertake.

I would like to emphasize in regard to all of the research done in the universities—indeed practically all research in biology and medicine in this field—that there has been no restriction, no withholding of data. All of us working in this field have been free to publish our results exactly as they have occurred. There is absolutely no effort on the part of AEC to withhold significant information.

I think it might be worthwhile to comment briefly on the very careful studies on the fallout problem that are made both in this country and abroad. As you have heard, the groups that are following this

field of work are several. In this country, of course, the Atomic Energy Commission is the first and most active. The Department of Defense plays a large role. The Federal Civil Defense Administration is keenly interested. The Public Health Service is carrying on a number of very valuable studies. These agents all cooperate with one another and with the Weather Bureau, the Department of Agriculture, and other governmental groups, as you have heard, that have special interests in the field.

I think it is pertinent also that there are in this country several unofficial but nonetheless very well constituted groups that are following this problem of fallout and research reated to it. The most expert are (1) the National Committee on Radiation Protection which in turn is affiliated with the International Committee on Radiation Protection, and the International Committee on Radiation Units. You heard from Dr. Taylor, its chairman, as to the way its work is carried on and what it does.

(2) The Committee on Radiation Effects of the National Academy of Sciences, which, as you know, has made valuable reports and are continuing to follow the problem.

Representative COLE. Mr. Chairman, I wonder if it might not be appropriate—I intended to ask Dr. Warren about it later—at this time to indicate the distinction between the United Nations Scientific Committee on Radiation, the International Committee on Radiation Protection, and the International Committee on Radiological Units There is coming to be an abundance of international committees in the field of radiology, and I would like you to explain the origin, the genesis of each, how they overlap and so forth.

Dr. WARREN. Yes. In the mid-1920's the people working in the field of radiology and radiobiology became concerned over the problems of radiation protection and the fact that many of the people who were going into radiology were being damaged by the radiation they received, and were not being taught how to protect themselves.

Consequently, in several countries National Committees on Radiation Protection were established, first informally. Those in this country were made up of representatives of scientific societies which cooperated with the National Bureau of Standards and at present the activities of the National Committee on Radiation Protection are housed at and aided by the personnel of the National Bureau of Standards.

The chairman of these various national committees joined informally in an international group. This international group first met every 3 years at the International Congress of Radiology. As the problems became more multiple they have met more frequently.

Representative COLE. You say it began in 1920?

Dr. WARREN. That began, I think, in formal fashion in either the late 1920's or the early 1930's. Its last meeting was the end of April in Switzerland. Dr. Taylor, from whom you heard, together with Dr. Wycoff of the Bureau of Standards, were the United States representatives there.

This is a purely informal group which has had such efficiency in its work, such scientific standing, that the AEC and practically all of the governmental and State agencies accept its ruling as official, and through the recording of these in the Federal Register they have the force of law.

These committees are entirely apart from the United Nations committee. If I could reserve comment on that for just a few minutes while I speak of some of the other agencies, I would like to come to that in more detail later.

Among other competent groups working in this country are many research groups in universities, in research laboratories throughout the country; the Federation of Atomic Scientists, who had a representative here, is also keenly interested. Then there are a number of other groups that have committees having to do with atomic energy, and concerned with problems of protection. Even the United States Chamber of Commerce and the AFL–CIO are interested in this field.

Abroad there is much emphasis on the problems of fallout and the official report of the British Medical Research Council contains much useful data which, as you have heard, correlates closely with those of our own National Academy of Sciences.

The World Health Organization has also considered certain aspects of the radiation problem.

At the instance of the United States, the General Assembly of the United Nations has appointed a scientific committee to study radiation problems. This committee consists of representatives of 15 nations. I am the representative for the United States. Dr. Brues, sitting beside me, is one of my alternates and has been extremely helpful. This committee has been studying the problem of fallout and related aspects of radioactivity most intensively, and its report will be presented to the General Assembly in June of 1956.

Representative COLE. May I inquire, Dr. Warren, if the meeting of this committee last fall in New York was the first of its meetings?

Dr. WARREN. No, that was the second of its meetings, Mr. Cole. There was one in Switzerland in April. The next, I believe, is scheduled for New York in January. However, there is a great deal of work done besides that being done at these various meetings. There is a secretariat which is continuing to digest all of the information that has been turned in. All of us receive information from all over the world. At the present time, I am reading through a stack of reports, from virtually every country in the world, that is close to 3 feet high. I have gotten two-thirds of the way through it now. Various members of this United Nations Committee have been given the responsibility with the secretariat of preparing separate chapters of this report which will probabily be about a 200-page report when it gets done.

Representative COLE. How long has the committee been in existence? Has it been about a year?

Dr. WARREN. As I remember it, it came into existence about a year and a half ago. Do you remember, Dr. Brues?

Dr. BRUES. November before last, I believe.

Dr. WARREN. Yes. It will make its report, as I say, for June 1958.

CHAIRMAN DURHAM. Why is it just 15 nations, Doctor?

Dr. WARREN. It was felt that a group of that size would be a group that would represent a high degree of scientific competence from the different countries. It would have available to it information from all over the world and all of the member nations of the United Nations and its allied organizations. It would be a small enough group to

work effectively. If it had been a group representing every one of the members of the United Nations——

Chairman DURHAM. Can you give us the names of the 15 countries?

Dr. WARREN. I believe I can. Australia, Argentina, Belgium, Brazil, Czechoslovakia, Egypt, France, India, Japan, Mexico, Sweden, U. S. S. R., United Kingdom, United States, and Canada.

Chairman DURHAM. Russia is a member of the group?

Dr. WARREN. Both Russia and Czechoslovakia are members of the group.

Representative HOLIFIELD. How many meetings has this committee had?

Dr. WARREN. This has had three formal meetings. The reason I was hesitant is that we have had so many informal meetings that it is very difficult to keep track of them. You might say that those of us working in the group are almost in continual session through contact with the secretariat by mail and so on.

Representative HOLIFIELD. Do you have an adequate secretariat to collate the material that is sent in and is there a wide collation and distribution?

Dr. WARREN. Yes, there is a very wide collation and distribution. The great bulk of the material has come from the United States, because we have accomplished more research than any other of the nations.

Chairman DURHAM. How do you report, Doctor? Do you report directly to the American representative?

Dr. WARREN. As the representative for the United States, I report to Mr. Lodge as the Ambassador to the United Nations. However, our committee as a whole is a creature of the General Assembly and reports to the General Assembly.

Chairman DURHAM. It reports directly to the General Assembly?

Senator ANDERSON. How does this information get to the people of this country?

Dr. WARREN. It is the responsibility of the various representatives to bring all pertinent information which is made available to them—this is all circulated—to the people of their country. The final report of the committee will be an open report to the General Assembly and through them to all the member nations and distributed throughout the world.

Senator ANDERSON. Can you give us the name of the fallout expert in Argentina?

Dr. WARREN. The head of the delegation is Captain Nuñez.

Senator ANDERSON. Is he a radiologist?

Dr. WARREN. He has a background in radiology and radiobiology. I might say that the different countries have chosen people with specific competences which vary with the countries. But each delegation from each country brings all the available information from that country. It is not restricted to the specific competence of the members of the delegation.

Senator ANDERSON. What sort of individual represents Egypt, for example?

Dr. WARREN. In Australia a physicist is the representative. In Belgium a medical man interested in radiobiology is a representative. In Canada a medical man who is a public health official represents the country. In Brazil, a distinguished biophysicist. In Czecho-

slovakia, also a biophysicist. I could go through the list. It is a very varied group.

Senator ANDERSON. This individual from Canada, has he been making any special studies of fallout?

Dr. WARREN. Yes.

Senator ANDERSON. Is that his field?

Dr. WARREN. He is relatively new to this field, but he was chosen by the Canadian Government because they felt him to be thoroughly competent in it.

Senator ANDERSON. Has he been doing work and making studies in fallout? Is that his specialty?

Dr. WARREN. I would say he is essentially an all around public health man who is especially interested in this field and is provided by his Government and the scientists in his country with a great deal of information in this field.

Representative COLE. You spoke of the report of this committee as being a final report. Does that mean that when the report is submitted in June of next year, that the committee will disband and discontinue its work?

Dr. WARREN. We have been appointed by the General Assembly to review the problem, to gather, collate and evaluate information, and to make a report. Our future is entirely in the hands of the General Assembly. I rather assume that they will ask us or successors to us to continue this work. I cannot speak for the General Assembly in any way, of course.

Representative HOLIFIELD. You may proceed.

Dr. WARREN. I would like to leave very briefly the discussion of research and make one further comment. That is this: The ultimate decisions with regard to weapons testing and with regard to the whole development of atomic energy will have to be made, as they have been made in the past, by you and other duly constituted representatives of our people. I believe that the advances in science within the next few years provided research is adequately supported and facilitated will permit obtaining much more conclusive data than now exist as to the feasibility of continued weapons testing. The concern of the world is for disarmament and the elimination of war, of course. I firmly believe as a physician that it is inexcusable for us to jeopardize our own safety and that of the rest of the free world in order to eliminate a risk of as low an order of magnitude as is constituted by any reasonable program of atomic weapons testing.

Senator ANDERSON. Do you think that the proposal made by Dr. Langham which was an overall control of the total tonnage of any fission products going into the atmosphere would jeopardize our production of weapons.

Dr. WARREN. I am not at all an expert in this field, Mr. Anderson. I would not have any opinion. I would hope that it might be feasible to work out some program of this type.

Senator ANDERSON. Would you read again the last paragraph.

> I firmly believe as a physician that it is inexcusable for us to jeojardize our own safety and that of the rest of the free world in order to eliminate a risk of as low an order of magniture as is constituted by any reasonable program of atomic weapons testing.

You there set yourself up as an expert in the field. I am not trying to say it is improper. You testified what you would do.

Dr. WARREN. Yes.

Senator ANDERSON. Having established that, do you thing Dr. Langham's proposal that an amount of 10 megatons of fission production going into the atmosphere each year which we are now doing is about the safe limit?

Dr. WARREN. I feel we would be safe in having that much. I would hesitate to say that is an absolute upper limit. I would think that is a reasonable amount. I would not be worried by a program at that level.

Senator ANDERSON. If you have not made studies in the field yourself, you recognize that the Los Alamos and the Livermore Laboratories have.

Dr. WARREN. Yes. They are most competent.

Senator ANDERSON. If they feel that is a top limit, does that suggest to you that is something we might look to as a proper guide or not?

Dr. WARREN. I would think that this might be very sound indeed. From my own knowledge from the medical standpoint, as I said, I would not be at all worried by a program at this level.

Senator ANDERSON. Almost every time when somebody comments, they talk about limitation on testing as if it meant the elimination of all progress and all testing of every kind. It is like saying to man he should be careful in the amount of protein he takes into his system. But a doctor will say if you do not take any protein at all, many things will happen to you. Somehow we do not get much comment on the suggestion of limitation. It is always that we will abolish it all. This was not the proposal of Los Alamos and certainly has not been my own.

Senator JACKSON. Dr. Warren, what this really boils down to is that we have two risks. One is the risk to the free world if we are not prepared to deal with an enemy that might well bring total atomic hydrogen catastrophe to all free nations. On the other hand, continued testing do present a danger of an undetermined nature to people. We do not have enough scientific data for scientists to speak scientifically, whether they are doctors or pure scientists. There are these two threats. Maybe between the two some kind of reasonable balance can be achieved. Don't you think that is a reasonable approach?

Dr. WARREN. Yes. I think that is a very reasonable approach. That is what I had in mind when I spoke of any reasonable program of atomic weapons testing.

Senator JACKSON. What was your last statement?

Dr. WARREN. I simply said that was the general idea I had in mind when I said "any reasonable program of atomic weapons testing."

Represenative HOLIFIELD. If there are no questions, I will ask Dr. Brues to proceed.

Chairman DURHAM. Before you proceed, Dr. Warren, on the second page of your statement I believe your statement concurs with the statement Dr. Libby made on yesterday in his recommendation of increasing the research program of the Division of Biology and Medicine.

Dr. WARREN. Yes, sir.

Chairman DURHAM. You say "not at the expense of the present research program," but still you recommend an increase in it. Could

you put that on a percentage basis as to exactly what you mean by that type of statement?

Dr. WARREN. What I would think is this, sir. One is that there are a number of other very important problems that are being worked on by the Division of Biology and Medicine than those having to do with fallout. We should not interfere with those. There should be an expansion of the program which I think could perhaps be on the order of—it is hard to say—of an expansion that went up, say, 50 percent in 3 years. Maybe that much again in another 3 years would be 1 that could be handled in very orderly fashion. It would not be wasteful. There would be adequate scientific personnel to see it through.

Chairman DURHAM. In some sections of the whole program, you would place more emphasis on this?

Dr. WARREN. Yes. I think, for example, that we must not lose emphasis on the portions of the program that have to do with how to cure atomic innjuries once they have occurred. We must not lose emphasis on the good things that can be done in the field of medicine and biology with atomic energy. I think it is important that in the development of this whole atomic-energy field that the developments in biology and medicine be advanced on a broad foundation. As an illustration, for example, we began our studies on radioactive strontium long ago before we knew how important radioactive strontium could be. I am sure there are other things in the program that do not look very important at the present time that we will be very glad in 1965 we had started earlier.

Chairman DURHAM. You mean that many new things are showing up on a daily or monthly basis that funnel off some of your research in that direction.

Dr. WARREN. That is right.

Chairman DURHAM. You do feel that the impetus should be on an increased expansion.

Dr. WARREN. I feel this very strongly, sir.

Representative HOLIFIELD. Dr. Brues, did you have any comment to make on the testimony which has been given?

STATEMENT OF DR. AUSTIN BRUES, ARGONNE NATIONAL LABORATORY

Dr. BRUES. Mr. Chairman, I have not prepared a statement, but there might be 2 or 3 things I might say, particularly with reference to the programs as they are carried out at the national laboratories which constitute a considerable portion of the work.

As Dr. Dunham mentioned, the national laboratories are run by contract with various agencies. Ours is operated by the University of Chicago. Yet they are large enough to operate essentially as independent institutions, and they are financed by the Atomic Energy Commission. The fact that there are several national laboratories was established in the original Atomic Energy Act, and in a sense grew out of the fact that there had been several sites at which research was carried out before the Commission came into being. I have felt personally that the diversification of the large research centers into several rather than bringing them together has been a good thing from a number of standpoints. I would say particularly be-

cause it allows a certain flexibility. There is no embarrassment, for instance, if an investigator in one laboratory holds a different view from an investigator in another laboratory; just as in the universities they get together and these things are reconciled by further work.

The programs are defined very much by the individuals who carry out the programs. The amount of masterminding, I think, has been kept at a reasonable and proper minimum. What people in the laboratories are mostly interested in is knowledge of what are the fields which are not being adequately tackled, and what, therefore, may be needed in the way of both basic and practical research. In that sense, of course, it is very useful to keep in contact with one another and with the authorities in the Commission.

The selection of personnel is done entirely at the level of the laboratories themselves, with only the restriction, which actually occurs very rarely, that is placed upon us by the security provisions of the act.

Chairman DURHAM. I agree with you, Doctor. I think the program has been well planned on the basis of the distribution in the universities of the entire country. I think if we built one huge laboratory we would not be as far along as we are today.

Dr. BRUES. The type of work is divided in our national laboratories and I think in most of them, and rather equally, between work which is straight programatic investigation kind of work where we are collecting numbers of figures, work which has more or less immediate practical objective, such as looking for treatments of radiation illness and so forth, and work which some people might consider useless, but as Dr. Warren mentioned turns out very often to be quite useful.

We have on occasion been asked if we could not perhaps think it would be important to gain some information in a certain area. Often, actually this information has been at that time fairly well gained by investigators who perhaps through an intuitive process or their curiosity have been working on it. This certainly was true of strontium. We have dogs in the laboratory which received some of the earliest radio strontium 10 years ago. This is not part of the program that would have been given us at the time, but obviously animals of this sort are very important, at least in determining the specifications for other experiments which may be started at this time.

Senator ANDERSON. When Dr. Dunham presented his paper, he said on strontium toxicity:

> In 10 to 15 years we will have experimental data in dogs which will firm up the maximum permissible body burden of strontium 90 for all ages, and will have determined whether strontium 90 is ever leukemogenic in dogs and presumably man, and will have gone a long way to settling whether or not the bone-tumor effects of strontium 90 have a threshold.

Are we to understand that to mean that in 15 years or so we will find out whether it is dangerous to let strontium 90 to be lying around, and if so, who much of it we can have?

Dr. BRUES. I think we are sharpening our knowledge on that all the time. I think it will take another generation of long-lived animals probably before we have the sharpest figure that we can reasonably ever expect to get.

Senator ANDERSON. But by that time if we can believe the upward sweep of testing, with other countries getting into the act, and everybody coming along with their own weapon testing, we could possibly

have done heavy damage to ourselves, could we not? Is there any way of speeding this up? We build a bomb by a process of speeding up. We have had estimates of how long it would take to develop a bomb.

Dr. BRUES. I think, sir, that the way in which this may be and probably will be speeded up is by the expansion not only of these predictable programs, but of the type of work which is going on in what I term the useless areas. In other words, investigation of how cancer is produced by radiation, and indeed how cancer is produced by other agents in comparison with this. I think that we may therefore arrive at basic knowledge in this way which will resolve some of the questions that we are also asking in case that should not pay off in the area of carrying out practical experiments.

I might say these animals which we treated 10 years ago, which have never shown signs of damage, received about 700,000 sunshine units of strontium. The only trouble with this is that there were only three of these dogs. As you have heard, you have to have large statistical numbers in order to sharpen things up.

Senator Anderson. Do you think it will be 10 to 15 years before we know whether testing at the present levels were continued would present any perils to people from strontium 90? I am confining it strictly to strontium 90.

Dr. BRUES. To answer that I would have to go into some questions that were discussed yesterday.

Senator ANDERSON. I do not mean to prolong this.

Dr. BRUES. I believe that it is very likely that we will get at some very fundamental truths about cancer sooner than that period which will tell us this, but we must at the same time be carrying on experiments which will give us the figures, so to speak.

Senator ANDERSON. I say that because the third paragraph of Dr. Dunham's paper says that it will be 10 or 15 years before we get sufficient data, and the fourth paragraph said that the true doubling dose for the mutation rate would take 8 years, if ever, and everything suggests an awfully long time. A tolerable mutation rate would take many years and never be exact. It is a very discouraging picture. The probabilities are that we will never know anything about anything. Is that your feeling?

Dr. BRUES. I would feel that perhaps I am entitled to be more optimistic than Dr. Dunham, because it would not be so easy to come back at me and chastise me for making a prediction that was not sufficiently conservative. I am more optimistic than is indicated there.

Senator ANDERSON. I was not trying to pick a quarrel with Dr. Dunham on this. I was hoping there would be a little more optimism than this "if ever, and we will never be exact."

Representative HOLIFIELD. We are looking for more sunshine.

Senator JACKSON. I was going to comment, is there such a thing as an exact science?

Senator ANDERSON. We hope we get some answers that we regard as pretty specific.

Representative HOLIFIELD. Would there be any other comments? Did you have something else, Dr. Brues?

Dr. BRUES. I would like to say one other thing very briefly, sir, and that is to underline Dr. Warren's remark that there has not been any

interference in my experience or the experience of anyone I know with the dissemination of results, no matter what they showed. I have actually some documentation on that. There was a statement made by one of the witnesses the other day that indicated that the principle of life shortening had been kept under cover for some reason not known to the speaker. I will, if you wish, produce some documentation that this was not only not kept under cover, but that these things were given to the public in a publication which came out of the first national radiobiological meeting which was attended by many of those that were present here, and in fact made the scientific public aware of all of the work which had been done in the field of life shortening, raised the questions of cell mutations, and many other things of that sort.

Representative HOLIFIELD. You will concede, however, that speeches before limited number of scientists does not take the place of information prepared which is readily understood by the public and widely disseminated. I am blaming no one for this, but there is a difference in the expression of opinion to a limited group of scientists and actually getting the story to the people.

Dr. BRUES. That is true, of course. That is the general problem which we also have, that of disseminating the knowledge. I think that this meeting served the important purpose of getting many people working in this field. It was quite speculative at the time, but it has resulted in a wide interest. For example, the brilliant work Dr. Hardin Jones told you about the other day was assisted by the fact that this subject was discussed at that meeting which was aided by the Commission.

Representative HOLIFIELD. At this time the Chair is going to interrupt the proceedings. There is a rollcall which the House members have to attend. We will proceed at 2 o'clock this afternoon in this room as we have some important statements which have to be made. We will resume with this part as soon as we return.

Senator ANDERSON. May I ask one question of Dr. Warren. I started out on strontium 90 a minute ago. When you were head of the AEC's Division of Biology and Medicine, was there a proposal put up to you for a study of the chronic effects of strontium 90, and was there some decision taken on it on the basis that it would be a ten-year and very costly study? Do you recall?

Dr. WARREN. My memory is—and it is a little vague—that there was a proposal made of a 10-year study using dogs that would have cost about $5 million a year, as I remember. It is something of that sort. I believe that strontium 90 was included along with radium, mesothorium, and other substances.

(A letter clarifying Dr. Warren's testimony follows:)

CANCER RESEARCH INSTITUTE,
NEW ENGLAND DEACONESS HOSPITAL,
Boston, Mass., August 6, 1957.

Mr. HAL HOLLISTER,
Joint Committee on Atomic Energy,
Capitol Building, United States Senate Post Office,
Washington 25, D. C.

DEAR MR. HOLLISTER: On July 5, 1957, as I was leaving on vacation, I sent a suggested addition to my testimony on the final day of the hearings. In reading this over I am not sure that it entirely answers the question raised by Senator Anderson. If it is not too late to make a change in the proof, the following might be a better way of expressing it.

"Another project that might be thought of in this connection was one made by the Los Alamos Laboratory, as I recall it, but the proposal for which was never formalized. This project had to do with the effects of the inhalation of particulate radioactive substances by dogs. After a good deal of preliminary study it was felt that certain phases of the proposal were very useful and significant. Since suitable inhalation chambers were already available at the atomic energy project at the University of Rochester and only additional animal quarters were required, this experiment was undertaken at the University of Rochester on the completion of the necessary changes in animal quarters."

Sincerely yours,

SHIELDS WARREN.

Senator ANDERSON. Did you not recommend pretty firmly against it?

Dr. WARREN. I recommended against that particular study but I recommended in favor of continuing studies in this field and got together a group of all the competent and interested people to establish at the University of Utah a study on dogs which started, as I remember it—I will have to ask Dr. Dunham as to the year—was approved and started in 1950, Dr. Dunham tells me. There had to be some construction which slowed it down for some months. That study is the one to which reference has been made now, and useful data are coming from this.

I think that one has to keep in mind this point, Mr. Anderson. One always has to weigh the types of research undertaken in relation to the funds and personnel available for that research at the time. When this research was proposed in its original form, it seemed impractical and too costly for the data that hopefully would be obtained. It looked as though this University of Utah study would be a sounder way of approaching the problem, and one within our means, both of available scientists and available funds. I still hold that opinion.

Senator ANDERSON. I do not question you on the available scientists at all, but I do believe that on funds for items of this nature since I have been a member of the Joint Committee, I have had to go to the Appropriations Committee many times and urge them to be somewhat liberal with the Atomic Energy, and I have always found them so, and I only hope that we do get some sort of expanded program in research that may give us a little quicker answers on some of these questions that are very disturbing to a great many people.

Dr. WARREN. I strongly agree with you, Mr. Anderson.

Senator ANDERSON. We will meet at 2 o'clock.

(Thereupon, at 12:15 p. m., a recess was taken until 2 p. m., the same day.)

AFTERNOON SESSION

Representative HOLIFIELD. The committee will be in order. I want to announce that the Chair will have to leave the room at 5 minutes to 3, and, for fear that I may not get back before the witnesses are concluded, I would like at this time, on behalf of the committee, to thank all the witnesses who have appeared during this group of hearings.

The purpose of the committee, as stated in my opening statement, has been adhered to. It was in a limited field. There are other fields

that are of interest to the committee, and undoubtedly the committee will want to go into some of those fields laer. There has also been a great deal of information filed, either in oral form or in written form, with the committee which the members have not had a chance to thoroughly study, nor has the staff had a chance to analyze. So it has been thought best by the subcommittee that we adjourn our meetings today for the purpose of studying these documents and receiving information which has been requested and has not yet been given to the committee. The committee will make a later determination as to whether there will be additional hearings to fill any gaps that we may find in our testimony on this particular specific subject, and also make the determination of going further into fields which are indirectly related to the subject of weapon fallout in the field of radiation. I speak of the fields of industrial hazards, which will encompass large-size reactors, and also the fields of weapon detection, which has a great deal to do with our ability to know when these explosions occur, and, also, our ability to evaluate accurately the amount of fission products which are thrown into the air. Of course, this has tremendous importance in evaluating the overall burden of radiation which would be finally deposited on the earth and its people.

We also have not had time to analyze the weapons effects handbook which is now in print by the military liaison group of the Defense Department and the Atomic Energy Commission, and we may want to go into some of the matters contained in there.

Another very important area in which the committee is interested—which may, in view of certain industrial developments, have to be considered—is the problem of waste disposal from reactors, the methods that are now used to dispose of this waste material, and the safeguards that are in existence at this time and plans for the future in order to protect the people from radiation from waste materials.

We will want to know the quantities, in tons or gallons, of this material which are now in existence, and which are being produced every day and every year, and we will want to have an estimate of the problem that will be involved in the disposal of waste when we have a hundred of these large-size reactors of 100,000 to 200,000, or 300,000 electrical kilowatts of capacity.

We will want to know how the nations abroad are disposing of this waste; whether they are placing them in the ocean, where the contamination might become global; and where the United States is disposing and the methods it uses.

So there are fields which have not been covered, but they are fields which the comittee will get to in the future, no doubt.

The committee reserves the right, of course, to accept and incorporate in the record additional scientific material which has been requested by the members or referred to by witnesses. We also, of course, reserve the right, as I mentioned at the beginning of our hearings, to include answers to specific letters of inquiry to scientists and witnesses which have been sent out and will be sent out for the purpose of clarifying or completing certain areas of inquiry.

Representative COLE. Mr. Chairman, before you do that, since you have indicated that in all likelihood you will not be here at the con-

clusion of the hearings this afternoon, I feel I should now express what I otherwise would have withheld until that time, and that is to join you in the compliment to the witnesses who appeared, and, more particularly, to pay a compliment to yourself for the fine manner in which these hearings have been conducted and the tenor that has attached to the hearings, for which much credit is due yourself. I compliment you and Mr. Hollister, as well, who has prepared this agenda, for the total accomplishments of these hearings which, I am confident, in the course of time will prove to be most valuable.

Representative HOLIFIELD. I thank the gentleman from New York very much.

Representative VAN ZANDT. Mr. Chairman, I would like to join my colleague from New York in that statement.

Representative HOLIFIELD. I am sure, without the help of every member of the committee and the hard work of the staff night and day, both before and during the hearings, we could not have had as successful a conclusion as I think we are going to have in these hearings.

Senator JACKSON. Mr. Chairman, I, too, would like to associate myself with the remarks that have been made with reference to these hearings. I think the chairman and Mr. Hollister and the staff have done a fine job.

I believe that these hearings cannot help but have a favorable impact in the long run on the American people and world opinion. I think the most important thing of all is that we have had the opportunity of having different points of view. Certainly, in large part, the views that have been expressed have been honest scientific opinion, even though the scientists and experts in the field have not always agreed.

Representative HOLIFIELD. Thank you very much. It would be remiss on my part if I did not thank all the witnesses who have appeared before us, and especially do I wish to thank the witnesses from the AEC, and from projects which are under the auspices of the AEC, for their cooperation in preparing their statements and presenting them to us in the complete way in which they have.

Dr. DUNHAM. Mr. Chairman, is it in order that I make a statement on behalf of the AEC, which has appeared before you in several forms, to thank you for the extremely courteous and interested attitude of the committee. I want to assure you that any further information you may require we will endeavor to provide for you.

Representative HOLIFIELD. Thank you very much.

By mutual agreement we are going to reverse the order of the witnesses who have prepared statements, and we will ask Dr. Waterman, Dr. Alexander, Dr. Bach, and Dr. Shelton, and any other scientists who may be present and wish to join, to comment on their statements at their conclusion. I think it may be an orderly way to take the three statements which we have, which I understand are not too long, and have them in order, and then have the roundtable discussion.

At this time we would like to have Dr. Schulert of Lamont Laboratory to be our first witness.

STATEMENT OF DR. ARTHUR R. SCHULERT, LAMONT GEOLOGICAL OBSERVATORY, COLUMBIA UNIVERSITY [1]

Dr. SCHULERT. I have a statement which I prepared for the record. However, I do not believe it is necessary to read the statement in its entirety, since it would involve the repetition of many things that have transpired this morning. The statement involves my personal opinion as to what we should do along the research line in view of the present state of affairs. This, as you know, was quite comprehensively reviewed by Dr. Dunham of the AEC this morning.

I would like to just mention a few of the highlights of this statement.

(The statement referred to follows:)

REMARKS PREPARED FOR PRESENTATION BEFORE THE JOINT COMMITTEE ON ATOMIC ENERGY, UNDER SECTION XII—THE IMPACT OF THE PRESENT STATE OF AFFAIRS: WHAT SHOULD THE RESEARCH PROGRAM IN THE PHYSICAL, GEOLOGICAL, BIOLOGICAL, AND MEDICAL SCIENCES BE?

Arthur R. Schulert, Ph. D., Research Associate, Lamont Geological Observatory, Columbia University, Palisades, N. Y.

This presentation is given as a personal opinion of what the future research program should be in view of the present state of affairs with regard to fallout as discussed at these hearings.

The various testimony which has been presented to date, in spite of conflict in detail, has agreed on 2 basic points: (1) Present fallout represents a definite hazard in that it produces deleterious effects in man and (2) these effects are very small compared to the effect of background radiation which has always been with us. As has been brought out in previous testimony the problem of what our national policy should be in view of this small hazard is not a scientific problem, but a sociological, or a moral one. I think from these considerations, most scientists, and in particular those of us who have appeared before this committee agree that weapons testing should be limited insofar as it is possible and consistent with national security, and indeed, if it were possible, that such testing should be completely eliminated.

However, I would like to reiterate a point brought out by Dr. Dunham on the first day of these hearings—namely, that if we are to proceed into the atomic age with the accelerating tempo of development of nuclear energy, we are faced with the inevitable consequence of a continuous world burden of fission product debris, even without bomb tests, and although we can control this debris, from, e. g., a reactor, to a large extent, a certain irreducible minimum is bound to find its way into the soil and food chain and into man. What I am pointing out is that, bombs or no, we are inevitably going to live with man-made radiation in our bodies. Accepting the opinion that any radiation is dangerous or at least undesirable, it seems to me that a major research effort should be directed at keeping the hazard of fission debris at a minimum. Since internally deposited Sr–90 is generally agreed to represent the greatest long-term hazard, a major component of this problem is to keep the human burden of Sr–90 at a minimum for any given amount of fission. Or, in other words, how can we keep bone deposition of Sr–90 as small as possible and how can we keep the relative body elimination of Sr–90 as great as possible?

May I reiterate that this study is basic no matter what direction the atomic age may take if we accept the philosophy that all radiation is harmful.

[1] Date and place of birth: February 26, 1922, Gladwin, Mich. Education: Bachelor of science in chemistry, Wheaton College, 1943; master of arts (physical chemistry), Princeton University, 1947; doctor of philosophy (biological chemistry), University of Michigan, 1951. Work history: Research assistant, Manhattan project, Princeton University, 1943–46; teaching assistant in chemistry, Princeton University, 1946–47; instructor in chemistry, Taylor University, 1947–48; research assistant in biological chemistry, University of Michigan, 1948–49; teaching assistant in biological chemistry, University of Michigan, 1949–51; fellow, Public Health Research Institute of New York City attached to the New York University Research Service, Goldwater Memorial Hospital, 1951–53; research fellow, Columbia Research Service, Goldwater Memorial Hospital, 1953–55; research associate, Lamont Geological Observatory, Columbia University, 1955–. (Submitted by witness.)

Before discussing in some detail what I feel should be done in this area, may I note that, of course, another important problem in which study should be continued and probably expanded is the problem of more precisely defining the physiological hazard per unit of radiation or per sunshine unit of Sr–90 with regard to such things as the genetic effects, leukemia, bone cancer, and life shortening, so that the degree of danger may be properly evaluated. Since the need in this area was brought out in testimony 2 days ago, and since it is outside of my sphere of activity, I will not discuss it further, except to say, that whatever the effects due to the presence of Sr–90 are, we can presume that they are proportioned to the Sr–90 concentration or approximately so, so that the basic practical problem remains one of reducing the human Sr–90 burden.

Now, if we are going to study the reduction of the body burden of Sr–90, and how this may be achieved, it is of initial interest to define what the present level is. This was dealt with by Dr. Kulp last week and I think the situation on this point is quite well in hand. We have been measuring Sr–90 in human bone samples for almost 4 years, and we now have a worldwide sampling network of over 25 stations sending us samples. We know what the average value for adult man is. We know that the young child is about five times this value. We have been able to follow the increment in Sr–90 in human bones from year to year. There are a few soft spots here that should be filled in. The sampling program should be expanded, particularly in primitive areas where because of low soil concentration of Ca and an exclusively local diet we may find higher values. Another problem is distribution and variation within a skeleton. We are currently attacking this problem through the extensive sampling and whole body determinations of 200 cadavers obtained through the cooperation of the New York City medical schools. We urgently need more information on children's skeletons, but such is difficult to obtain for obvious reasons.

Besides measurements in the bones it is of interest and importance to know the concentrations along the early stages of the biological chain—namely the foodstuff, and the soil. I feel here that the program might be stepped up somewhat particularly with regard to the food monitoring. Although milk has been followed quite adequately, other foodstuffs, to my knowledge have not. It seems to me that these food supplies should also be carefully monitored. Soil data should be extended, particularly in foreign locations.

Now to come back to what I initially stated to be, in my opinion, the key practical problem. How might we reduce the human skeleton deposition of Sr–90 for a given soil concentration of the isotope? We look for ways to block the Sr–90 transmission along the various steps of the biological chain. For completeness I'll just mention two possibilities which I haven't studied, but which have been considered, and which might be considered in the future. One is to bind the Sr–90 in the soil so that it cannot enter the plant. The other is to remove the Sr–90 selectively from the food before human consumption, which seems practically impossible, except perhaps from milk. Let us then consider the problem of minimizing the bone deposition for a given concentration of Sr–90 in the food. Considerable experimental work of this nature has been conducted by Dr. Laszlo in which he studied tracer doses of the short lived isotope Sr–85 instead of Sr–90.

Let me briefly explain the use of Sr–85. Sr–85 is another radioactive isotope of the element of strontium. It differs in that it has a relatively short half life of 65 days compared to the 28 years of Sr–90. We expect the Sr–85 to behave chemically and biologically in a manner identical to Sr–90—such a principle being well established and indeed the basis of a tracer work.

What Dr. Laszlo does is to conduct what we call a balance study; i. e., he carefully measures the amount of Sr–85 which the person ingests, and the amount which is excreted over a period of time in order to determine the net amount retained. He has found that certain agents, specifically, Ca gluconate, some chelating agents, and ammonium chloride reduce the net amount of strontium retained in the body.

From a practical standpoint this work has suffered from two difficulties which I think we can now correct: (1) The number of patients who are candidates for Sr–85 administration is strictly limited and (2) the Sr–85, being given over a short interval of time, is not distributed within the bone in the same manner as the Sr–90, which has been ingested continuously in small quantities for years, so that the results are not completely comparable.

We can now obviate the limitation and get at the precise problem since the level of Sr–90 in the food which man eats and the excreta which he eliminates

is sufficient to be adequately measured. Thus we can now study Sr–90 balance directly in humans and, having determined what the net retention is under normal conditions, we can determine to what extent the net retention may be reduced by control of diet and by various prophylactic and therapeutic measures.

May I add that although it is possible to conduct such experiments on ourselves, and we at Lamont have just completed the dietary phase of such an experiment, it is most practical for the most part to conduct such a study in a hospital metabolic ward such as Dr. Laszlo maintains. Such an extended study is contemplated for the near future.

In conclusion I would like to add that in my mind the extensive study of fallout points up a related problem with which we should all be concerned. If we agree, as we do, that it would be desirable to reduce the present fallout burden from Sr–90, then if we are to keep our proper perspective it seems that we should be even more concerned with other much greater sources of radiation around us that might be reduced. Since it is true that living in a brick house versus a wooden house may give a person an additional radiation equal to 20 times that currently received from Sr–90, should we not be concerned with investigating how one might reduce the background from these particular building materials, or the use of adequate substitutes with less radiation to take their place? If a chest X-ray may give 50 times the radiation currently received from Sr–90 in a year and a fluoroscopic examination will give over a thousand times this amount, should not these things be more rigidly controlled, the practitioner and the layman alike better informed as to their hazards, and basic research conducted so that these necessary medical tools may be used with a minimum of radiation? I realize that in the case of the X-ray, some such work is being done, and I'm not saying that the Atomic Energy Commission should get into this area. In fact they probably should not, but I am merely attempting to again put the fallout problem in perspective by noting that these are still much greater risks even in the field of radiation. Of course the fallout hazard still has the peculiar moral problem in that a person may choose whether or not he'll have an X-ray or live in a brick house, whereas in the case of fallout, there is no place to hide.

Dr. SCHULERT. First of all in the statement I have given the point of view, which is my point of view, that although as has been mentioned there is a considerable area of disagreement which has been evident during these hearings, yet there are some basic general areas of agreement, and we ought to emphasize what these areas of agreement are, and what should be done on the basis of what we do know.

I think we all agree that first of all—this is axiomatic at this point—a definite hazard does exist.

Point No. 2 is that this hazard, though it does exist, is very small compared to the effects of the natural radiation which has always been with us.

I might also interject here, in view of what has been said, that this hazard is small in my estimation and even if we stop bomb testing, we will still have the hazard to some degree due to reactors and so forth. In the industrial use of energy one can control the fission debris largely; yet there is a small irreducible minimum which is bound to get away and get into the human body.

If we take the philosophy that any radiation is hazardous and should be reduced, if possible, then this problem is with us, and the basic research which I will mention today is of importance. Of course, if tests continue it is more important; if nuclear war ensues it is very vital to the life of many people and possibly the Nation as a whole.

Then it seems the basic problem is twofold. Again I am being very axiomatic. First of all to define the hazard, and second of all, to reduce this as much as possible.

In the area of defining the hazard, the first thing might be to determine how much radiation exists. I think again we agree that

the principal hazard is the internal deposition of strontium 90. So the most important phase is to determine just how much strontium 90 is in the human body.

I think on this score we are in fairly good shape. This may sound like boasting since our Lamont Laboratory has been engaged in this phase. I should for the record also indicate that this was underway long before I came to Lamont and I am here in the absence of Dr. Kulp to take his place, and it is more of his show than mine, actually.

We do know pretty well—we have talked about the various uncertainties today—as to how good we know the data and how exact they are. We do know the average concentration of strontium 90 in humans. We know it in adults. I would say we know it to a factor of at least 1.5. We do know that the concentration in children is about 5 times this number from zero to 4 years old. We have been able to follow the increment from year to year. We have gotten some information—perhaps not enough yet—on the geographical variation in this concentration.

Representative COLE. Doctor, would you mind my interrupting?

Dr. SCHULERT. No, go right ahead.

Representative COLE. When you say you do know the concentration in children up to 4 years is 5 times this amount, what amount do you mean?

Dr. SCHULERT. The amount that is present in adults from 20 and above.

There are a few soft spots which I might fill in here. I think we need to expand the program particularly in primitive areas where, because of low calcium in the soil, and a local diet, we might find people with somewhat higher strontium 90 concentrations than the average. We are striving very much to get more data on the bones of children because this is the most important factor, since they are the higher values. As you might imagine, this is the hardest thing to do. We realize it is a rather grim problem to get bones at autopsy from children. The data is more limited here than we would like.

There also remains a little more work to be done on the correlation of the bone data with the data from the diet. I was made aware of this yesterday. We have bone samples from Chile, and we have learned at these hearings that the fallout comes down with the rain. Therefore, Chile, having very low rainfall would be expected to have a low amount of strontium 90 fallout, and it does. Dr. Alexander of the United States Department of Agriculture points out that Chile gets its milk from Wisconsin—dried milk—and this is their main source of calcium (and also strontium 90), and thus their bone concentration of strontium 90 is not as low as one might expect from their soil data.

Representative COLE. Doctor, on the question of milk, you have indicated the principal element of hazard is the strontium 90 and the method of being exposed to that hazard is ingestion. I think you will agree that the consumption of milk is probably the one single item of diet which contains the greatest concentration of this element.

Dr. SCHULERT. Yes.

Representative COLE. I am curious to know, and perhaps you cannot answer, whether there is any body of medical opinion whatever which feels that this degree of hazard of strontium 90 in milk is of

such size as to warrant discouragement of the consumption of milk by growing children?

Dr. SCHULERT. This raises an interesting point. It is true that even the average adult—and we have gotten the figures from many countries on this; do not ask me how many because I do not know offhand—in most places a good deal more than half of your calcium and therefore the strontium 90, we presume, comes from the milk. However, it is also true that the concentration of strontium 90 per gram of calcium is lowest in the milk. That is due to the fact that the cow very fortunately fractionates against the strontium 90. So that the milk that a cow produces has only about one-seventh the strontium 90 per gram of calcium that the cow's diet consists of.

We feel that the important number is the strontium 90 per gram of calcium due to the fact that they go together in the body. Therefore, I would say that the best place to get your calcium (and your strontium 90) is in the milk.

Representative COLE. You still have not answered my particular question. I say maybe you may not know whether there are any doctors of any considerable number or slight number who feel that this content of strontium 90 in the milk is of such amount as to warrant parents to find some substitute for milk.

Dr. SCHULERT. I do not know of any. As I tried to indicate, this would be bad advice. It is better to get your calcium from milk than other food sources, unless you are thinking of synthetic sources.

Representative COLE. I do not know what the alternative sources would be.

Dr. SCHULERT. From any natural source the milk would be the best source of calcium and it has the lowest amount of strontium 90 per gram of calcium.

Representative COLE. Of all the foodstuffs?

Dr. SCHULERT. Of all the foodstuffs we measured.

Representative HOLIFIELD. But it does have more calcium than any other foodstuffs.

Dr. SCHULERT. Yes.

Representative HOLIFIELD. And, therefore, the effect is that although it has less per gram of calcium, it has more in its totality than other foods.

Dr. SCHULERT. That is right.

Representative HOLIFIELD. Therefore, the child should have the milk to get the calcium for his bones and in getting that he must take in an unusual amount of strontium 90, more than he would if he used some other foods?

Dr. SCHULERT. No, you have to have a certain amount of calcium in your diet. I forget the figure. Maybe it is 10 grams. I don't know. If you have to have 10 grams of calcium, you better get it from milk because you will have less strontium 90 for this 10 grams of calcium than from other food.

Senator ANDERSON. I hope we do not leave the impression that milk is not still one of the finest foods for a child.

Dr. SCHULERT. That is right.

Senator ANDERSON. With or without regard to the dangers of strontium 90, there is no finer food in the world for children.

Senator JACKSON. Let us not limit it to children.

Senator ANDERSON. I know what Congressman Cole was trying to get to. In these discussions of how much strontium 90 there might be in milk, somebody might read it and say "I better be careful how much milk my child can absorb." I am completely out of the dairy business. I have sold all my dairy cows, so I am speaking as a disinterested witness. Surely there is nothing in the world that will do the child as much good in the formation of the good solid bone as the constant drinking of sufficient quantities of rich milk. I am very happy that Mr. Cole brought up this question so that before this hearing adjourns we could insert in the record firmly the fact that a child can drink all the milk he wants to all day long and not suffer from strontium poisoning so far as anybody now knows.

Representative COLE. I am very happy that the Senator has emphasized this point. I no doubt was derelict in accepting the doctor's answer with the simple word "no." He said he knew of no doctor who was concerned about the strontium 90 in the milk.

Dr. SCHULERT. You said concerned or was the suggestion that we avoid milk?

Representative COLE. Concerned.

Dr. SCHULERT. Concerned, certainly.

Representative COLE. I meant concerned to the point where they would recommend discontinuance or lessening of consumption.

Dr. SCHULERT. To correct the record, the calcium requirement for a day is 1 gram and not 10.

Senator JACKSON. Do you feel that you can make any kind of prediction now as to what will happen to people as far as strontium 90 is concerned if testing continues at the same current rate?

Dr. SCHULERT. In my prepared statement, and I can say it here——

Senator JACKSON. This is a factor that is not in the natural background. That is why I am asking this question. This is a new element.

Dr. SCHULERT. That is right.

Senator JACKSON. Could you tell us what in your best scientific judgment is apt to occur assuming that testing continues at the same rate, and that the strontium 90 continues at approximately the same rate, as a hypothetical matter?

Dr. SCHULERT. This has been discussed in the hearings before.

Representative HOLIFIELD. The Chair was going to say that this subject was treated exhaustively yesterday in the conference. I have no objection to the question.

Senator JACKSON. I was asking his opinion.

Dr. SCHULERT. It is my feeling that we can compare strontium 90 radiation at least to cosmic radiation, because the energy is roughly the same. To the other natural background it perhaps makes some difference. But the strontium 90 radiation today is about 2 percent of normal background. If testing continues at the present rate, it is calculated that this will go up by a factor of about 8 ultimately. So that this would make it 16 percent of the normal radiation.

Senator JACKSON. What about bone cancer? What is going to be the pattern?

Dr. SCHULERT. Whatever the effect of natural background is today, this would be increased by 16 percent. You can make certain assumptions as to how much bone cancer is due to natural radiation, and this

is uncertain, but whatever the effect is, it would be 16 percent greater. That is the way I look at it.

Senator ANDERSON. I have one question I have been wanting to ask with reference to this. The chart shows that 98 percent of the total radiation is background, and 2 percent may be accumulation from testing and other things. Is there a possibility that the 98 percent may not be as destructive as the 2 percent? By that I mean, is there a possibility that we may have developed through thousands of years a tolerance to cosmic rays that we do not have to strontium?

I see some shaking of heads, but nonetheless I recognize that when we first put out DDT, it was very effective for certain types of insects. Gradually they have developed quite a tolerance to it. We can go through a whole list of remedies and new devices that we have taken, such as penicillin in the human, and the human develops the situation after a while where his diseases do not respond to penicillin, because a tolerance has been developed to it. Could it be that over thousands and maybe millions of years of existence humans have become accustomed to cosmic rays, and therefore are not damaged by the natural background radiation to the same degree that they are bothered by this wholly new substance, strontium 90, that we never knew until 1945?

Dr. SCHULERT. I think in science in general, and particularly in biological science, it is difficult to state anything with absolute certainty. A scientist thinks in terms of probabilities. My own opinion is that if the energy of the cosmic radiation is very close to that which we get from strontium 90, if we have accustomed ourselves to radiation from cosmic ray, we have also accustomed ourselves to the radiation from strontium 90, there would be no added factor.

How sure am I? I can only guess, and I can say I am 99 plus percent sure of this. Since the energies are not exactly identical, it is possible that this precise energy of strontium 90 could hit a vital enzyme in a very special way to break a very critical bond and be much more hazardous. I say the chance is one in a thousand or less. This is the best I can do.

Senator ANDERSON. I realize that nothing is provable, perhaps, with reference to this, but it does seem strange that for a long, long time we have stressed the benefit of sunshine. I recall when I first went to the Southwest suffering with tuberculosis the very first thing the doctor stressed to me was that sunshine was going to be my great curative power. We have had testimony that all types of certain rays—I am not talking about cosmic rays, but strontium 90 and others—may be harmful. I am wondering if natural background radiation is also harmful because we have been talking about living in the sun. If you look at the population statistics, there is a tendency in this country to move into the more sunny States. I just wondered if it is not possible that the human being has not developed a tolerance to sunshine that perhaps makes it beneficial to him. It is a matter that probably is not of importance. I wondered why we worried about strontium 90, if we are worried, and I am, if only a minute particle of radiation we are now receiving and such a large part is already natural background.

Dr. SCHULERT. I share your reaction to the fact that so much is made of the fallout of strontium 90 when the natural background we have

about causes much more radiation. In fact, in the conclusion of my remarks, I do bring out the fact that I think this discussion of fallout should remind us of more important radiation hazards. If it is true that living in a brick house today on the average will give you 20 times the radiation that our present load of strontium 90 will give us, is it not important to try to change our technology to either get this radiation out of the bricks or find substitutes for the brick? If it is true that the average chest X-ray will give you 50 times the radiation that you get from strontium 90 in a year today, and if a fluoroscopy gives you better than a thousand times, should not these be more rigidly controlled? Shouldn't both the practitioner and the layman be educated, and shouldn't research be done so that these things can be reduced to a minimum, still consistent with their need in medical practice?

I think this is true and the people get frightened about fallout and forget the fact that they have had six X-rays in the last 3 years, or had a fluoroscopy and don't worry at all about it.

Representative HOLIFIELD. You may proceed.

Dr. SCHULERT. The other area of defining the hazard, if we confine it to strontium 90, which is the principal hazard, after we know this level, what will the physiological effects be. This has been dealt with in earlier testimony this week. This is an important problem. Of course, research should be continued so we can define as well as possible what the physiological hazard is.

From what I gathered earlier in the week, these things are known to a degree. The degree is not good. Perhaps it is a factor of three. This is much better than knowing nothing about it. We know it to a degree, but it is important to define the degree more precisely. Then going on to trying to reduce the hazard, there are a number of aspects we can consider along this line. Principally again if we stick to strontium 90, how do we reduce the amount of strontium 90 that remains or finds its way to the bone for a given amount of strontium 90 which is released or finds its way into the soil.

If we consider the strontium 90 in the soil, there may be ways to reduce its uptake by plants. This has been considered and it is a tough problem. Once it gets into the food there may be ways to get it out of the food in some instances, but the only example I can think of, is the case of milk. It is possible to take the strontium and calcium out of milk and replace your calcium so that you have milk with no strontium 90.

This is, I am sure, very expensive and technologically it would be very difficult to do on a routine basis.

I would like to confine my remarks primarily to the fact that given a certain amount of strontium 90 in the food, how can we reduce the amount that lodges in the bone. There has been some study on this. Dr. Laszo, the chief of the division of neoplastic diseases at Montefiore Hospital, has studied this problem. He has studied the problem in patients who have received for medical purposes strontium 85. Strontium 85 is another isotope of the element strontium. It differs by virtue of the fact that it has a physical half life of only 65 days whereas strontium 90 has a half life of 28 years. We would expect the strontium 85 to behave chemically and biologically in the manner identical to strontium 90. In fact, this is the basis of all tracer work,

and has been well established, I think. Therefore, we can assume that this is true, and use strontium 85 in our experiments.

What Dr. Laszlo has done is to run what we call a balance study in patients, and the balance study involves the careful measurement of the ingestion of the material, strontium 85, and then a careful measurement of its elimination. Thus if you take the amount ingested, subtract the amount which is eliminated, you have the net uptake by the body. So our problem, then, resolves itself into how can we make this net uptake as low as possible.

Dr. Laszlo and others have found that you can do this to some extent by administering certain therapeutic agents, such as calcium gluconate, chelating agents, ammonium chloride, and others. There are two difficulties, I should say, with the experiments.

One is that these are in hospital patients, and the number of hospital patients who are candidates for strontium 85 administration are strictly limited. So it is a very limited study.

Secondly, when we test this removal we test the removal of strontium 85 which is administered over a short period of time, and this is not the same as testing the removal of strontium 90 which has been ingested in small quantities for a number of years. So the results are not directly comparable.

I think we can now obviate these difficulties because at the present level of strontium 90, it is now possible to measure directly the strontium 90 intake of an individual and his excretion, so that we can do this on you or I or anybody, and having found the net retention under today's conditions, then try various agents to reduce this net retention.

I should add, however, that although you can do it on yourself, and I can do it on myself—in fact, our Lamont group has just completed a dietary phase of such a study, although we don't have the numbers as yet—for the long haul it is much more convenient, shall I say, to do this on hospital patients in a metabolic ward. Shall I say it is socially inconvenient to carry around a suitcase.

So we plan to expand our studies in this direction, not only getting the net retention under present conditions and studying them with various prophylactic and therapeutic agents, but this will give us direct results on this discrimination factor which has been bandied back and forth, another factor which should be known with more precision.

If we can actually do the discrimination under normal conditions with strontium 90 and common calcium, this should give us the answer in this area.

I believe that concludes my remarks.

Representative HOLIFIELD. Thank you very much, Dr. Schulert.

Senator JACKSON. Thank you. You have presented a very good statement.

Representative HOLIFIELD. Are there any comments on Dr. Schulert's statement that anyone would care to make at this time?

Dr. DUNHAM. I am only glad to say that I am happy he was given the opportunity to tell you of the painstaking work involved in these discrimination factors.

Representative HOLIFIELD. Then we will have the Public Health Service witnesses on next. We have Dr. LeRoy E. Burney and Dr. James A. Shannon. Before they present their statements I will insert

in the record at this point a statement from Dr. Henry Blair, director, University of Rochester Atomic Energy Project and his associates.

(The matter referred to is as follows:)

THE UNIVERSITY OF ROCHESTER,
SCHOOL OF MEDICINE AND DENTISTRY,
Rochester, N. Y., June 10, 1957.

A SCIENTIFIC STATEMENT TO THE JOINT CONGRESSIONAL COMMITTEE ON ATOMIC ENERGY

(Atomic energy project—Administered by the department of radiation biology under contract with the United States Atomic Energy Commission)

Mr. Chairman, the following statement represents the considered scientific judgment of the undersigned relative to one of the directions in which we believe the research program in biological and medical sciences dealing with radiation effects should be oriented for the next 10 to 20 years.

Increased emphasis and support should be afforded to long-term studies of radiation effects in longer lived species of animals such as dogs and monkeys whose general physiology and life span more closely approximate those of man.

Much of the present discussion and difference of opinion concerning the biological hazard of atomic fallout and the hazard of exposure to ionizing radiations result from the fact that data from which one can confidently draw conclusions which potentially affect the future of man and of our biosphere are sparse. Many of the available data are based upon painstaking attempts to reconstruct the conditions under which humans were accidentally exposed to toxic materials as long as a quarter of a century ago. There are obvious limitations to the reliability of such data. Other data are based upon careful experimentation with laboratory rats and mice. As useful as the information obtained in studies of such short-lived animals has proven to be, one must be extremely cautious in extrapolating such information to problems affecting man.

By way of perspective we might note that a life-span study using mice requires 3 to 4 years from inception to completion. Similar studies in the laboratory rat may require 4 to 5 years to complete. Studies in longer lived species such as the dog and monkey, whose physiology and life-span characteristics make them more nearly comparable to man, may require from 7 to 15 years for completion.

It is only when long-term studies, some of which are now in progress, have been completed and the information analyzed will we be able to assess with confidence the influence of atomic energy upon man's biological future.

Scientists are somewhat hesitant to undertake such studies for two very important reasons:

1. Such chronic experiments are extremely difficult to perform and sometimes may appear to be impossible to complete. Undertaking these experiments may involve an investment of 5 to 10 years of a man's time before significant scientific contributions appear. Unfortunately a scientist is judged frequently by his immediate scientific productivity and not necessarily by what he might contribute many years hence.
2. The scientific hazards of long-term investigations are of such a nature that even after many years of intense and careful work the experiments may "blow up" as a result of disease or epidemics or accidents in the experimental colony.

Thus the scientist who undertakes work of this nature takes two deliberate and calculated risks—the probable decrease in immediate scientific productivity upon which advancement depends; and the possibility that the experiment may never answer the questions it is designed to answer.

Laboratory program directors and responsible scientific administrators support long-term programs with some trepidation, because such programs are extremely expensive to support and tend to reduce the flexibility of a laboratory in pursuing important shorter term objectives. The nature of fiscal support of research is such that a minor fluctuation in the level of support afforded a laboratory which has long-term research commitments can wipe out much worthwhile short-term research, whereas a substantial budgetary fluctuation may wipe out partially completed work involving many years of scientific effort.

We stress the need for balance and continuity in the support of fundamental and applied research of a short-term nature and research of a long-term nature.

To permit one type of research to prosper at the expense of the other is to jeopardize scientific progress.

Some of the problems resulting from exposure to radiation which are subject to systematic attack by long-term investigations are the following:

1. The relationship of radiation exposure to somatic and genetic mutation.
2. The factors influencing development of cancer and the experimental therapy of cancers.
3. The aging process, whether aging results from exposure to ionizing radiation, exposure to toxic chemical agents, or to natural causes.
4. Fertility and sterility.

The above concepts and opinions are based upon many years of personal experience and observation of both the physical details of performing scientific research and in administrative responsibility for broad programs.

We believe that a statement by the members of the Joint Committee on Atomic Energy affirming its belief in the necessity and desirability of performing long-term animal experiments in the broad fields of radiation effects, carcinogenesis, and cancer therapy, of the aging process, and of genetics would have a widespread and beneficial effect upon both the scientists who must eventually perform this research and upon the public as a whole, whose concern about these problems is already evident.

HENRY A. BLAIR, Ph. D.,
Director, Atomic Energy Project and Professor of Physiology.
HAROLD C. HODGE, Ph. D.,
Chief, Division of Pharmacology and Professor of Pharmacology and Toxicology.
WILLIAM F. BALE, Ph. D.,
Chief, Division of Radiology and Biophysics and Professor of Biophysics.
JOE W. HOWLAND, Ph. D., M. D.,
Chief, Medical Division, and Professor of Radiation Biology.
LAWRENCE W. TUTTLE, Ph. D.,
Chief, Radiation Toxicology Section, and Assistant Professor of Radiation Biology.
GEORGE W. CASARETT, Ph. D.,
Assistant Professor of Radiation Biology.

Representative HOLIFIELD. Dr. Burney, we are happy to have you before us again. Would you like to proceed with your statement?

STATEMENT OF DR. LEROY E. BURNEY,[2] SURGEON GENERAL, ACCOMPANIED BY JAMES G. TERRILL, JR., CHIEF, RADIOLOGICAL HEALTH PROGRAM, PUBLIC HEALTH SERVICE, DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Dr. BURNEY. Thank you, sir.

Mr. Chairman and members of the committee, it is a privilege to appear before this committee, and I do appreciate, on behalf of the

[2] Date and place of birth: December 31, 1906, Burney, Ind. Education: Butler University, Indianapolis, Ind., 1924–26; Indiana University, bachelor of science, 1928; doctor of medicine, 1930. Passed Indiana State Board of Medical Examiners, 1930. Rockefeller fellowship in Johns Hopkins University School of Hygiene and Public Health, 1931–32, doctor of medicine in public health. Work history: Interned at U. S. Marine Hospital, Chicago, Ill., 1930–31; commissioned in Regular Corps of the Public Health Service, 1932; established first mobile venereal-disease clinic service in Brunswick, Ga., 1937–39; Assistant Chief, Division of States Relations, Public Health Service, Washington, D. C., 1943–44; detailed to Navy for 5 months in 1944 and sent overseas by War Shipping Administration to investigate and determine effective measures for diminishing amount of communicable diseases in various Mediterranean ports, especially venereal diseases; director of district No. 4, Public Health Service, New Orleans, La., 1945; secretary and State health commissioner, Indiana State Board of Health, on detail from the Public Health Service, July 1, 1945, to August 1954; Assistant Surgeon General and Deputy Chief, Bureau of State Services, Public Health Service, August 1954 to 1956. Recess appointment as Surgeon General, Public Health Service, August 1956. Membership: American Medical Association, Indiana State Medical Association, Indianapolis Medical Society, fellow of American College of Physicians, fellow of American Public Health Association, past president of State and Territorial Health Officers, conference of State and Provincial Health Officers, Indiana Public Health Association, founders group and trustee, American Board of Preventive Medicine, regent for region 4, American College of Preventive Medicine, Commission of Chronic Illness. (Submitted by Department of Health, Education, and Welfare.)

Public Health Service, an opportunity to discuss briefly the deep interest which the Public Health Service has in the subject of radiation and radioactive fallout, and the activites in which we are engaged.

As the principal health agency of the Federal Government, the Public Health Service is concerned with all those factors in our society which affect the health of people. We in the Service believe—as do our colleagues in public-health work throughout the country—that the emerging and challenging problem of radiation deserves the most careful study and attention. I believe two essential points have been brought into focus:

The first is the great increase in sources of radiation as a result of the accelerating use of nuclear energy for all purposes—military, industrial, and scientific. This will require an acceleration in radiological health research and in the development and application of public health control measures.

The second point is that the varying views that have been expressed by scientists illustrate the lack of definite, generally accepted knowledge about the effects of radiation on human health. It is clear that we do not yet know the full consequences of chronic low level exposure. Geneticists have raised the question of the genetic effect of radiation on future generations.

What has been the role of the Public Health Service with respect to the problem of radiation? What can be done to protect people from its possible hazards and at the same time promote its beneficial uses? What is needed to help State and local agencies which are responsible for health preservation of the public?

In considering these questions, we must keep clearly in mind that health agencies must deal with the total problem of radiation, of which fallout, of course, is a part. For example, the efforts of the Public Health Service in this problem go back over several decades.

In the 1930's, the Public Health Service industrial health personnel shared in the investigation of radium poisoning among painters of clock dials, and later helped solve the problem of safe disposal of radioluminous instrument dials. In 1940 the Public Health Service conducted basic work on radium and X-ray health hazards in hospitals.

Dr. Leonard Scheele, the former Surgeon General, participated in this work, along with Dr. Lorenz, who was mentioned this morning.

Research conducted by the Service staff members contributed significantly to the establishment of human tolerance for radiation and performed other staff work at the Manhattan District project. As a result of these studies, Dr. Egon Lorenz served as a consultant in radiation protection at the Manhattan District. Early in the development of mass X-ray programs, the Service developed techniques for safeguarding the health of the operators of X-ray machines. In 1948 the Service set up a branch to conduct training and investigations in the growing field of radiological health. That branch has been active in providing services to the States in this field.

In addition, the Public Health Service has carried out for the Atomic Energy Commission and Joint Task Force 7 the monitoring of levels of radioactivity at the Nevada and Pacific Proving Grounds. We have presented a technical paper on this activity for your records. (See statement of James G. Terrill, Chief, Radiological Health Program, USPHS, p. 328.) In this activity, we have concentrated on

measuring the radiation received by people in the offsite test areas and on studying the means to reduce exposure. We have also studied reactor waste treatment and discharge problems in cooperation with the Atomic Energy Commission.

In sum, therefore, our concern has been with the total radiation exposure of people, whether the source is natural background, medical equipment, nuclear reactor plants, or weapons testing programs. We have undertaken to discover how much and what kinds of radiation emanate from the various sources and how each affects human beings.

The problem is complex. Ionizing radiation, for example, is an important tool in medical diagnosis and treatment, and each year helps conserve the health of thousands of people. Yet it can obviously cause undesirable and sometimes dangerous effects and, if not used with utmost care, can produce, for example, the cancer it is employed to cure. Thus public health agencies are faced with the problem of promoting the maximum use of this valuable tool and at the same time of developing protective measures against the potentially harmful effects. The deceptive latent period between exposure and effect, the cumulative nature of the action, and the difficulties in measuring radiation have commanded the attention of health workers throughout the Nation.

The National Academy of Sciences has said that the healing arts probably constitute the largest single manmade source of external radiation at the present time. The fact that exposure from other sources is on the increase makes it necessary to reexamine and reassess the use of radiation for research and for healing purposes. Exposure levels from a given source which might have been acceptable in the past may now—or in the future—become hazardous when added to the exposure from other sources.

Turning now to the specific problem of fallout, there seems to be general agreement that the principal long range hazard from weapons testing is through the production and assimilation of strontium 90. The most significant question in the strontium picture is the maximum permissible concentration for human beings. This aspect of the problem is further complicated by the fact that children probably assimilate strontium to a greater degree than adults because their bones are growing, and the rapidly growing tissues of the child are more susceptible per unit of radioactivity. The current concern with this radioactive element must not preclude, however, the continuing study of other radioactive elements, either alone or in combination.

Along with many other agencies we in the Public Health Service recognize the potentialities of the proportional or linear relationship of radiation effects to dosage, particularly with regard to genetic, cancerogenic and aging effects. Pending further evidence, we believe that the recommendations of the National Committee on Radiation Protection represent a reasonable balance between radiation effects and exposure. There is a great need, however, to measure existing radiation levels accurately and to resolve in a more definitive way the biological relationships involved.

The question of biological relationships will be discussed in greater detail by the Director of the Public Health Service's National Institutes of Health. But I do wish to point out now that we are vitally interested in obtaining better data on the fundamental relationships

beween radiation and man. We do not expect to obtain conclusive data in this area in a short period of time. Satisfactory answers will require careful and extensive studies.

Much of our current information on the effects of radiation, particularly the delayed effects of low-level radiation on human beings, is extrapolated from studies with animals. These studies indicate that the damage produced from such exposures is generally similar to diseases and other abnormal conditions already present in the population from causes other than radiation. It follows, therefore, that statistically valid analyses of changes in the incidence of such conditions in the population are needed in order to assess the impact of radiation on man.

A sound scientific basis for collection of data of this type is through epidemiological followup studies of relatively large numbers of people who are incidentally exposed to known amounts of radiation. Studies can be made with groups in certain industries and the healing arts in which the exposure history can be obtained or measured. Epidemiological studies with these groups are fundamental to the problem of obtaining data on which to base realistic, maximum permissible exposure limits, as well as to calculate the risks incidental to any given radiation exposure.

One such study which the Public Health Service is conducting is a long-term followup of the health status of uranium miners on the Colorado Plateau. Several years ago work was initiated to determine the extent of radioactive contamination of the atmosphere in uranium mines and to study and put into effect appropriate control measures. The environmental aspect of this study is continuing, and periodic clinical examinations are being made on more than 1,100 uranium miners. One such clinical examination is now being made on this group of 1,100 miners. Additional information on this study has been submitted to your committee for the record.

Senator ANDERSON. Does that study show anything thus far?

Dr. BURNEY. No, sir.

Senator ANDERSON. They have been mining on the plateau for many years. Have you see anything yet?

Dr. BURNEY. No, sir. We have not found anything in this period of time, but we rather doubted that we would, although some of these people go back to around 10 years of having worked in this area. A large number of them worked in this atmosphere only for 2 or 3 years. With the long latent period we are not surprised not to have found anything.

Senator ANDERSON. Is there any difference in the big Indian area of Utah as compared to the sort of open pit mining that has taken place in the grants area?

Dr. BURNEY. May I refer that to Mr. Terrill here, who has been doing that work?

Senator ANDERSON. I do not care who you refer it to. Is there a difference in the development of exposure inside a mine like the Mivida mine as contrasted with the open mining that Anaconda is doing in the Ambrosia Lake area?

Dr. BURNEY. I would suspect that there is, but I would want Mr. Terrill to answer that question.

Mr. TERRILL. The principal method utilized to lower radiation exposure in the mines is related to ventilation. So the actual concentrations to which the miners are exposed are a function of the ventilation in those mines, as well as the concentration of the uranium ores.

Senator ANDERSON. That is interesting, but does it answer my question? There is no ventilation in the Anaconda open-pit mine. Therefore, I am trying to find out is there any difference in the way a miner is exposed inside a mine of the Mivida nature as contrasted to the open-pit mining in the Anaconda mine?

Mr. TERRILL. Sir, I got the location of the Anaconda mine, but what was the other place?

Senator ANDERSON. The Mivida mine in the Big Indian country in Utah. Take any one. I do not care which one you take.

Mr. TERRILL. They have been relatively small mines.

Senator ANDERSON. They are ventilated?

Mr. TERRILL. Yes.

Senator ANDERSON. Does a miner working in the shaft of that mine with a relatively high uranium content ore, contrasted with work outdoors with the low-grade ore of the Anaconda Co. pick up more harmful radiation? Seventy percent of all known uranium reserves of this country are located where the open-pit method can be used.

Mr. TERRILL. Generally a person who is mining in an open pit would receive less radiation exposure than one who was working in a mine where you are dependent on artificial ventilation. On the basis of field tests the natural ventilation prevents the accumulation of radon and its decay products and thus reduces exposure very significantly.

Senator ANDERSON. Do we have any study which proves that?

Mr. TERRILL. We have a detailed study on underground mines, sir, and I think we have submitted it for the record. We have not studied open pits so intensively because the gross measurements have been so low. All of the details and all the mines that we have investigated have been written up and published in an article by Holiday and others working out of our Salt Lake City field station.

Senator ANDERSON. Does the examination of these 1,100 miners show that they get a relatively larger exposure than those individuals not working in mines, and do we therefore conclude that miners working in the unanium mines need to be rotated more than miners working in a lead, copper, or coal mine?

Mr. TERRILL. Generally they receive more exposure to radioactive materials than other miners.

Senator ANDERSON. I am sure they receive more exposure if they are in a uranium mine than if they are in a coal mine, but what does it do to them?

Mr. TERRILL. That is what the clinical studies are intended to determine over a period of time.

Senator ANDERSON. Then is the answer, We do not yet know?

Dr. BURNEY. The answer is we do not know. In this study, as in similar environmental health programs, we have made use of the cooperative relationships built up through the years with the State and local health agencies. In assessing the health problem, these agencies must consider exposure from all sources of ionizing radia-

tion—weapons, reactors, industry, medicine, and natural background. If the total exposure begins to approach maximum permissible concentrations, it is important that public health agencies seek ways to assess the significance and to apply techniques to maintain exposure at the lowest practicable level.

Senator ANDERSON. In your statement you say the most significant question in the strontium picture is the maximum permissible concentration for human beings, and here you have said something about the maximum concentration.

Dr. BURNEY. We have been using the recommendations of the National Committee on Radiation Protection.

Senator ANDERSON. Do you subscribe to that?

Dr. BURNEY. Yes, sir.

Senator ANDERSON. Did you make an independent investigation of it?

Dr. BURNEY. I can't answer that. We do work with Dr. Taylor and his group on this.

Mr. TERRILL. The Public Health Service is represented on the National Committee on Radiation Protection and all of the data that we collected through our National Institutes of Health or other studies are routinely submitted to the National Committee, and considered by it and our membership on it; yes, sir.

Dr. BURNEY. In other words, control procedures to minimize individual hazards should accompany any anticipated increase in radiological exposure.

Through measurement and epidemiological techniques, we should be able to evaluate in the field many of the sources and to obtain reliable estimates of exposure. This should lead to specific and practicable suggestions on ways in which radiation exposure can be reduced. For example, our studies of water decontamination indicate that the most practical method of reducing radiation exposure from water supplies is through adequate waste treatment and through monitoring and bypassing that portion of the water supply which may temporarily contain undesirable amounts of radioactivity. In order to do this, it will be necessary to do more frequent sampling of water supplies to keep track of any increases over normal background radiation.

Senator ANDERSON. When you refer to waste treatment, is there some established pattern of treating wastes?

Dr. BURNEY. We are investigating, again in some instances, with the Atomic Energy Commission and some on our own at the Robert A. Taft Sanitary Engineering Center at Cincinnati, the problem of treatment of industrial wastes or other radioactive waste. As far as I know, there is no economical and satisfactory method at the present time for major sources of nuclear waste.

Senator ANDERSON. I read your sentence again. For example:

> our studies of water decontamination indicate that the most practical method of reducing radiation exposure from water supplies is through adequate waste treatment.

In order to find that out, you must have know what adequate waste treatment was. What was it?

Dr. BURNEY. This would depend on the elements that were present in this treatment. We do know that normal waste treatment, includ-

ing filtration and aeration and even chlorine treatment, does not remove all of the radioactive material. But it does help to a certain extent.

Senator ANDERSON. To what percentage would you estimate aeration would remove the waste?

Dr. BURNEY. I will have to ask Mr. Terrill for the answer.

Mr. TERRILL. Mr. Anderson, we have found that normal types of treatment remove radioactivity only in terms of percentages comparable with other similar chemical substances. In most instances in the radioactive waste treatment we are interested in removing substances by several orders of magnitude. Those orders of magnitude, of course, vary anywhere from your high-level wastes that you have to treat at AEC processing installations on down to relatively low levels of waste that you get either industrially or from medical uses. Special waste treatment methods, such as distillation and ion exchange remove radioactivity by many factors of 10, but these methods are comparatively expensive and don't solve the disposal problems.

Dr. BURNEY. His question was what percentage of removal can we expect from waste treatment.

Senator ANDERSON. That is right. You have a reactor operating at Hanford. It has radioactive wastes. What percentage of the radioactive waste in there could be handled by aeration, as you have suggested? Practically none?

Mr. TERRILL. Practically none.

Senator ANDERSON. You could say that for the other accepted fashions?

Mr. TERRILL. Yes; for normal waste-treatment methods.

Senator ANDERSON. We do have a problem of waste disposal, do we not, that has not yet been solved?

Dr. BURNEY. That is correct.

What is needed, generally speaking, is a program that will be alert to any changes in our radiation environment and that will be ready to institute control measures which may be called for.

In the field of medicine, advances are being made in radiological techniques which result in lower exposures to clinical personnel as well as to patients. A concerted effort on the part of the health professions is needed to control harmful amounts of radiation in connection with diagnositic procedures—and such an effort is already underway.

These are but a few of the areas in which action can be taken. Their effectiveness, however, depends to a considerable extent on the participation by official health agencies and educational and scientific institutions, as well as on the close cooperation of the civilian and military agencies of the Federal Government.

In order to assume their proper roles, States and local agencies—in whom basic responsibility for protecting the health of their citizens rests—need to develop more technical competency in the field of radiological health. The Public Health Service has been able to provide short orientation and special training courses, primarily to States and local health personnel. We have also used our commissioned officers in monitoring radioactivity levels for the Atomic

Energy Commission in Nevada, thus providing firsthand field experience to personnel from health agencies.

To meet the long-range needs of health agencies, we believe it will be necessary for the professional and scientific schools of the country, which provide the basic training for our health workers, to strengthen their programs. Some Federal assistance is being provided through our general public-health traineeship program and through the research grants and fellowship programs administered by the Public Health Service's National Institutes of Health. It is apparent, however, that more specific emphasis will be necessary to provide the number of trained persons required.

In summary, we believe that radiation is a health problem of growing significance. Health agencies are, of course, looked to for leadership and counsel wherever a health problem exists and they must face the problem of radiation as it relates to health with vigor and determination. To do the job will require cooperation among all concerned at all levels of government. The Public Health Service pledges itself to this ideal.

Senator ANDERSON (presiding). Are there any questions?

Chairman DURHAM. Doctor, I am sorry I was not here at the beginning of your statement. What kind of a setup have you got in your department for this type of operation?

Dr. BURNEY. Basically we have two groups that are concerned with this problem. One is at the National Institutes of Health, where research and training in the broad field of physical biology which includes radiobiology has been done for many years, in which research is done within the various institutes—the National Cancer Institute, the Heart Institute, the Arthritis and Metabolic Institute—and also in which grants are made to outside scientific institutions for research in the field of radiobiology.

We have at this present year, I believe, about $2 million worth of grants going to scientific institutions for research in radiobiology itself. Then training programs for training research workers in the various sciences related to radiobiology—biophysicists, biochemists, and similar groups.

Chairman DURHAM. Is that entirely related to the field of radiation?

Dr. BURNEY. Yes, sir. It includes the effects of radiation in production of cancer. It is not entirely on the matter of radiation exposure as we are talking about it here.

Chairman DURHAM. Is all of that information made available to the different State public-health agencies?

Dr. BURNEY. Yes, sir, it is. Then in addition to that, in our field activities we have a very effective relationship with the Atomic Energy Commission and have had for a number of years in working with them on field studies. We have one or more men working at the Oak Ridge Laboratory, on the industrial waste problem that Senator Anderson mentioned, to try to solve that. We are also doing work at our sanitary engineering center in Cincinnati, in trying to evolve methods of disposing of radioactive wastes, and measuring radiological effects.

We are concerned in our air-pollution program as well as our water pollution with developing better sampling techniques, both in air and

water. Of course, radioactivity detection is an important part of that.

Chairman DURHAM. You have full cooperation from the AEC in all matters pertaining to this?

Dr. BURNEY. Yes, sir; we work very closely with them.

Chairman DURHAM. Can you give the committee the amount of money that your agency is spending on this program?

Dr. BURNEY. Excluding the National Institutes of Health—as I say, $2 million of those funds is for extramural research grants—Dr. Shannon may be able to tell you exactly how much we spend on our intramural programs. He tells me $250,000 on direct research.

Chairman DURHAM. Do you submit a separate budget for this, Doctor, identified as radiation health hazard?

Dr. BURNEY. No, sir, it is not; it is part of the National Cancer Institute budget, part of the arthritis and metabolic-disease budget. We do have a special Radiation-Study Section which reviews all of the requests.

Chairman DURHAM. This is a growing matter of concern. Have you given any thought to treating this as a separate item?

Dr. BURNEY. We do have, other than our research program at the National Institutes of Health, a separate item for radiological health. That at the present time amounts to $347,000. We requested quite a sizable increase in 1958. The House allowance was not quite what we asked for.

Chairman DURHAM. Would that apply on waste hazards?

Dr. BURNEY. Yes, sir. Research in waste treatment. It applies also to the training of State and local personnel in this area. It applies to the epidemiological studies we are trying to do, and it applies to the technical help we give the States. For example, one State wanted to develop a radiological health program. They had no experts.

They asked us to lend them an expert in the field for 2 years until they could develop their own people. At the end of that 2 years' time, they had their own people. We brought our person back and he is working with us again.

So that is the kind of technical assistance that we give to the States.

Senator ANDERSON. Thank you, Dr. Burney.

(A supplementary statement by Dr. Burney follows:)

STATEMENT TO SUPPLEMENT PRESENTATION BY THE SURGEON GENERAL, PUBLIC HEALTH SERVICE, DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, BEFORE THE JOINT COMMITTEE ON ATOMIC ENERGY HEARINGS ON THE NATURE OF RADIOACTIVE FALLOUT AND ITS EFFECTS ON MAN

An example of the long-term epidemiological and statistical study needed to better define the effects of ionizing radiation on humans is the continuing evaluation of the health status of uranium miners on the Colorado Plateau. Several years ago work was initiated to determine the extent of radioactive contamination of the atmosphere in uranium mines and to study and put into effect appropriate control measures where needed. The environmental aspect of this study is continuing and the data were reviewed in a recent publication by the Public Health Service.[1]

---

[1] Control of Radon and Daughters in Uranium Mines and Calculations on Biologic Effects: U. S. Department of Health, Education, and Welfare, Public Health Service Publication No. 494.

Various aspects of the degree of environmental contamination and measures for its control have also been reviewed in other publications.[2 3 4 5 6 7 8]

Armed with knowledge regarding the levels of radiation exposure, and following pilot work during previous years, a full-scale epidemiological followup study was initiated in 1954. At that time, physical examinations were conducted on 1,124 uranium miners in the field, to serve as a baseline for future observations. At yearly intervals since that time, a census has been conducted to define the current status of each individual in the study and this month a series of followup physical examinations will begin. To date, the followup has been quite successful. Although the group under study presents technical problems in followup that are somewhat more complicated than might be true in other groups, primarily because of its somewhat transient nature, approximately 95 percent of this original group have been successfully contacted since their original examination. It is anticipated that by the use of more sophisticated methods which are available to organizations with competencies in the field of epidemiology, this followup rate may approach 100 percent.

To date, it is much too soon to derive any definitive conclusions from this study. Among the reasons is the fact that previous experience,[9 10] has indicated that the damage, if any, might be delayed for many years after the beginning of exposure. The following table indicates the percentages of individuals under study falling in each of several categories with regard to the number of years of exposure to date:

Percentage distribution of miners by years exposure

Years	*Percent*	*Years*	*Percent*
Less than 1	38. 2	4 to 6	14. 4
1 to 2	20. 2	7 to 9	4. 2
2 to 3	11. 6	10 to 12	1. 0
3 to 4	8. 6	13 and over	1. 8

Although this study illustrates some of the problems which one encounters in conducting a long-term epidemiological and statistical study, we believe it also demonstrates the fact that epidemiological studies in this field can be successfully prosecuted.

Dr. Shannon, will you come forward please?

STATEMENT OF DR. JAMES A. SHANNON, DIRECTOR, NATIONAL INSTITUTES OF HEALTH, PUBLIC HEALTH SERVICE [10a]

Dr. SHANNON. Mr. Chairman and members of the committee, the Surgeon General of the Public Health Service has emphasized the

---

[2] Kusnetz, H. L. Radon daughters in mine atmospheres: A field method for determining concentrations. Amer. Ind. Hyg. Assoc. Quarterly, 17: 85, 1956.

[3] Tsivoglou, E. C., and H. E. Ayer. Emanation of radon in uranium mines and control by ventilation. Arch. Ind. Hyg., 8: 125, 1953.

[4] Idem. Ventilation of uranium mines. Arch. Ind. Hyg., 10: 363, 1954.

[5] Ayer, H. E. Control of Radon and its Daughters in Mines by Ventilation. United States Atomic Energy Commission AECU-2858. The Commission, Washington, D. C., 1954.

[6] Coleman, R. D., H. L. Kusnetz, P. F. Woolrich, and D. A. Holaday. Investigations on the supplemental control of radon and radon daughter hazards in mine atmospheres. Amer. Ind. Hyg. Assoc. Quarterly, 17: 405, 1956.

[7] Holaday, Duncan A. The Radon Problems in Deep-Level Mining. A. M. A. Archives of Ind. Health, 12: 163–166, 1955.

[8] Tsivoglou, E. C., H. E. Ayer, and D. A. Holaday. Occurrence of nonequilibrium atmospheric mixtures of radon and its daughters. Nucleonics, 11 (9): 40, 1953.

[9] Peller. Lung Cancer Among Mine Workers in Joachimsthal, Human Biol., 11: 130–143 (1939).

[10] Sikl, H. The Present Status of Knowledge About the Jachymov Disease (cancer of the lungs in the miners of the radium mines). Acta. Unio Intern. contra Cancrum, 6: 1366–1375 (1950).

[10a] Date and place of birth: August 9, 1904, Hollis, New York City. Education: Bachelor of arts, College of the Holy Cross, 1925; doctor of medicine, New York University, 1929; doctor of philosophy, New York University, 1935. Work history: Instructor in physiology, College of Medicine, New York University, 1932–35; assistant professor of physiology, New York University College of Medicine, 1935–41; guest investigator, physiological laboratory, Cambridge University, England, 1936; assistant professor of medicine, New York University College of Medicine, 1941–42; associate professor of medicine, New York University College of Medicine, 1942–46; director, Squibb Institute for Medical Research, New Brunswick, N. J., 1946–49; special consultant to the Surgeon General, Public Health Service, 1946–49; associate director in charge of research, National Heart Institute, National Institutes of Health, 1942–52; associate director, National Institutes of Health, in charge of intramural affairs, 1952–55; director, National Institutes of Health, 1955–. (Submitted by Department of Health, Education and Welfare.)

need for further research on the problems of radiation. It is in this area, and in the area of scientific training, that the activities of the National Institutes of Health relate to radiation.

The National Institutes of Health, as you know, is a research and training organization within the Public Health Service. We support medical and related research in universities and medical schools through grants which currently total about $90 million per year. We administer a training program which helps support the training of about 5,000 scientific personnel annually. We also carry on basic and clinical research in our own laboratories.

Our contribution to the problems of radiation lies primarily in further exploration, in our own laboratories and through research grants, of the manner in which radiation affects human beings. As the Surgeon General has pointed out, a more thorough understanding of the manner in which radiation affects biological systems is necessary.

The basic research inquiry into the effects of radiation as it relates to health, I believe, can best be approached in the context of physical biology, broadly defined. Physical biology includes the study of the means by which all physical phenomena act within biological systems.

It includes the effect of heat, cold, pressure, light, sound, vibration, acceleration and deceleration, electrical properties of molecules, the movement of material across membranes and ionizing and nonionizing radiation. As a field of study it depends heavily upon modern mathematics and physics and scientific instrumentation.

We share the view of those who believe that physical biology as a discipline will, in all probability, exert a dominant force upon medical and biological research over the coming decades.

Research in physical biology has for some time been a part of the total program of the laboratories of the Public Health Service's National Institutes of Health. We have, in fact, a Laboratory of Physical Biology in which research in some of the areas noted above is now being carried on. This laboratory, for example, has been under contract by the Armed Forces special weapons project in studies dealing with post radiation infections. This collaborative project is still continuing.

Another illustration, in a different field, is found in the contribution of the late Dr. Egon Lorenz of the National Cancer Institute to which Dr. Burney has already referred. His work in the general physiology of radiation effects, including protection against radiation damage, stands as a significant contribution in this field.

The National Cancer Institute conducts a substantial research program in clinical radiology, together with a basic program relating to the biological effects of ionizing radiation.

At the National Institutes of Health, we hope to place the highly specialized research in radiobiology within the context of the broad area of physical biology. For example, we are establishing a panel on mathematics attached to the research operation in physical biology. This panel will work on the applications of advanced mathematical techniques to biological problems, including the use of a large electronic computer in the solution of these problems.

Organizationally, we hope to place research in radiobiology in a single organization—the existing Laboratory of Physical Biology.

We intend to expand this laboratory at a moderate rate, primarily because of problems relating to research facilities and the shortage of scientific manpower in this field.

Research scientists themselves initiate the studies which are supported by NIH grants. Hence there are limits in the degree to which it is possible or advisable to change emphasis in fields of study. Experience has shown, however, that when an area of study is promising, investigators can be persuaded to enter or intensify their efforts. This is done primarily through persuasion by colleagues and a change of research emphasis by free choice. About $4.5 million per year in research grants now supports work in physical biology; of this amount, $1.2 million is for work in radiation biology.

Scientific manpower is in extremely short supply in the entire field of physical biology, including radiobiology. Because this is a relatively new field, fully satisfying career opportunities are few in number. A sound program for strengthening the field of physical biology should therefore encompass the establishment of a number of academic posts with adequate status, stability and income.

Attraction of additional students is, of course, also important, as is the content and vigor of their graduate instruction. There are waves of advance in fields of science which attract people as new and exciting fields open up. Nuclear physics has been one such field. Its immense popularity is shown by the fact that in 1951, 36 percent of all physicists in the age group 25 to 29 were nuclear physicists. Among those in the 50- to 54-age group, only 12 percent were nuclear physicists.

While the distribution of advanced students by field is fixed to a substantial degree by the stage of evolution of a science, this distribution can be affected—without producing accompanying harmful effects—by a carefully considered program.

The Institutes are establishing such a program as part of a total effort to increase the pool of research manpower for medicine and related scientists. Last year, for example, a senior research fellowship program was established. The fundamental objective of this program is to provide stable support for the most promising young investigators in the 5 to 10 years after they complete work for the doctor of philosophy in sciences basic to medicine. This is the critical period when too many younger investigators drift out of research. This obviously results in a loss of scientific manpower. In addition, the medical schools find it difficult to train people of high caliber in the teaching posts available for the sciences basic to medicine.

In fiscal year 1937, about 1,000 postdoctoral and 1,000 predoctoral fellows were preparing for research careers with the aid of fellowship funds of the NIH. In addition, almost 3,000 individuals were in training with the aid of training grants.

Over the past few years, the Institutes have emphasized the significance of training to the future of research in medicine and the related sciences. We are referring here to training not at the highly specialized technical level, but at a high level of scientific competence.

The total training activities of NIH have expanded rapidly. In fiscal year 1956, the appropriation for training activities approximated $17 million, while $33 million was provided for fiscal year 1957.

With this increase, substantial funds will be available to support advanced students in physical biology. Gradually, emphasis will shift

to the problem of recruitment and the problem of the content of training. The problem of motivation of students to study science—a problem which is fundamental to our national scientific effort—is shared by many groups ,and principally the National Science Foundation.

On the other hand, we believe that NIH can help universities set up courses in physical biology which will take students to the frontiers of knowledge. We also believe that NIH can help outstanding men in the field to find outstanding students who have already acquired a sound university grounding in science. Drs. Schmitt and Bolt of the Massachusetts Institute of Technology are working, on this problem in biophysics with the aid of a Public Health Service grant.

In conclusion, I should like to stress our conviction that a soundly conceived and operated research training program is a prerequisite to the successful solution of applied problems of radiobiology—of which the problem of radiation is one. In the field of manpower training we believe that the Public Health Service can make a most significant contribution to resolution of the problems related to radiological health.

Senator ANDERSON. Thank you, Doctor. I am glad to hear of the development of this work. Are there any questions?

Representative COLE. Mr. Chairman, I want to comment just a little bit on the doctor's statement, comparing the number of nuclear physicists in 1951 of the 25- to 29-age group as against those at that time of the 50- to 54-age group. The younger group is 36 percent of the total, and the older-age group is 12 percent of the total.

Frankly, I am not so much impressed by the fact that 36 percent of the total in 1951 were nuclear physicists, although that is encouraging, but I am very much impressed by the 12 percent in the upper-age region. That would lead me to conclude that as long ago as 25 or 30 years ago scientists became interested in nuclear physics as a profession. Is that a fair conclusion?

Dr. SHANNON. No, sir. This field was really opened up fairly widely in the 1930's. I think of the 12 percent of the older physicists who are in nuclear physics now, a sizable proportion of them are in there as a result of what one might call a retread, with the acquisition of new skills. In other words, I do not believe, despite the fact of the upswing of nuclear physics in the 1930's, that a sizable proportion of them entered this field initially. A 50-year-old physicist would have completed his degree in the 1920's. This is far too high a proportion to believe that they originated as nuclear physicists.

Representative COLE. I suspected there might be some such explanation.

Dr. SHANNON. The reason for quoting the figures, sir, is to indicate how one can in a carefully planned course, providing it is an intelligently planned course and makes sense, divert scientific talent without at the same time interfering with individual decisions.

Senator ANDERSON. Are there any additional questions? If not, this concludes what we are doing, with four exceptions. We want to make sure that if there are any closing and final statements that anyone wants to give, that we will not miss them. I particularly want to call on four people.

Dr. Waterman.

Representative VAN ZANDT. Mr. Chairman, may I ask Dr. Burney a question before he leaves the stand?

Senator ANDERSON. Yes.

Representative VAN ZANDT. Dr. Burney, you conclude your statement by saying, "To do the job will require cooperation among all concerned at all levels of government." That poses this question: How active are the several States in setting up some kind of control of radiation and the use of radiation?

Dr. BURNEY. That has been spotty, Mr. Van Zandt. Let me say first that there are several States, New York, California—I don't want to leave out any States, but I am naming those as examples—who have developed some good personnel and have what we would term a good radiological health program. But within the last 12 months there are a large number of other States which have recognized the importance of this problem, which have asked us for help in assigning personnel, or have sent personnel to our courses for training in order that they can go back and set up a program of their own. I would say it has been spotty, but it is growing now in State and local health departments.

Representative VAN ZANDT. With the development of knowledge and its dissemination, are you satisfied with the progress that is being made by the several States in setting up the necessary controls?

Dr. BURNEY. No, sir, but I would like to say that I am not satisfied with what the Public Health Service has done, either. I would not be too critical.

Representative COLE. I have a question of Dr. Shannon. I had to hurry over your statement because I had to answer a quorum call. I happen to be a member of another subcommittee of this Joint Committee that has to do with the problem of shortage of physicists, and so forth. Testimony that we took last year led us to believe that there is a terrific shortage. I am just wondering how long is this shortage going to exist on the effort that is being made today?

Dr. SHANNON. I would say, to pick a figure in years, certainly it will be here with us for at least 5 years. That figure is taken from an appreciation that a graduate degree takes a minimum of 3 years, and even with very active prosecution of programs, a 5-year period is much too short a time to figure that the shortage will have been remedied.

One of the important things is that wholly apart from the importance of physicists to programs in radiobiology, physicists now have a tremendous amount to contribute to the total field of biology and medicine. They are just coming into their own. You will have competition for physicists, biophysicists, physical chemists, and the like, from wholly new areas that were not available to compete for them as short a time ago as 5 years.

It is really an appreciation of this acute shortage which we feel will grow that led us a year and a half ago to mke our first positive effort to provide training opportunities for these university people.

Representative VAN ZANDT. Dr. Shannon, with your knowledge of the overall field, is it proper to say that there is a tremendous amount of enthusiasm?

Dr. SHANNON. Yes, sir.

Representative VAN ZANDT. And the field is attracting a lot of young capable people?

Dr. SHANNON. I will take the "yes" back until I am sure I know precisely what you have reference to. There is a tremendous amount of enthusiasm for entering training as physicists. There is not a proportionate amount of enthusiasm for entering the more restricted field of radiobiologists. There is essentially little in the way of professional training programs. We in the Cancer Institute have been attempting to develop professional personnel along these lines in a very positive way for a 5-year period. We have been wholly unsuccessful. This has led us in the past year to redefine our interest as being much broader than radiobiology, to redefine it in terms of physical biology, where we feel that the career opportunities of sufficient breadth will open up to attract people to them. But at the present time there are not sufficient university posts in the field of physical biology to really attract the brilliant youngster. We feel that the field has to be defined broadly and the research opportunity given which will automatically attract people into the field.

Representative VAN ZANDT. Thank you.

Representative COLE. How long does it take to make a nuclear physicist after he has finished high school?

Dr. SHANNON. Roughly 7 or 8 years. This would be the conventional college course with a 3- or 4-year graduate course superimposed.

Representative COLE. That would lead to a doctorate?

Dr. SHANNON. Yes, sir.

Senator ANDERSON. Dr. Waterman of the National Science Foundation.

STATEMENT OF DR. ALAN T. WATERMAN, NATIONAL SCIENCE FOUNDATION [11]

Dr. WATERMAN. I have no prepared statement, Mr. Chairman. I can, however, make some general comments.

First of all, with regard to the interest of the National Science Foundation in this program, we are, as you know, concerned primarily with basic research and training in the sciences and in that program we do not ourselves conduct research; but we provide support for research by means of grants to institutions for the support of work by scientists on projects selected from these that come to us. In that program we do have in the biological sciences a number of grants that are concerned with research in genetics and related problems, which are basic to the general topic of this committee. When it comes to work which is more directly of the applied nature, then this is not a province of the Foundation. We would leave this to the National Institutes of Health, the Public Health Service, and other agencies.

[11] Date and place of birth: June 4, 1892, Cornwall-on-Hudson, N. Y. Education: Bachelor of arts, Princeton University, 1913; doctor of philosophy in physics, Princeton University, 1916. Work history: Instructor in physics, University of Cincinnati, 1916–18; 2 years military service with Science and Research Division of Army Signal Corps in World War I; faculty member of Yale University in department of physics until 1948, with leave of absence during 1927–28 on a national research fellowship to King's College, London; to Massachusetts Institute of Technology in 1937, and to Office of Scientific Research and Development from 1942 to 1946; appointed Director of the National Science Foundation April 6, 1951, and reappointed April 6, 1957; serves as a member of the Defense Science Board and the Advisory Panel on General Sciences of the Department of Defense, and of the Science Advisory Committee and the Committee on Specialized Personnel of the Office of Defense Mobilization. He is also a member of the President's Advisory Committee on Weather Control; board of directors of the Center for Advanced Study in the Behavioral Sciences; board of trustees of Atoms for Peace Awards and the board of directors of the American Association for the Advancement of Science. (Submitted by Witness.)

93299°—57—pt. 2——29

That is to say, we are not concerned with research which is aimed at the diagnosis, treatment, and cure of disease; but, rather, a better understanding of the fundamental processes which occur in the body or in nature which have to deal with these subjects.

We have a fellowship program which provides about 800 predoctoral fellowships in all the sciences each year. It is a national program. We also have about 100 postdoctoral fellowships, and we have certain other postdoctoral fellowships for really senior research investigators, and faculty science fellowships primarily for teachers.

In our assistance to training in science, we have 96 summer institutes who select the field of radiobiology, but they are not large in number.

In our assistance to training in science, we have summer institutes which we are financing this summer, whereby teachers of science can get together in their individual sciences for 6 or 8 weeks, for the study of teaching methods and study of research material for teaching use. Six of those summer institutes are conducted and sponsored jointly by ourselves and the Atomic Energy Commission in the field of radiobiology. It is largely to have the teachers informed with regard to this subject.

In addition, we have provided funds to the Atomic Energy Commission for a program at Oak Ridge which will train high-school teachers in instructional methods and send them around the country so that the schools of the country and educational institutions can learn directly about nuclear energy and radiobiology. Those are the activities in our program which bear directly on this program.

The research and training aspects in this whole field are regarded as very important indeed.

With respect to the general subject, I cannot qualify as a research expert in particular fields. However, I am a physicist by profession and for the past 15 years or so have been concerned with the broad features of various aspects of this whole subject, such things as nuclear weapons, nuclear power, fallout, radiobiology, and similar problems.

In the middle of so much discussion, it seems to me, it pays to keep attention on some of the main features and not lose sight of them. This problem that we are dealing with is puzzling for a very fundamental reason, because the two alternatives are not really comparable. On the one hand, one has the national issue of the danger of war and the need to have superiority in the latest weapons. If it were clear that anything we do could establish a clear superiority in a weapon of any kind, and by that act prevent war, I am sure the problem would be much clearer, and the country would do whatever is necessary. Unfortunately, the evidence in these matters is not easy to come by, and the conclusion is very puzzling to make. But this is a national question which involves primarily our own country and the countries of the free world, and is of the gravest importance.

On the other hand, we have the question of fallout and the dangers of radiation. This is much more a personal and individual matter. There are individual reactions to this. Also it applies to all mankind and not only our country.

Furthermore, it involves a type of consideration which in the history of mankind has always been a very serious thing to face: the danger of the unknown. When one realizes a danger he has no

control over, and knows nothing about, to him it is a big danger, much more of a danger than a car running down the street which he can see and thinks he can avoid.

The next thing is that the consequences pointed out by scientists are frankly ugly and repulsive to him, for example, such matters as the effect on future generations. So this becomes a problem which to the individual is much more serious in his mind than many of the ordinary, more dangerous problems we face. That has to be reckoned with.

It has been said before, but bears repeating, that what we are talking about in this fallout danger and other radiation danger is not a new danger. It is a danger that has already existed in the lifetime of mankind. The level of that is quite considerably greater than any new dangers we are talking about. So we are merely talking about adding to the present danger. This then becomes a matter of probabilities, and probabilities are not an easy thing to get clear.

For example, if it were established that carrying a watch with a radioactive dial ran 1 chance in 100 million of proving fatal to the carrier, I suspect that many people would continue carrying watches. There would certainly be a few who would stop at once. On the other hand, if the risk were 1 in 10, probably nobody would carry radioactive dial watches.

The matter of estimation of the probability in this case is a hard thing to do and it reacts differently on different people. To take an illustration which is closer to this problem, we know, for example, that cosmic rays come in to the earth in greater quantities at the Poles than they do at the Equator, due to the magnetic field of the earth. This means that the danger from radiation from cosmic rays is greater at the Poles than at the Equator. Because that is true. does that means that the population immediately should move to the Equator? I think that poses the kind of question we are up against. It is probably true they are safer there, but is it worth it? So one runs into that type of consideration in dealing with the whole question.

I have just one more comment to make and then I would be very glad to answer any questions. While the difficulties in this are great, the promise of future valuable results and data bearing on the problem is also very great. By the evidence you have heard today, there are a number of fields of research that are now being prosecuted vigorously and in capable hands. We can confidently expect that these will produce results. It will be everyone's hope that these results can be speeded up in every possible way. Probably as research goes on, more different methods will occur to us. The power of this kind of research is very great. From our limited point of view at the present time we can see certain areas that are bound to be promising. We know we are going to get more precise information on the basis of which to make decisions and in certain other directions we can find that certain constructive things can be done to forestall the dangers, such as some treatment by which the body can deal with these menaces more efficiently. However, at any given time there are things that are unforeseen that will be discovered that will be most important.

My personal view about this is that we should lose no opportunity to prosecute this research in competent hands, and also that we lose no opportunity to see to it that those who are attracted and have

ability along the lines of this type of research are persuaded to go into it. Research cannot be done without competent people, and we should therefore see to it that we train these competent people. Only by the systematic study in competent hands will we really reach a good conclusion. In the meantime it seems to me that today's hearings and the other hearings which have been held are pretty convincing testimony that by the interest and thoroughness with which this committee has approached these problems and by the competence and thoroughness with which the Atomic Energy Commission and the Institutes of Health and others have been working along these lines, we have every reason to believe that we are on the right track. If anything else is needed—and I think it is—we have the two committees that have been mentioned—the National Committee for Protection Against Radiation and the committee of the National Academy of Sciences that can be counted upon to monitor this from the standpoint of desirable standards of tolerance levels, and so on, and also from the standpoint of review of the research which is going on. If that is done, and continued actively, it seems to me that we have the situation as well in hand as we could possibly hope.

Senator ANDERSON. Thank you very much. Are there any questions?

Chairman DURHAM. I have one statement. I do not believe I ever heard a finer summary of the whole problem in so few words.

Dr. WATERMAN. Thank you.

Representative COLE. I was going to say exactly the same thing, except I may have used different words. I think it is most appropriate, Doctor, that you are the last witness. I am not sure but it presently appears you are the last witness to make this résumé, and a synopsis of the subject matter that is of concern to this committee and the cause of the hearing. Like Mr. Durham, I compliment you on your capacity to pierce through the maze of all this information that has been given and heard in the last 2 weeks and lay out in the ledger for us and the public to evaluate the different factors that are to be taken into consideration in striking a balance on this problem.

Dr. WATERMAN. Thank you, sir.

Senator ANDERSON. Thank you very, very much, Dr. Waterman.

Dr. Alexander, do you have any short comment you wish to make before we are thraugh with this hearing?

Dr. ALEXANDER. I might say a few words on the part of the Department of Agriculture.

Senator ANDERSON. We certainly do not want that Department left out, we assure you.

Dr. ALEXANDER. I thought you would not. I always feel that Senator Anderson has an interest in the Department of Agriculture from some years back.

The program of the Soil Conservation Service of the Department of Agriculture is entirely one of support to the fallout program that has been presented here. We collect samples, select areas of work and help in interpretation of results in relation to food supplies for man and animals.

The Agricultural Research Service carries out basic research that contributes to our understanding of the behavior of fallout in soils, plants, and animals. We in the Department of Agriculture are glad to be a part of this program.

I might say for Mr. Cole and Mr. Anderson that my grandchildren are drinking copious quantities of milk, and I have no fear whatever for their future from the standpoint of the hazard of strontium 90.

Senator ANDERSON. From the size of you, you have drunk some yourself.

May we find out if either of the representatives of the Department of Defense have a word as we close?

Dr. SHELTON. I have no formal statement for you, but I do want to say a few words in order that the committee be informed of the fallout research being conducted in the Department of Defense, and principally in the Armed Forces special weapons project, a name which has occurred repeatedly in testimony to you.

I would like to repeat very briefly the pertinent portions of some of that effort.

In regard to Dr. Dunham's statements to you concerning the AEC research program in fallout and effects on man, several of the programs mentioned are jointly funded by the AEC and DOD. Dr. Dunham's organization and the organization which I represent work rather closely together on these common problems.

Representative COLE. At that point, Dr. Dunham indicated the total as I recall of $30 million. Does that indicate that the AEC's portion is $30 million, or does that $30 million include defense appropriations?

Dr. SHELTON. When we jointly fund a program, I would imagine he would include his contribution to that program and probably not include our contribution in the amount he has given.

Representative COLE. Are you going to tell us what your figure is?

Dr. SHELTON. I will indicate roughly those numbers; yes, sir. I wanted to indicate to you that the Department of Defense participation in documenting fallout and its effects on man are of a research nature, and we do have a broad interest. The AFSWP research directly related to fallout and its effects on man can be conveniently divided into three rather distinct areas. The first area in which the Department of Defense through the Armed Forces special weapons project has participated has been the documentation of fallout on the weapons test series. This activity has been principally concerned with the local fallout. Considerable effort has been expended and is now being expended in analyzing and reducing the data obtained to date. We anticipate our main effort in future will be along these lines.

It must be realized that millions of dollars and an extremely large effort has been expended in documenting fallout in order that from the standpoint of the Department of Defense the weapons can be properly employed, if necessary. We are not really complacent, however, regarding local fallout.

Senator ANDERSON. I saw you jump across a couple of pages.

(Discussion off the record.)

Dr. SHELTON. I do have a few more statements to make.

We are not complacent in the Department of Defense regarding local fallout and hence we are not complacent about that portion which is left over, which will manifest itself in the latitudinal and the worldwide fallout. As new weapons and brust conditions arise, we will certainly document those, as well as appears feasible.

There is a second large area in which we are participating and that is because of the importance of the worldwide fallout. The Depart-

ment of Defense does have a stratospheric program. The Department of Defense program has been coordinated with a similar project in the AEC. The Department of Defense will continue research on worldwide sampling and we do utilize the private organizations for analysis of those samples.

There is a final and third large area of research in the Department of Defense, and that concerns the laboratory programs on the effects of ionizing radiation on man. Lieutenant Colonel Hartgering of Walter Reed gave you the results of the uptake of various isotopes in man during testing.

Chairman DURHAM. How much of your work is classified and how much is declassified?

Dr. SHELTON. That changes from time to time. During a test series the work under progress at that time may be classified. Indeed, the subject just mentioned as performed by Colonel Hartgering is an example; we did not know during the periods of taking those samples on the Nevada operation, and finally the Pacific operation, just what would turn up. We did not want to reveal the number of shots. So that was classified. It was classified at one time, and we reviewed that subject and we were happy to present those results to you and they were unclassified. What is classified today may not be classified tomorrow.

Chairman DURHAM. I am speaking primarily of radiation and fallout.

Dr. SHELTON. Details of the fallout from a specific shot of a specific yield, having a specific burst condition, has been treated in a classified manner. It reveals specific information on that particular event. The AEC and we have periodically released fallout patterns of a generalized nature to indicate the areas and hazards involved. So we do the best we can in getting out as much information in an unclassified manner. We use classified information to develop the unclassified.

Representative VAN ZANDT. Dr. Shelton, how long does it generally take you to complete your study and then release to the public nonclassified information?

Dr. SHELTON. If something is of a startling nature, such as the obvious very large fallout patterns being involved with thermonuclear weapons, such as the March 1954 shot; we released the fallout patterns from that within a period of 4 to 5 or 6 weeks following the event. It was put in the best way we knew how not to divulge specifically the weapon, but to provide the necessary information to the people, and that came out jointly by the AEC and Department of Defense.

Representative VAN ZANDT. In other words, it takes about 4 or 5 or 6 weeks for you to delete the classified material from the information which you receive after a test.

Dr. SHELTON. Typically for us to really understand what we have and to put it out out and not have to retract and change it. We have consolidated a position of accuracy of the data by then.

We are continuing the human measurement program at Walter Reed, and we are extending that to other lines of approach. Dr. Langham mentioned the LASL whole body counter. We anticipate using a whole body counter in the Department of Defense. We would have available to us a large group of people whom we know where they

have been stationed, and where they will be stationed in the future. We will be checking some personnel and we hope to make a contribution in that area.

Concerning the effects of strontium 90 on man, we are jointly funding with the AEC two sizeable projects, one being the strontium 90 programs on dogs, and we have another one with them at Oak Ridge. At the present time we are spending in these two projects and others something over a quarter of a million dollars annually on research directly related to the biological effects of fallout on man. None of our medical programs are classified. That is, the results come right out as they are produced at those individual places.

So I have covered, then, the three broad areas in which the Department of Defense continually and has in the past expended quite an effort. We are well aware of the problems and we are making our contribution to solving the problems.

Senator ANDERSON. Thank you very much. I am privileged to check some of the work in one of the areas where the special weapons project is interested. It is a very active operation. I know how much you have done, and I want to compliment you on it.

This brings to a close the first part of these hearings. I do hope as a result of these hearings the various agencies which have testified, the various groups that have been represented, the scientific fraternity generally, will examine these hearings and give the committee such guidance as they may be able to for any subsequent hearings we may have.

We are going to regard these as adjourned, merely until additional material may be obtained and the committee, I am sure—and I am speaking for the chairman of the committee as well as the chairman of the subcommittee—thank all the witnesses very sincerely for their fine contributions.

Chairman DURHAM. I want to thank the scientific profession for the excellent hearings we have had. It is something that will be valuable for the future and to the country at large.

Senator ANDERSON. Thank you all.

(Thereupon at 4 p. m., Friday, June 7, 1957, the hearings were adjourned.)

APPENDIXES

Appendix 1

Principal Technical Speeches and Papers on Fallout by Commissioner W. F. Libby

[Reprinted from Science, v. 122, July 8, 1955: 57–58]

Dosages From Natural Radioactivity and Cosmic Rays

W. F. Libby[1]

The radiation dosages that people receive from the natural radioactivities and cosmic rays have been calculated and are given in Tables 1, 2, 3, and 4. Some direct observations are given in Table 5.

Table 1 gives the dosages in milliroentgens per year for exposures at various altitudes directly over ordinary granite, typical sedimentary rock, and open oceans. Surface dosages decrease with height above the ground because of air absorption; 50-percent reduction occurs for every 370 ft.[2] For comparison purposes, it is interesting to note that in the United States, the average exposure *rate* from total fallout from atomic tests on 1 Jan. 1955, was about 1 mr/yr.[3] The total dose during 1954 probably averaged about 15 mr,[3] principally because of the Pacific tests in the spring.

The values listed in Table 1 were calculated on the following basis. The roentgen was taken to be 100 ergs of energy per gram of water. (Actually this definition is that of the rad, the internationally recognized unit of radiation dosage. For gamma rays it is nearly equal to the roentgen, which is 93 ergs/g.) The absorption coefficients of all radiations in tissue were taken as being equivalent to those of water. The dosages from the natural radioactivities in the earth were calculated on the approximation that the energy absorbed per gram by the human body on the surface of the earth is, to a sufficient approximation, equivalent to that absorbed per gram by the top layers of the rock of the earth's surface itself from the gamma radiation emitted by the rock.[4] In other words, the total gamma-ray energy produced in a gram of granite from the thorium, uranium, and potassium contained was taken to be equal to the energy absorbed per gram of human tissue in the human body on the surface, except that a factor of 2 was used to correct for the geometric loss. It was interesting to observe that this simple method of calculation gave results in good agreement with those based both on separate consideration of each of the complicated radiations emitted by thorium and uranium in the rocks and on the use of the individual absorption coefficients for these radiations in tissue, together with correction for the "buildup" factors as the radiation is scattered and diffuses out of the rock.[5]

The abundance of uranium, thorium, and potassium in granite were taken as 4×10^{-6} g/g (*1*), 13×10^{-6} g/g (*1*), and 0.03 g/g, respectively (*1*). In selecting these numbers, it was realized that these were only averages and that fluctuations around these values do occur, that uranium contents as high as 200 ppm have been found in granite, and that thorium has been found as abundant as 500 ppm in some granites.

---

[1] The author is a member of the U. S. Atomic Energy Commission.
[2] H. Faul, Nuclear Geology (Wiley, New York, 1954).
[3] M. Eisenbud and J. H. Harley, Science 121, 677 (1955).
[4] This calculation was kindly suggested by L. D. Marinelli of Argonne National Laboratory.
[5] U. Fano, Nucleonics 11, No. 8, 8 (1953); 11, No. 9, 55 (1953).

For sedimentary rocks, the general average figure of one-fourth of the values quoted for granites has been used. It is realized, however, that this is very approximate, because the amounts of the various radioactive minerals in the sedimentaries fluctuate widely. The abundances of uranium and potassium in sea water were taken, respectively, as 1.3×10^{-9}g/g[1] and 3.5×10^{-4}g/g[1]. The abundance of potassium in the human body was taken as 2×10^{-3}g/g,[6] and the abundance of carbon in the human body was taken as 18 percent. For the calculation of the dosage from radium assimilated in drinking waters throughout the normal lifetime, the bone weight was taken as 10 percent for the adult man. But for relatively brief periods of assimilation when the radium would be expected to be concentrated in the small volumes of the bone most metabolically active, the figure of 1 percent was used. All these numbers of the human body were taken as being equivalent to those of the "standard man".[7]

The dosages resulting from cosmic radiation were calculated from the ionization chamber data of Millikan et al.[8] From these data the dosages were calculated at altitudes up to 20,000 ft. and at the latitude of 55° N (geomagnetic) as well as at the geomagnetic equator. The results are given in Table 2. It should be mentioned that the biological effects per unit energy may be larger for cosmic radiation, because it consists of high-energy particles rather than gamma radiation.

The natural radioactivity in the human body contributes the dosages given in Table 3. Of the 19-mr/yr dosage from potassium, 17 mr/yr is from the beta rays of the potassium itself. These were taken to be of a mean energy 40 percent of the maximum energy of 1.36 Mev. The specific activity of natural potassium was taken as 1800 beta rays per gram, per minute and 180 gamma rays of 1.45-Mev energy per gram, per minute.[9] The gamma rays that contribute the remaining two units of the dosage of potassium were calculated on the basis of the assumption that only half of the gamma-ray energy is actually absorbed in the body. This leads to the result that in a packed crowd the radioactivity from the potassium in one's neighbors' bodies contributes an additional dosage of 2 mr/yr.

TABLE 1.—*Total radiation dosages from normal background radiation (mr/yr)*

Altitude of ground surface (feet)	Ordinary granite		Typical sedimentary rock		Open ocean	
	Equator	55° N	Equator	55° N	Equator	55° N
Sea level	143	147	76	80	53	57
5,000	150	170	83	103		
10,000	190	230	123	163		
15,000	270	350	203	283		
20,000	414	560	347	493		

The dosage from carbon was calculated on the basis of the assumptions that the body is 18-percent carbon; the specific radioactivity of carbon is 15 disintegrations/g, per minute;[10] and that the mean energy of the beta radiation is 40 percent of the maximum energy of 167 kev.[9]

In Table 4, various ordinary but somewhat unusual circumstances are used to illustrate the types of exposure that can occur in normal living. A wristwatch worn 24 hr/day that has a luminous dial assumed to have 1 μc of radium per watch—a figure perhaps slightly larger than the average—would give the central body, including the sex organs, a dosage of about 40 mr/yr. An airplane pilot flying 24 hr/day with an instrument panel consisting of 100 dials with 3 μc of radium each would receive, at an average distance of 1 yd, a dosage of 1300 mr/yer.

In order to check whether the dosages calculated here and given in Tables 1 to 4 are essentially correct, some direct measurements reported by various

[6] L. D. Marinelli, private communication.
[7] "Recommendations of the International Commission on Radiological Protection," NBS Handbook No. 47 (1950), p. 16.
[8] R. A. Millikan and H. V. Neher, Phys. Rev. 50, 15 (1936); H. V. Neher and W. H. Pickering, ibid. 61, 407 (1942).
[9] "Nuclear Data," Natl. Bur. Standards U. S. Circ. 499 (1950), suppl. 1, 2, and 3.
[10] W. F. Libby, Radiocarbon Dating (Univ. of Chicago Press, Chicago, 1952).

observers are given in Table 5. They agree reasonably well with the external component of the total dosages given for sea level in Table I—the residues after subtracting 20.5 mr/yr, the dosage from body radioactivities given in Table 3.

It is interesting that the variations in natural dosage are large and under certain conditions the natural dosage may be nearly 100 times higher than the minimum—the dosage of seafarers. The fallout dosage rate in the United States on 1 Jan. 1955—1 mr/yr—was only 2 percent of this lowest natural dosage rate. Of course, during a test period when bombs are fired, the fallout dosage rates may approach, or somewhat exceed, the natural dosage rate for a few days before decay and weathering processes reduce them in a few weeks to rates that are small percentages of the natural background.

TABLE 2.—*Cosmic ray dosages*

Altitude (ft.):	*Dosages (mr/yr)*
Sea level	33 to 37
5,000	40 to 60
10,000	80 to 120
15,000	160 to 240
20,000	300 to 450

TABLE 3.—*Radiation dosages from the natural radioactivity of the human body*

Source of radioactivity:	*Dosage (mr/yr)*
Potassium	19
Carbon	1.5
Radium (bones only), uniform distribution	6.7
Radium (bones only), nonuniform distribution	[1] 67

[1] The radium content of the human body is based on data of A. F. Stehney of Argonne National Laboratory.

TABLE 4.—*Radiation dosages in various ordinary circumstances*

Radiation source	Location	Dosage (mr/yr)
Wrist watch (1 μc of Ra per watch)	Central body, including sex organs, at average distance of 1 ft.	40.
Luminous dials in airplane cabin (100 dials with 3 μc of Ra each).	Pilot is taken to be at an average distance of 1 yd from the dials.	1300.
X-rays [1]	Lumbar spine, anterior-posterior	1500 each.
	Lumbar spine, lateral	5700 each.
	Pregnancy, anterior-posterior	3600 each.
	Pregnancy, lateral	9000 each.
Uranium ore (0.1 percent—the minimum accepted by the AEC for purchase).	Flat surface ground	2800.
	Mine with all walls of ore	5600. (neglecting radon)
Phosphate rock (commercial fertilizer 0.01 to 0.025 percent U).	Flat surface ground	280–700.
People	Packed in crowd	2.

[1] A. H. Sturtevant, "Genetic effects of high-energy irradiation of human population," address given 11 Jan. 1955 at California Institute of Technology.

TABLE 5.—*Experimental data for hard background radiation (mr/yr)*[1]

Observer	Cosmic rays	Gamma rays: From air	Gamma rays: From ground	Cosmic and gamma rays (total)	Location
Sievert and Hultquist	44	--------	--------	121–150	Streets of Stockholm
				104–182	Over igneous rocks, Sweden
				94	Clay soil[2]
				104	Wood houses (average center of room)
				145	Brick and concrete houses (types 1, 2)[2]
				296 (max., 520)	Brick and concrete houses (type 3)[2]
Cowan	--------	--------	--------	98	Outdoors, Brookhaven, N. Y., measured[3]
Hess and Vancour	34	2	53	90	Outdoors, Fordham Univ. campus, N. Y., 1 m above ground[4]
Burch	31–34	--------	62	94–96	Leeds, England[5]

[1] Kindly collected by L. D. Marinelli of Argonne National Laboratory.
[2] R. M. Sievert and B. Hultquist, "Variation in natural gamma radiation in Sweden," Acta Radiol. 37, 388, 399 (1952).
[3] F. P. Cowan, "Everyday radiation," Phys. Today 5, No. 10, 10 (1952); also AECU-1138.
[4] V. F. Hess and R. P. Vancour, "Ionization balance of the atmosphere," J. Atm. and Terrest. Phys. 1, 13 (1950).
[5] P. R. J. Burch and F. W. Spiers, "Radioactivity of the human being," Science 120, 719 (1954); P. R. J. Burch, "Cosmic radiation," Proc. Phys. Soc. London A67, 421 (1954).

[Reprinted from Science, April 20, 1956, Vol. 123, No. 3199, pages 657–660]

RADIOACTIVE FALLOUT AND RADIOACTIVE STRONTIUM

W. F. Libby[1]

The radioactivity that falls out of the atmosphere after the explosion of a nuclear weapon is called the radioactive fallout. In the ordinary atomic bomb, for example, for each 20,000 tons of TNT equivalent of explosive energy, about 2 pounds of radioactive materials are produced. In these 2 pounds are some 90 different radioactive species varying in lifetime from a fraction of a second to many years. This mixture of radioactivity decreases in radioactivity in such a way that for every sevenfold increase in age, the total radioactivity is decreased tenfold. Thus the radioactivity by 7 hours after the explosion has decreased to one-tenth the radioactivity of 1 hour, and in 49 hours to 1/100, in 2 weeks to 1/1000, in 3 months to 1/10,000, and so forth.

The conditions of fallout are largely determined by the amount and type of material vaporized into the fireball of the bomb itself. A bomb fired in the air contributes such a relatively small amount of matter to the cloud that the particles formed after dissipation of the enormous energy released are of necessity very tiny and therefore very slow in settling. The result is that most of the radioactivities are expended in the air and the area over which the fallout occurs is rendered very large indeed, extending to the ends of the earth in minute although detectable amounts.

A bomb fired on the surface of the earth, however, may have an appreciable portion of its radioactivity reprecipitated within relatively short distances, while bombs fired beneath the surface of the earth may place essentially no fallout radioactivity in the atmosphere. Therefore, the question of the area of contamination to be expected from nuclear weapons cannot be answered categorically without specifying the degree of contact of the fireball with the surface of the earth and probably also specifying the characteristics of this surface. Obviously water would differ considerably from soil in its ability to precipitate radioactive fallout. The coral in the southern Pacific islands that are used for the larger United States weapons tests will under the great heat decompose to form calcium oxide, which will then rehydrate to form calcium hydroxide, which in turn will absorb carbon dioxide to form a crust of calcium carbonate. Obviously such a complicated series of chemical reactions will make the fallout particles from the great tests at Eniwetok differ from what would be observed if

[1] The author is a member of the U. S. Atomic Energy Commission.
[1] The author is a member of the U. S. Atomic Energy Commission. This article is based on a speech given at Northwestern University, Evanston, Ill., Jan. 19, 1956.

the same weapons had been fired over ordinary sand or granite. We cannot imagine all of the details in which the nature of the soil will affect the local fallout, but it is clear that the effects will be substantial.

In the weapon test operations, great care is taken to insure that no danger results from fallout. Criteria are used that are meant to insure that this is so. However, it is well to note that it is from the test operations that we have learned what we do know about the problem of civilian defense against fallout. We must speak of test experience, for it is the only source of experimental information about the phenomena of radioactive fallout.

The radioactivities resulting from the burst of a nuclear weapon can be classified as follows: (i) radioactivities induced in the environment and (ii) products dependent directly on the nature of the weapon. The environment can be made radioactive only by neutrons, but all nuclear weapons involve large numbers of neutrons, some of which are certain to escape into the surroundings.

RADIOACTIVITIES INDUCED IN THE ENVIRONMENT

Taking air bursts first, our problem is: What do neutrons do to air? The answer is simple. They make radioactive carbon, C^{14} which has a half-life of 5600 years. Fortunately, this radioactivity is essentially safe because of its long lifetime and the enormous amount of diluting carbon dioxide in the atmosphere. The cosmic rays themselves make neutrons, which, of course, make radiocarbon. In fact, the earth has on its surface a total of 80 tons of radiocarbon from the cosmic radiation. Now, since each neutron forms one C^{14} atom of mass 14 times the neutron's mass, this corresponds to 5.2 tons of neutrons, and we see that this enormous number of neutrons would have to be produced and escape in order that nuclear weapons would just double the feeble natural radioactivity of living matter due to radiocarbon. Such an increase would have no significance from the standpoint of health. The atmosphere itself contains only 1.5 percent of the total carbon with which the cosmic-ray-produced radiocarbon is mixed, the main part being dissolved in the sea, so we expect that nuclear weapons could produce a short-range rise in the radiocarbon content of the carbon dioxide in the atmosphere, which later would decrease as the atmospheric carbon dioxide mixed with the sea. Therefore, only 1.5 percent as many neutrons would be required to double the natural radiocarbon content of atmospheric carbon dioxide for this time before mixing with the sea could occur, or about 78 kilograms or 170 pounds of neutrons. To orient ourselves, the 20,000-tons-of-TNT-equivalent atomic weapon involves the fission of 1 kilogram of uranium or plutonium and the liberation of about 10 grams of neutrons. If all these neutrons escaped into the atmosphere, it would obviously require 7800 such weapons to double the radiocarbon content of the atmospheric carbon dioxide even with no mixing with the sea, and about 520,000 with complete mixing. These correspond to explosive energies of 156 and 10,400 megatons of TNT, respectively, if all neutrons formed escaped. A reasonable escape figure might be 15 percent, so we can expect that nearly 1000 megatons of fission would be necessary just to double the atmospheric radiocarbon content, and that about 66,000 megatons would be necessary for the same effect on a long-term basis.

The interchange between the atmosphere and the sea water, which is constantly taking place, would deplete and remove the excess radioactive carbon dioxide. Now it is known from measurements of the radioactive hydrogen, tritium—which is also made in the atmosphere by the cosmic rays—that this interchange is slow. In fact, we learn that the radioactive water is formed by the burning of the tritium made by the cosmic rays is not diluted by more than the top 100 meters or so of sea water in its lifetime of about 18 years. The carbon dioxide dissolved in the water is about equal to the total in the air. In other words, a dilution by more than the twofold that corresponds to the dissolved carbon dioxide in the top 100 meters of ocean water would take longer than 18 years. However, the dilution by this factor of 2 would occur essentially immediately within a matter of weeks or months. Therefore, we would have to double our estimates for even the short time scale activation of the atmosphere to reach the enormous figure of 2000 megatons of fission required. Thermonuclear weapons, of course, also involve neutrons. For a given energy release, they produce somewhat more neutrons than fission weapons; however, the order of magnitude of atmospheric activation would not be greatly different. So our estimates apply to all nuclear weapons. The essential point is that the atmosphere is difficult to activate and the activities produced are safe. In addition to

carbon-14, there are a few others produced in low yield; they include tritium and very short-lived products, but none is produced in sufficient amounts to be hazardous.

For weapons fired on the surface, the activation of the surface materials is a possibility, but in general it appears that most of the neutrons form stable isotopes and that the amount of radioactivity produced, at least with ordinary surface materials, is relatively small. The principal radioactivities produced by nuclear weapons are produced in the weapons themselves, and not in the environment.

RADIOACTIVITIES PRODUCED IN WEAPONS

Turning now to the radioactivities naturally produced in nuclear weapons themselves, probably the most important is radioactive strontium, which has a half-life of 28 years. The first reason this is so important is that strontium is chemically similar to calcium, which is one of the main mineral constituents of the body. Bone consists principally of calcium phosphate, and for this reason radiostrontium, like calcium, is deposited in the bone. The amounts of ordinary nonradioactive strontium naturally present are so small that the radioactive strontium will follow ordinary calcium into the body. The second reason that radioactive strontium, Sr^{90}, is an important fallout radioactivity is that it has a long but finite lifetime—28 years half-life, 40 years average life—and thus has a persistent effect. Third, because of its bone seeking property, it stays in the body a long time. Fourth, the probabilities of body ingestion can be high. Finally, the fifth reason for its importance is that strontium–90 is produced in high yield in the fission reaction—about 4 or 5 percent of all fissioning atoms yield this isotope.

In order to orient ourselves about this, let us consider the maximum permissible concentration recommended by the National Committee on Radiation Protection for AEC workers for radiostrontium—1 microcurie for the standard man, whose body is taken to contain 1000 grams of calcium in total. The maximium permissible concentration is of course well below any level at which one would expect any damaging effects to appear. On the basis of experiments with animals, statistically observable increases in the number of bone tumors *should not* be expected to appear at *less than* 10 times this level.. As we go above this figure, the chance for bone tumors occurring increases rapidly so that the likelihood of bone cancer with 30 to 40 times that figure is appreciable.

INTAKE OF STRONTIUM–90

Let us consider in some detail the mechanism by which this most important fallout radioactivity produced in nuclear weapons might be expected to enter the human body. The first point is that from the point of view of fallout there are essentially two classes of nuclear weapons—the high-yield megaton weapons and the lower-yield kiloton weapons. All nuclear weapons produce atomic clouds that rise to heights dependent on the energy released, and the clouds from the megaton class of weapons rise rapidly up through the tropopause and pass into the top layer of the atmosphere, which we know as the stratosphere. This part of the atmosphere is essentially isolated from the lower layer in which we live, the troposphere, and where all of our normal winds, storms, and so forth, occur. Therefore, radioactivity produced in megaton weapons is placed largely immediately in the stratosphere, while the smaller kiloton weapons produce clouds that in general do not reach into the stratosphere, but stop near the tropopause—the imaginary boundary between the stratosphere and troposphere—and have the bulk of their radioactivity left in the troposphere.

In the troposphere where rain occurs, any particulate matter will be washed down in a period of days or weeks. It is easy to show, for example, that 0.1 inch of ordinary rainfall will probably remove essentially completely all particulate matter except for that which is so small as to be almost of molecular dimensions. In other words, for 0.1 inch of rainfall one can be quite certain that the air between the layer in which the rain originates and the ground is washed clean of fallout activity, except for the minute fraction that may be so small that it moves with the air out of the way of the falling raindrops as they make their way toward the earth; and even this tiniest fallout material is likely to be precipitated also, for it will migrate rapidly by molecular-type motion and in this manner is likely to absorb itself on other particulate material and so be rained out. For these reasons, tropospheric radioactive fallout does not stay in the atmosphere for more

than a matter of weeks. It may make two or three trips around the earth in a given latitude before being entirely removed, but its lifetime in the atmosphere will be a matter of weeks.

This is in very sharp contrast to the material that is placed in the stratosphere by megaton weapons; this material appears to stay there for a matter of years. Perhaps 10 years is a good average, at least for the weapons fired to date. It is well to bear in mind that this conclusion may be dependent on the nature of the material carried up in the cloud, but our present experience indicates that the fallout from megaton weapons that does not occur essentially in the first few hours or days, and is therefore deposited mainly locally, is deposited only at a very slow rate corresponding to an average time in the stratosphere of about 10 years. As a result of this long residence time in the highest layers of the atmosphere, the winds mix and distribute the radioactive material broadly over the earth and one finds, when the fallout does finally find its way down into the troposphere where the rain and snow wash it out, that the rates of recipitation are relatively uniform over the entire earth's surface.

Returning now to radiostrontium—at the rate of 1 kilogram of fission for 20 kilotons of TNT equivalent, 2 megatons of fission energy would be equivalent to very nearly 1 millicurie of strontium-90 per square mile of the earth's surface, or about 79 disintegrations per minute, per square foot of the earth's surface. The average soil of the earth has about 20 grams of calcium that is in a form available for plant metabolism in the top 2.5 inches for each square foot of area. Now, recalling the maximum permissible concentration level of 1 microcurie per standard man and noting, as will be shown later, that in order that this concentration not be exceeded, the topsoil of the earth should not contain any more radiostrontium than would correspond to 10 times the concentration in the human body that is just permissible—that is, 1 microcurie per 1000 grams of calcium, or 2200 disintegrations per minute, per gram of calcium—we find that 11,000 megatons of fission energy would produce this average level of radioactivity. Actually, as I will indicate, there can be a concentration of strontium-90 in the soil about 10 times greater than the recommended maximum permissible concentration before one would expect a man living in such an environment to accumulate a maximum permission concentration. The afore-mentioned 11,000 megatons of fission energy would yield a strontium-90 content in human beings just equal to the maximum permissible concentration (MPC) ; at less than 10 times this value, or below 110,000 megatons energy equivalent, statistically observable incidence of bone tumor should not appear; but at 30 to 40 times the MPC, or 330,000 to 440,000 megatons, the likelihood of untoward effects would be appreciable. Even the lowest of these figures is very far in excess of the total energy released to date.

KINDS OF FALLOUT

High-yield weapons fired near the surface have a portion of their activity deposited in and on particles large enough to fall out in the first few hours or days. Thus we have three kinds of fallout from high-yield weapons.

1) The first, or local, is due mainly to large-sized particles. This may cover a considerable area depending mainly on winds. In the 15 February 1955 release of the Atomic Energy Commission that described the experience in the Marshall Islands in the Castle test series in the spring of 1954, some 7000 square miles were described as being contaminated by this type of fallout.

2) The second fallout from the high-yield weapons is that portion which resides on the small particles, but which never reaches the stratosphere and thus stays in the troposphere until it is carried down by rainfall or settles out. There is thus a band of fallout in the same general latitude as the test site; the material may circle the earth two or three times before it is precipitated, but it does fall out within the first few weeks.

3) However, a large part—half or more depending on firing conditions—of the radioactive yield from high-yield weapons resides in the third category, which is the fallout that occurs from the stratosphere itself. Of course, some of the large local fallout may form particles which were lifted into the stratosphere, but which were so large and so bulky that they fell out rapidly anyhow. The finely divided material that reaches the stratosphere apparently stays there for years in the main. A slow leakage through the tropopause into the troposphere occurs—apparently something like 10 percent per year descends. Measurements of the strontium-90 content of soils, rain and snow, and biological materials on a worldwide basis have all shown that strontium-90 fallout occurs all over the

world at rates that are not very dissimilar from one another, except that there is a tendency in the middle latitudes in which the tests are conducted for an extra fallout, presumably of the aforementioned tropospheric variety. Since the completion of the Castle series of tests two years ago, this worldwide rate of fallout has approximated 1.5 millicuries of radiostrontium per square mile, per year. We thus see that radioactive fallout from the stratosphere is a very slow process. This is very fortunate indeed, since the high-yield weapons thus have a major part of their radioactivity dissipated in the atmosphere in a harmless way if they are fired in the air or on the surface.

The fallout apparently occurs in the final step by a washing down of the tropospheric air by rain together with direct falling. The radiostrontium descends from the stratosphere into the troposphere by the processes of diffusion and falling, and is then caught up by the tropospheric weather and in a matter of a few days is deposited. Reasonable estimates for the middle latitudes indicate that the average life in the troposphere is about 1 week.

DEPOSITION OF STRONTIUM-90

The radiostrontium comes down mainly in raindrops although fine morning mists and fogs may be particularly effective in this regard also, as well as surface contact and direct falling. It descends on the foliage and on the soil. That fraction of it which falls on plant leaves has a good chance of being absorbed directly into the plant—much in the way that most modern leaf fertilizers operate. The Eniwetok tests were conducted on coral islands and as a result their fallout may be largely water-soluble. In any case, direct measurements of the radiostrontium content of alfalfa and other crops showed them to be appreciably higher in radioactivity than the soils on which they grew, strongly indicating that a leaf assimilation mechanism is important. The rain falls and carries radioactivity, but when it runs off to the rivers and the seas it is nearly pure because of the action of the soil in absorbing the fallout, so that rivers are essentially free from radiostrontium. Lakes and reservoirs have a content that corresponds approximately to their surface areas only. The radiostrontium is absorbed in the top 2 or 3 inches of soil and held there very tenaciously. Plowing, of course, buries it more deeply, but it appears that in unplowed soil the radiostrontium does not move in a matter of 2 or 3 years.

The researches on radiostrontium conducted by the Atomic Energy Commission have been extensive. The AEC has sampled soils on a world-wide basis and submitted the samples for analysis of radiostrontium content to the Health and Safety Laboratory of the New York Operations Office of the Atomic Energy Commission, the Lamont Geological Observatory of Columbia University, and the Enrico Fermi Institute for Nuclear Studies at the University of Chicago. Direct fallout collected on gummed papers, milk and cheese, alfalfa, animal meat and bone, and even human bodies has been extensively studied. On the basis of the information so obtained, it is possible to say unequivocally that nuclear weapons tests as carried out at the present time do not constitute a health hazard to the human population insofar as radiostrontium is concerned, and it is believed with good reason that radiostrontium is likely to be the most important of the radioactivities produced. It is well to note that since radiostrontium is assimilated in the bones it constitutes essentially no genetic hazard, for its radiations do not reach the reproductive organs.

The milk and cheese radiostrontium content is not as high, relative to that of the grass which the cows eat, as one might expect. There appears to be a discrimination against the fallout material such that the calcium in milk and cheese is roughly one-fifth to one-tenth as radioactive with radiostrontium as the grass that the animals eat. There are various possible physiological explanations of this, and the conclusion itself may not be completely certain, but the data available to date indicate this to be true. In addition, the plant uptake of radiostrontium from soil does discriminate somewhat against radiostrontium as compared with calcium. The calcium taken up from the soil into the plant has in general about one-half the radiostrontium content that the soil calcium has. These two results protect the human population against ingestion of radiostrontium, since milk and cheese are the principal sources of calcium in the human diet. We find, therefore, that the radiostrontium content of human bodies is the lowest of all animals measured and is lower than the average soil and the average foliage by tenfold. The Sr^{90}-to-calcium ratio in young people—

whose bones are still forming—corresponds to about 1/1000 of the maximum permissible concentration recommended for adults—1 microcurie per standard man containing 1000 grams of calcium. The average soil in the United States contains about 10 times more, whereas abroad the radiostrontium content in other areas of the world not subject to the local test fallout is about one-third of that for the United States.

The surface air itself contains radiostrontium due to the fallout from the stratosphere and corresponding to the average time between rainstorms in which it can collect. Filtration of air at sea level discloses radiostrontium on filters if the filters are fine enough, even in periods when bombs are not being tested; thus the only fallout is from the stratosphere reservoir from the high-yield weapons. Measurements in the antarctic on snow samples collected there show that the fallout rate in January and February 1955 was comparable with that observed in the middle latitudes.

CONCLUSION

Finally, although the main part of the radioactivity from high-yield weapons fortunately dissipates in the stratosphere, the small but very significant part that falls out within a few hundred miles of the site of the explosion for weapons fired on the surface constitutes a very real hazard and nothing I have said should be interpreted otherwise. The weapons tests are conducted with great attention to this and the other dangers and every effort made to protect against misadventure. What we have learned from the studies I have described—which by the way have been conducted under the name Project Sunshine—is that these local precautions should be entirely adequate and the worldwide health hazards from the present rate of testing are insignificant.

Reprinted from the Proceedings of the NATIONAL ACADEMY OF SCIENCES.
Vol. 42, No. 6, pp. 365–390. June, 1956.

RADIOACTIVE STRONTIUM FALLOUT

BY DR. W. F. LIBBY

COMMISSIONER, UNITED STATES ATOMIC ENERGY COMMISSION

Communicated May 2, 1956

CONVERSION FACTORS

"Average soil"	= 20 gm Ca/ft^2 in top 2.5 inches
1 MPC unit in any medium	= 1 μc Sr^{90}/kg Ca
	= 2,200 dpm Sr^{90}/gm Ca
1 megaton fission distributed uniformly over entire earth	= 0.0009 MPC unit
	= 0.5 mc Sr^{90}/mi^2

I. EXPERIMENTAL MEASUREMENTS

Strontium 90 is of particular importance among the fission products because of chemical and physical characteristics which result in comparatively high retention in the skeleton. These are chemical similarity to Ca, an element essential to both plants and animals; an average life of about 40 years; and a low rate of elimination from the skeleton. On the basis of studies of the comparative effects of Sr^{90} and Ra^{226} in experimental animals and of the effects of Ra in humans, the generally accepted maximum permissible body burden (skeletal content) of Sr^{90} in adult humans is 1 microcurie. Since the body of the average adult contains about 1,000 gm. of Ca, this is equivalent to saying that the maximum permissible average concentration of Sr^{90} in the adult skeleton is 1 μc/1,000 gm. of Ca. For purposes

of this discussion. this ratio of Sr^{90} to Ca, in whatever medium it may occur, is designated an "MPC unit." One MPC unit of Sr^{90} in the human body is considered to be safe—a significant risk occurring only at much higher dosages.[1] The majority of analyses for Sr^{90} encountered in this work were of the order of a few thousandths of 1 MPC unit. For purposes of orientation, it is helpful to remember that 0.001 MPC unit corresponds to 1/1,000 μc of Sr^{90}/kg of Ca, or 2.2 dpm of Sr^{90}/gm of Ca. The small weights of Sr^{90} involved in both the radiostrontium and normal strontium being considered, and the similarities of the element to Ca, justify the assumption that its distribution in the body will follow that of Ca in a general way.

Two megatons of fission will produce 1 millicurie (mc.) of Sr^{90}/mi^2, if the fission products are uniformly distributed over the earth's surface. If this amount of radioactivity is mixed with the available Ca in the soil, an average of about 20 gm/ft² in the top 2.5 inches of soil, the specific radioactivity produced is 0.0018 MPC unit. It is observed that most of the Sr^{90} fallout is concentrated in the top 1 or 2 inches of soil. For example, in Tables 1 and 2, which show the Sr^{90} burden in the fall of 1953 in the soil of twelve farms in the Wisconsin-Illinois area as well as in the alfalfa and the milk of the cows fed thereon, we note that the top inch of soil contains about 56 per cent and the next 5 inches contain the remaining 44 per cent of the total Sr^{90}. Recently some evidence has been discovered that the radiostrontium finds its way to greater depths.[2] In Table 2 data are given for Iowa soil collected in 1937 which, as expected, shows no Sr^{90}. The average available Ca content of the domestic soils was 8 ± 1 gm Ca/ft²/in., the average fraction of total Ca exchangeable was 68 ± 3 per cent, and the average Sr^{90} content was 4.7 ± 0.4 mc/mi².

TABLE 1

BIOSPHERE Sr^{90} ASSAYS* (WISCONSIN MILKSHED—PRE-CASTLE, OCTOBER, 1953)
(Values Are Given in Terms of 0.001 MPC Units, Except where Noted)

Farm†	Soil 0″–1″	Soil 1″–6″	Total Sr^{90} (Mc/Mi²)	Alfalfa	Milk
Grabow, Wisconsin	26.2	6.7	4.5	12.8	1.7
Oliver Swain, Wisconsin	7.4	2.2	3.1	5.3	1.3
Swanson, Illinois	15.8	2.5	9.2	7.1	1.2
Holcomb, Wisconsin	8.7	1.8	5.1	8.3	1.6
Lewke, Wisconsin	10.2	2.9	3.5	20.9	2.3
Premo, Wisconsin	13.1	2.5	3.8	4.1	0.7
Kurpeski, Illinois	16.3	5.6	4.0	7.4	1.3
Austin, Illinois	22.4	4.7	4.7	5.0	1.8
McKee, Illinois	8.1	0.9	6.3	14.8	1.4
Blomberg, Illinois	1.7	<0.3	4	9.5	1.2
Van Winkle, Illinois	13.8	7.9	3.8	5.0	...
Carver, Illinois	42.1	5.6	3.3	2.3	...
Average	16.8	3.9	...	8.9	1.4
Average Sr^{90} (mc/mi²)	2.6	2.1	4.7 ± 0.4		
Total Sr^{90} (mc/mi²)		4.7 ± 0.4			

* E. A. Martell and W. F. Libby, Project Sunshine Bull. No. 10, January 10, 1955; E. A. Martell, *ibid.*, Suppl. 3, September 1, 1955.
† Samples collected by Dr. Lyle T. Alexander, Chief, Soil Survey Laboratory, Plant Industry Station, Beltsville, Maryland.

As might be expected because of the similarity of the Sr chemistry to that of Ca, milk and cheese show radiostrontium without exception. Figures 1*a*, 1*b*, 1*c*, and 1*d* show the data for both foreign and domestic samples.

TABLE 2*

1953 DOMESTIC PRE-CASTLE SOIL SAMPLES

Location	Lab. No.	Date Sample Taken	Area of Sample (Ft.²)	Depth of Sample (Inches)	Ca Extracted (1 *N* NH_4Ac Reflux) (Gm.)	Weight Sample Extracted (Pounds)	Exchangeable Ca, Analytical Method (Me/100 Gm)	Total Weight Sample Taken (Pounds)	Calc. Exch. Ca in Sample (Gm.)	Calc. Ca. (Gm/Ft²)	Per Cent Ca. Extracted (10th Col.)	Milli-MPC Units	Sr^{90} (Mc/Mi²)	Total Sr^{90} (Mc/Mi²)
Rock Co., Wis., Site No. 1, Knox fine sandy loam, Grabow Farm	531665	9/28/53	1.5	0–1	3.2	10.0	4.8	11.0	4.8	3.2	72.7	26.2	2.3	4.5
Rock Co. Wis. Site No. 1, Knox fine sandy loam	531665	9/28/53	1.5	0–1	1.0	10	4.8	11.0	4.8	3.2	(6 *N* HCl after NH_4Ac extract)	24.6	...	
Rock Co., Wis. Site No. 1, Knox fine sandy loam	531666	9/28/53	0.3	1–6	3.0	10	3.5	11.0	3.5	11.7	93.8	6.7	2.2	
Rock Co., Wis., Swain Farm, Site No. 2, Knox fine sandy loam	531667	9/28/53	1.4	0–1	10.8	9.5	8.5	10.5	8.1	5.8	147	7.4	1.2	3.1
Rock Co., Wis., Swain Farm, Site No. 2, Knox fine sandy loam	531668	9/29/53	0.3	1–6	7.4	9.5	9.8	10.5	9.3	31.0	88.1	2.2	1.9	
Winnebago Co., Illinois, Swanson Farm. Site No. 3, Carrington lib. sil	531669	9/29/53	1.2	0–2	9.5	6.0	13.9	7.0	8.8	7.3	125	15.8	3.2	5.9
Winnebago Co., Illinois, Swanson Farm, Site No. 3, Carrington lib. sil	531670	9/29/53	0.24	1–6	10.0	6.5	13.5	7.5	9.2	38.3	125	2.5	2.7	
Rock Co., Wis., Holcomb Farm, Site No. 4, Carrington sil	531671	9/29/53	1.33	0–1	9.4	6.5	14.9	7.5	10.1	7.6	107	8.7	1.8	3.4
Rock Co., Wis., Holcomb Farm, Site No. 4, Carrington sil	531672	9/29/53	0.36	1–6	11.6	8	14.3	9.0	11.7	32.5	111	1.8	1.6	
Dave Co., Wis., Lewke Farm, Miami sil, Site No. 4	531673	9/30/53	1.2	0–1	5.7	7	7.7	8.0	5.6	4.7	116	10.2	1.3	3.5
Dave Co., Wis., Lewke Farm, Miami sil, Site No. 4	531674	9/30/53	0.28	0–6	7.0	8.5	8.8	9.5	7.6	27.1	103	2.9	2.2	
Columbia Co., Wis., Premo Farm, Miami sil, Site No. 6	531675	9/30/53	1.2	0–1	5.0	7.5	10.2	8.5	7.9	6.6	72.4	13.1	2.4	3.8
Columbia Co., Wis., Premo Farm, Miami sil, Site No. 6	531676	9/30/53	1.2	0–1	2.0	7.5	10.2	8.5	7.9	6.6	...	12.5	...	
Columbia Co., Wis., Premo Farm, Miami sil, Site No. 6	531676	9/30/53	0.30	1–6	8.2	9.0	6.7	10.0	6.1	20.3	149	2.5	1.4	
McHenry Co., Illinois, Miami sil, Kurpeski Farm, Site No. 7	531677	9/30/53	1.16	0–1	5.0	7.0	6.4	8.0	4.6	4.0	122	16.3	1.8	4.0
McHenry Co., Illinois, Miami sil, Kurpeski Farm, Site No. 7	531678	9/30/53	0.30	1–6	5.0	9.0	4.7	10.0	4.3	14.3	132	5.6	2.2	
McHenry Co., Illinois, Miami sil, Austin Farm, Site No. 8	531679	10/1/53	1.3	0–1	4.2	8.0	6.8	9.0	5.6	4.3	85.7	22.4	2.7	4.7
McHenry Co., Illinois, Miami sil, Austin Farm, Site No. 8	531680	10/1/53	0.31	1–6	3.9	9.5	5.1	10.5	4.9	15.8	88.6	4.7	2.0	

TABLE 2—*Continued*

Location	Lab. No.	Date Sample Taken	Area of Sample (Ft.²)	Depth of Sample (Inches)	Ca Extracted (1 N NH_4Ac Reflux) (Gm.)	Weight Sample Extracted (Pounds)	Exchangeable Ca, Analytical Method (Me/100 Gm)	Total Weight Sample Taken (Pounds)	Calc. Exch. Ca in Sample (Gm.)	Calc. Ca. (Gm/Ft²)	Per Cent Ca Extracted (10th Col.)	Milli-MPC Units	Tota Sr^{90} (Mc/Mi²)	
McHenry Co., Illinois, McKee Bros. Farm, Site No. 9, Drummer rich	531681	10/1/53	1.14	0–1	16.0	7.0	25.9	8.0	18.8	16.5	97.0	8.1	3.7	6.3
McHenry Co., Illinois, McKee Bros. Farm, Site No. 9, Drummer rich	531681	10/1/53	1.14	0–1	10.0	7.0	25.9	8.0	18.8	16.5	(6 N HCl after NH_4Ac extract)	4.0	...	
McHenry Co., Illinois, McKee Bros. Farm, Site No. 9, Drummer rich	531682	10/1/53	0.24	1–6	23.5	9.0	27.4	10.0	24.9	103.8	105	0.9	2.6	
McHenry Co., Illinois, Blomberg Farm, Site No. 10, Drummer rich	531683	10/1/53	0.85	0–1	8.0	5.0	25.6	6.0	13.9	16.4	69.0	1.65	(0.8)	
McHenry Co., Illinois, Blomberg Farm, Site No. 10, Drummer rich	531683	10/1/53	0.85	0–1	5.0	5.0	25.6	6.0	13.9	16.4	(6 N HCl extraction following NH_4Ac)	4.4	2.0	
McHenry Co., Illinois, Blomberg Farm, Site No. 10, Drummer rich	531684	10/1/53	0.22	1–6	17.5	8.0	25.4	9.0	20.8	94.5	95.1	0.3	...	
Will Co., Illinois, Van Winkle Farm, Plainfield sand, Site No. 11	531685	10/2/53	1.23	0–1	3.1	9.0	3.5	10.0	3.2	2.6	107	13.8	1.0	3.8
Will Co., Illinois, Van Winkle Farm, Plainfield sand, Site No. 11	531686	10/2/53	0.26	1–6	3.8	10.5	3.2	11.5	3.3	12.7	127	7.9	2.8	
Will Co., Illinois, Carver Farm, Site No. 12, Plainfield sand	531687	10/2/53	1.38	0–1	2.1	9.0	2.8	10	2.5	1.8	91.3	42.1	2.1	3.3
Will Co., Illinois, Carver Farm, Site No. 12, Plainfield sand	531688	10/2/53	0.35	1–6	2.2	11.0	2.4	12	2.6	7.5	91.7	5.6	1.2	
Utah, College Pasture	531689	10/53	1.45	0–1	11.2	7.5	28.0	7.5	19.1	13.2	58.6	1.38	0.51	0.98
Utah, College Pasture	531690	10/53	0.265	1–6	13.2	8.5	28.8	8.5	22.2	83.4	59.4	0.20	0.47	

Average Ca content (exchangeable): 8.0 ± 1 gm Ca/ft²/in = 8.6 ± 1 mg Ca/cm²/in.

Average fraction of total Ca exchangeable = 68 ± 3 per cent (assuming 6 *N* HCl to extract all Ca).

$$\frac{Sr^{90} \text{ concentration, 1–6 inches}}{Sr^{90} \text{ concentration, 0–1 inches}} = 0.23 \pm 0.027.$$

Effective depth at top 1 inch concentration = 2.15 inches = 19 mg Ca/cm² = 18 gm Ca/ft².

Pre-atomic samples:

Iowa, Carrington loam	C-2916	1937	0–3	0 ± 0.00005 MPC units
Iowa, Carrington loam	C-2917	1937	3–13	0 ± 0.00005 MPC units

* All radiostrontium measurements made in Chicago Sunshine Laboratory (see E. A. Martell and W. F. Libby, Project Sunshine Bull. No. 10, January 10, 1955; E. A. Martell, *ibid* Suppl. 1, March 1, 1955; Suppl. 2, June 1, 1955; Suppl. 3, September 1, 1955; and E. A. Martell. Project Sunshine Bulletin 11, December 1, 1955).

The amount of radiostrontium found in humans is shown in Figures 2*a* and 2*b*. The data show that the present Sr^{90} content probably averages somewhat less than 0.001 MPC unit in young people. Apparently a number of barriers protect the human skeleton from this fallout radioactivity.

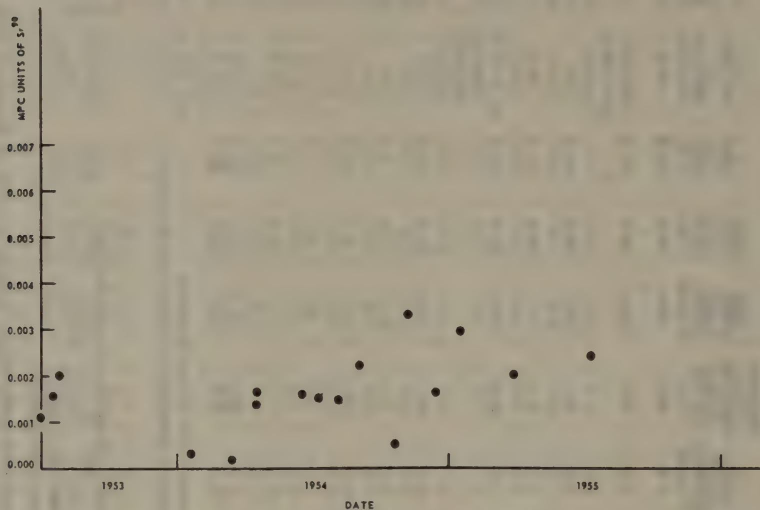


FIG. 1*a*.—Wisconsin cheese—Sr^{90} content

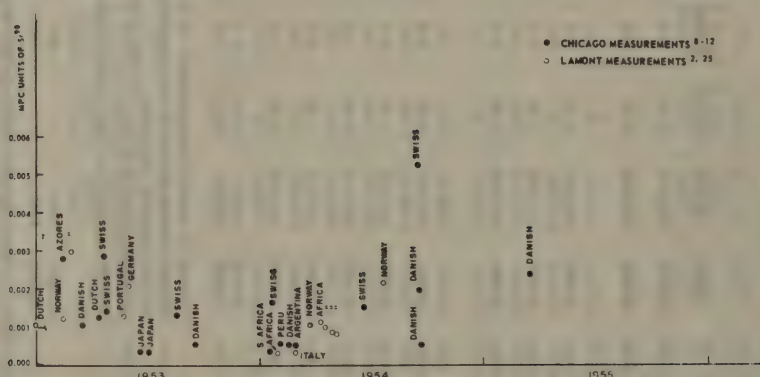


FIG. 1*b*.—Foreign cheese—Sr^{90} content

Measurements have been made on animals, principally cattle and sheep. These data are given in Figures 3*a* and 3*b*. We see here that the contents are much higher than those for milk and human samples, apparently due to selective deposition of Sr in the animal bones, which protects the milk and thus human bone.

Data for foreign soil samples collected just before the Castle test series are presented in Table 3 and Figures 4*a* and 4*b*. From these data we deduce that the band around the earth bounded by the latitudes 60° N. and 10° S. shows a deposition of 0.8 megatons (MT) equivalent of Sr^{90}, in addition to some 0.4 MT which appears to be nearly uniformly deposited, as would have been expected from a slow deposition from a large stratospheric reservoir. No evidence for longitudinal variation is apparent in Figure 4*b*.

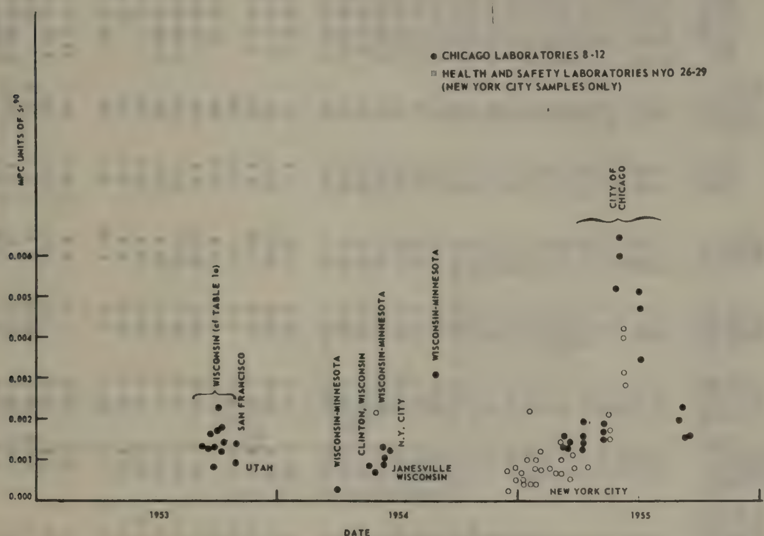


FIG. 1*c*.—U.S. milks—Sr^{90} content

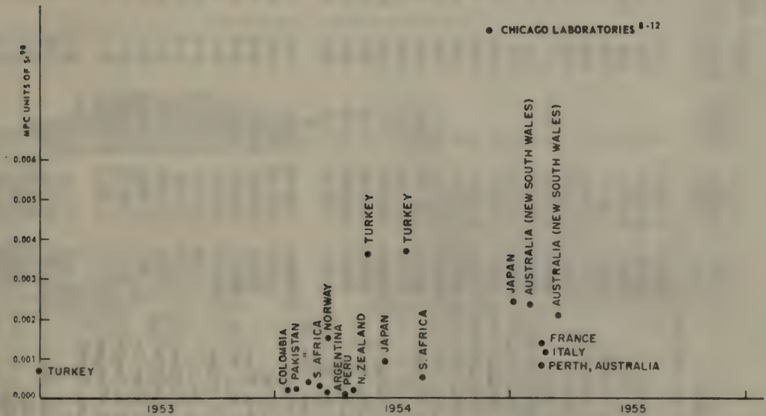


FIG. 1*d*.—Foreign milks—Sr^{90} content

TABLE 3*

Sr^{90} CONTENT AND OTHER PERTINENT DATA ON FOREIGN SOIL SAMPLES COLLECTED BEFORE CASTLE

Lat./Long.	Location	Lab. No.	Date Sample Taken	Area of Sample (Ft²)	Depth of Sample (Inches)	Ca Extracted (1 N NH_4Ac Extraction (Gm.)	Weight Sample Extracted (Pounds)	Exchangeable Ca, Analytical Method (Me/100 Gm.)	Total Weight Sample Taken (Pounds)	Calc. Exch. Ca in Sample (Gm.)	Calc. Exch. Ca (Gm/Ft²)	(Gm/Ft²/In.)	Per Cent Ca Extracted (7th Col.)	Sr^{90} Content (Milli-MPC Units)	Sr^{90} Fallout (Mc/Mi²)
35° S./60° W.	Buenos Aires, Arg. (8 miles apart)	54569	3/15/54	2	0–4	8.7	8	15.0	45.7	62.2	31.1	7.8	80	0.45 ± 0.03	0.39
35° S./60° W.	Buenos Aires, Arg.	54570	3/15/54	2	0–4	6.6	8	10.2	45.7	42.3	21.2	5.3	89	0.44 ± 0.03	0.26
2° N./104° E.	Bin Tong Park, Sing.	54571	3/4/54	2	0–4	0.01	8	0.4	40.6	1.5	0.73	0.13	3	≤ 50	≤1.0
2° N./104° E.	Tengah Air Base, Sing.	54572	3/4/54	2	0–4	0.3	8	0.4	42.5	1.54	0.77	0.19	100	22 ± 2	0.5
30° N./75° E.	Molir Village, Pakistan	54671	2/25/54	2	0–4	4.3	4	18.4	60.5	101	50.5	12.6	64	0.27 ± 0.03	0.38
30° N./75° E.	Hub River, Pakistan	54672	2/25/54	2	0–4	4.0	4	16.6	65.3	98.4	49.2	12.3	66	0.35 ± 0.04	0.48
52° N./0°	Rothamstead, Eng.	54675	4/?/54	7 (est.)	0–3	4.5	4	13.5	95.8	117.4	16.8	5.6	92	1.31 ± 0.07	0.61
5° N./75° W.	Bogotá, Colombia (plowed)	54715	3/?/54	2	0–4	4.3	4	13.8	29.9	37.5	18.7	4.7	86	0.67 ± 0.04	0.35
5° N./75° W.	Bogotá, Colombia (grass)	54716	3/?/54	2	0–4	4.8	4	13.9	51.0	64.4	32.2	8.1	95	0.91 ± 0.08	0.82
5° S./30° E.	Leopoldville, Belgian Congo	54717	3/?/54	2	0–4	2.4	4	4.6	51.1	21.3	10.7	2.7	All	0.92 ± 0.08	0.27
5° S./30° E.	Leopoldville, Belgian Congo	54718	3/?/54	2	0–4	4.5	4	13.2	69.4	83.2	41.6	10.4	94	0.21 ± 0.04	0.24
12° N./45° E.	Aden Protectorate, SW Arabia	54421	3/?/54	2	0–4	9.6	8	23.7	32.0	68.9	34.4	8.6	56	0.51 ± 0.03	0.49
12° N./45° E.	Aden Protectorate, SW Arabia	54422	3/?/54	2	0–4	9.4	8	19.7	30.0	53.7	26.8	6.7	66	1.05 ± 0.10	0.78
60° N./10° E.	Oslo, Norway (Walsh)	54412	4/27/54	2.21	0–2	9.8	8	17.0	16.2	25.0	11.3	5.7	79	1.51 ± 0.05	0.48
60° N./10° E.	Oslo, Norway, No. 1	54410	4/27/54	2.21	0–2	11.0	8	18.0	16.0	26.2	11.9	5.9	84	1.44 ± 0.06	0.48
32° N./36° E.	Beka'a Valley, Lebanon	54293	2/25/54	1.5	0–3	20.2	7	41.5	17.0	64.1	42.7	14.2	77	0.86 ± 0.05	1.0
35° N./2° E.	Boghari, Algeria, No. 3	54359	2/22/54	2.21	0–2	13.2	8	29.8	19.0	51.4	23.3	11.6	61	3.4 ± 0.08	2.2
35° N./2° E.	Boghari, Algeria, No. 1	54360	2/22/54	2.76	0–2	13.7	8	34.3	19.4	60.4	21.9	10.9	55	3.8 ± 0.1	2.3
35° N./2° E.	Boghari, Algeria, No. 2	54361	2/22/54	2.21	0–2	10.0	8	25.8	18.3	42.5	19.4	9.7	53	3.46 ± 0.08	1.9
35° N./40° E.	Village 41 km. SE. of Damascus, Syria	54295	2/26/54	2.21	0–2	12.4	7	27.1	14.4	35.4	15.6	7.8	72	1.9 ± 0.08	0.82
35° N./40° E.	Tel Muskan, Syria	54296	2/26/54	2.21	0–2	18.6	7	29.6	20.0	53.8	24.3	12.1	99	1.1 ± 0.10	0.75
52° N./0°	Ffostill, Breton, Wales	54415	4/?/54	2.78	0–2	8.0	8	11.4	11.4	11.8	4.27	2.1	97	3.3 ± 0.12	0.39
52° N./0°	Montgomery, B.I., Wales	54416	4/?/54	2.78	0–2	9.6	8	11.6	8.3	8.7	3.1	1.6	All	...	...
52° N./5° W.	Cardigan, B.I., Wales	54417	4/?/54	2.78	0–2	0.3	5	1.3	10.2	1.2	0.4	0.2	50	97 ± 9.1	1.1
52° N./0°	Suffolk, Eng., No. 4	54418	4/?/54	1.74	0–2	15.2	7	37.2	16.6	56.1	32.2	16.1	64	1.37 ± 0.06	1.2
52° N./0°	Suffolk, Eng., Pudding Crnr.	54419	4/?/54	1.74	0–2	18.1	8	38.0	16.8	58.0	33.3	16.7	66	0.89 ± 0.05	0.83
52° N./0°	Suffolk, Eng., Old Orchard	54420	4/?/54	1.74	0–2	16.6	8	36.2	17.9	58.8	33.8	16.9	63	0.94 ± 0.09	0.89
45° S./170° E.	New Zealand, Hutt Co.	531804	11/?/53	5 (est.)	0–3	7.9	8.5	13.0	44.0	51.9	10.4	3.5	79	≤ 0.21	≤0.06
45° S./170° E.	New Zealand, S. Canterbury	5485	1/18/54	5 (est.)	0–3	6.5	9	10.8	47.2	46.3	9.3	3.1	74	≤ 0.18	≤0.05
45° S./170° E.	New Zealand, Whangarei Co.	5471	1/18/54	5 (est.)	0–3	0.9	8	1.7	37.7	5.8	1.20	0.4	75	2.51 ± 0.09	0.084
20° S./45° W.	Belo Horizonte, Brazil (3 km. apart)	54288	3/?/54	2	0–4	0.2	10	0.4	42.0	1.5	0.8	0.2	50	13.5 ± 1.25	0.30
20° S./45° W.	Belo Horizonte, Brazil	54289	3/?/54	2	0–4	0.6	8	0.7	26.5	1.7	0.9	0.2	All	4.17 ± 0.3	0.10
35° S./75° W.	Santiago, Chile	5472	12/?/53	30	0–1	10.2	8	27.2	50.4	124.5	4.15	4.1	51.5	≤ 0.33	≤0.04
30° S./30° E.	Natal, S. Africa	54399	2/?/54	2	0–4	2.6	8	4.2	29.5	11.2	5.6	1.4	86.7	0.49 ± 0.08	0.076
30° S./30° E.	Natal, S. Africa, 3 mi. SE.	54400	2/?/54	2	0–4	1.7	8	1.2	47.0	5.1	2.55	0.6	...	9.80 ± 0.71	0.70
15° N./120° E.	Philippine Islands	54401	2/26/54	2	0–4	14.8	8	18.6	48.4	81.7	40.8	10.2	All	1.47 ± 0.21	1.67
15° N./120° E.	Philippine Islands	54402	2/26/54	2	0–4	3.5	8	5.6	51.7	26.3	13.1	3.3	85.4	20.1 ± 2.3	7.3

* See note to Table 2.

Average: 8.1 ± 0.8 gm Ca/ft²/in = 8.8 ± 0.9 mg Ca/cm²/in.

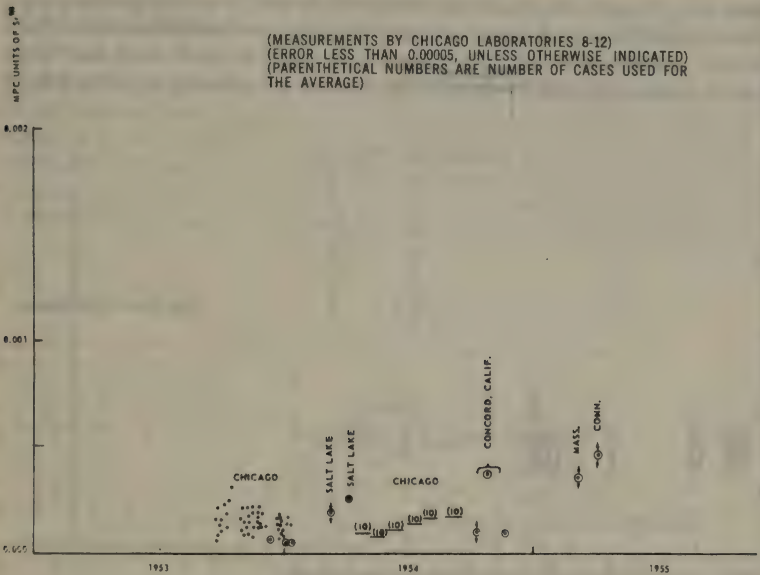


Fig. 2*a*.—Human stillborns

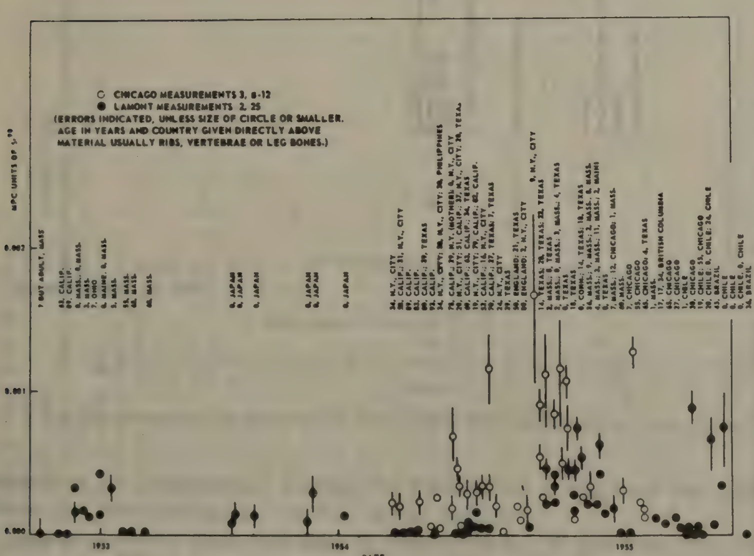


Fig. 2*b*.—Human bone—Sr^{90} content

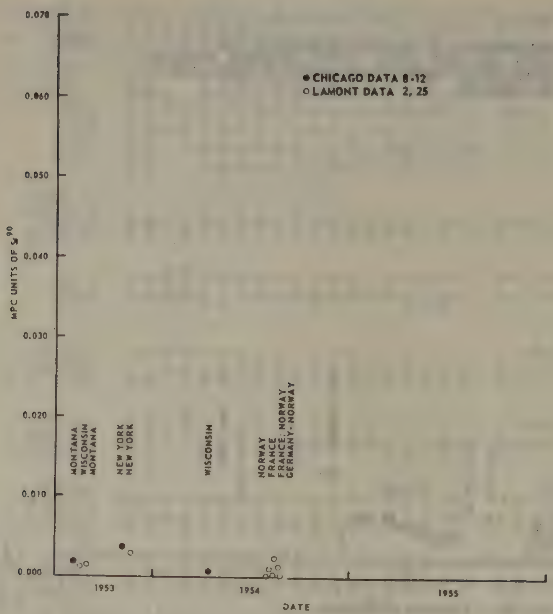


FIG. 3a.—Calf bones

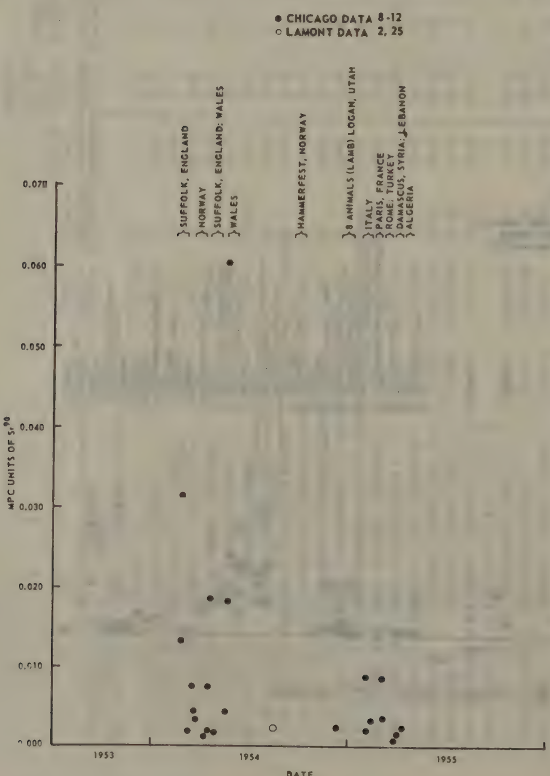


FIG. 3b.—Sheep bones

The actual heights of rise of the bomb clouds are the basis for the assumption that all distant fallout from megaton weapons occurs from a stratospheric reservoir, while that from those of lower yield occurs from the troposphere. Actually, the height of the tropopause varies with the season, so that the season must be con-

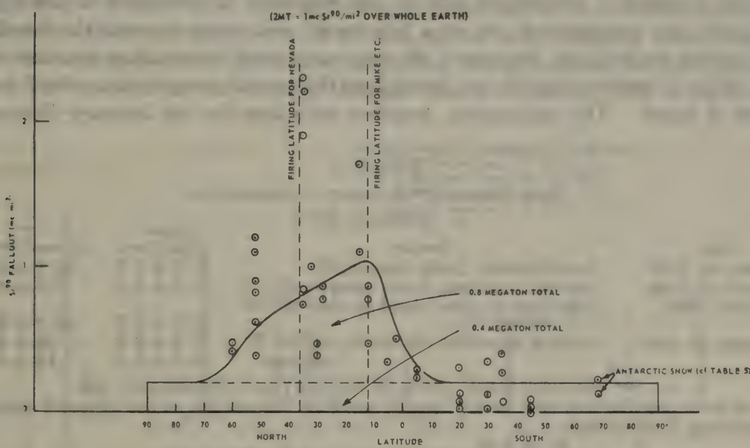


FIG. 4*a*.—Latitudinal distribution of foreign pre-Castle Sr^{90} fallout (soil assays)

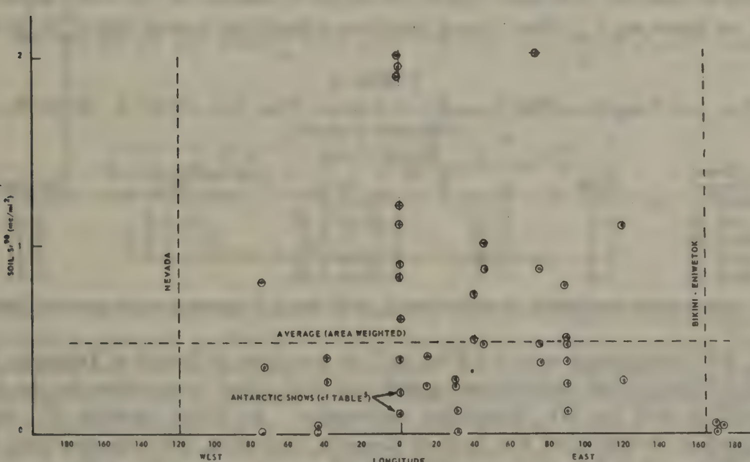


FIG. 4*b*.—Longitudinal distribution of pre-Castle Sr^{90} soil data from foreign samples.

sidered in the assignment. During the Pacific tests it has been near 55,000 ft.; hence our classification has validity in this respect.

Two of the most important data in Figures 4*a* and 4*b* are those from the antarctic series. The samples were snow cores, collected for the Chicago Sunshine

Laboratory and for cosmic-ray tritium analysis,[3–5] by Mr. Paul Humphrey of the United States Weather Bureau in January and February, 1955, at Admiral Byrd Bay (69°34′ S.; 00°41′ W.), at Atka Bay (70°35′ S.; 08°06′ W.), and at Little America III. The data are given in Tables 4 and 5. Table 4, Part A, gives the Sr^{90} and T contents of surface snow at four locations. From the T concentrations and the expected T production rate in this region[3–5] (T produced in the Castle test itself was precipitated out in a few weeks and never entered the Southern Hemisphere appreciably, because of the large amount of water taken into the cloud with which it became mixed[6]), we determined the annual precipitation to be 7.8 ± 2 inches of water. This calculation, together with that for the January–February,

TABLE 4

POST-CASTLE Sr^{90} FALLOUT IN ANTARCTICA

A. SURFACE SNOW

Date	Location	Sr^{90} (dpm/Liter)	T (Atoms/10^{18} H's)
January 15, 1955	Near Quonset, Little America III	3.2 ± 0.3	14.1 ± 0.6
January 17, 1955	One-half mile east, Little America III	3.1 ± 0.7	7.5 ± 0.6
February, 1955	Atka Bay, 6 miles inland on shelf (70°35′ S.; 08°06′ W.)	5.3 ± 0.5	19.2 ± 0.8
February 19, 1955	Admiral Byrd Bay (69°34′ S.; 00°41′ W.)	2.0 ± 0.2	24 ± 5
	Average	3.4 ± 0.5	14 ± 3

B. Sr^{90} PRECIPITATION RATE IN JANUARY AND FEBRUARY, 1955

Annual snow precipitation rate from T assay* and 0.59 T's/cm²/sec as the expected antarctic cosmic-ray T production.

$$\rho = \frac{4.7 \times 0.59}{14} \text{ meters/yr} = 7.8 \pm 2 \text{ inches of water.}$$

Sr^{90} rate of precipitation:

3.4 ± 0.5 dpm/liter = 62 dpm/ft²/yr for 7.8 inches of water/yr = 0.8 ± 0.2 mc/mi²/yr.

* H. von Buttlar and W. F. Libby, "Natural Distribution of Cosmic Ray Produced Tritium. II," *J. Inorg. and Nuclear Chem.*, **1**, 75, 1955; L. A. Currie, W. F. Libby, and R. Wolfgang, *Phys. Rev.*, **101**, 1557, 1956.

TABLE 5

PRE- AND POST-CASTLE Sr^{90} FALLOUT AT ADMIRAL BYRD BAY (69°34′ S.; 00°41′ W.)
(Collected 2/19/55)

SNOW CORE

Age (Years) (7.8 Inches Water/Yr)	Depth (Ft.)	Density	Sr^{90} (dpm/Liter)	T (Atoms/10^{18} H's)	Sr^{90} Rate* (Mc/Mi²/Yr)
0–0.54	0–1	0.35	1.95 ± 0.20	24 ± 5	0.46
0.54–1.04	1–2	0.32	1.7 ± 0.2	12.5 ± 0.8	0.40
1.04–1.52	2–3	0.30	0.48 ± 0.04	13.5 ± 0.7	0.11
1.52–2.16	3–4	0.41	0.90 ± 0.06	...	0.21

* Assumed annual precipitation, 7.8 inches water/yr, on the basis of T contents of surface waters (cf. Table 4).

1955, Sr^{90} precipitation rate of 0.8 mc/mi²/yr, are given in Part B of Table 4. The result for the annual precipitation agrees well with direct observation by the Atka Expedition and by Mr. Humphrey personally.[7] At Little America IV, direct observation showed that the floor of the tent projecting from the ice front was beneath only 7 or 8 feet of snow after roughly seven years. Since the density of the snow was about 0.35, this would correspond to about 5 inches of annual precipitation. Other observations[7] checked this general magnitude. In Table 5 a core taken at Admiral Byrd Bay was measured for both Sr^{90} and T. From these data we observe the pre-Castle fallout rates of 0.11 and 0.21 Sr^{90} mc/mi²/yr. The surface rate at this site is 0.43, which is less than the general average in the area for January and February, 1955, of 0.8 mc/mi²/yr—as shown in Table 4—hence it

may be that the pre-Castle values at this site are low also and should be increased by the ratio 0.8/0.43, or by 90 per cent, to 0.2 and 0.4, respectively.

The average Sr^{90} content of rain and snow in the Chicago area since the fall of 1952 was calculated by weighting each datum by the total rainfall observed in the particular storm. The data on the Sr^{90} content of Chicago rain are given in Figure 5.[8-12] It is clear that large fluctuations can occur in individual storms. However, these extremes were, in general, of low total rainfall; hence the effect on the average is small. It is interesting that the antarctic snows have about the same Sr^{90} content as the average Chicago rain of Figure 5. We recall that pre-

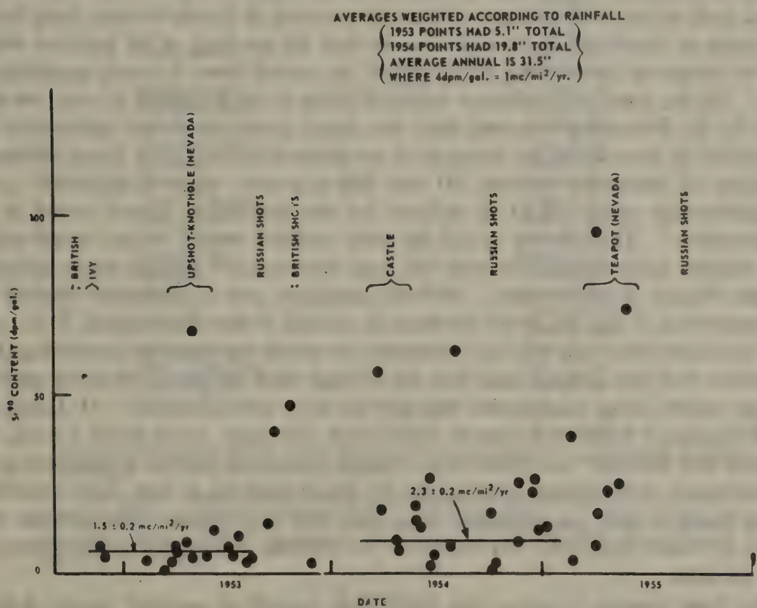


FIG. 5.—Sr^{90} content of Chicago rains

cipitation there is only one-fourth to one-fifth that in Chicago. According to the mechanism espoused in this paper, these fluctuations are to be expected, because the fallout from the stratosphere is thought to be steady and continuing, and the washing-out by rain is expected to carry down the fallout accumulated since the last precipitation from the particular air mass involved. Many of the samples given in Figure 5 and in Project Sunshine Bulletins Nos. 10 and 11[8-12] have been measured for T as well;[3-5] hence further correlations of the type described above for antarctic snow (Tables 4 and 5) can be made.

In Tables 1 and 2 the Sr^{90} contents of soils in the midwest region of the United States were shown to have an assay of 4.7 mc/mi² in October, 1953. The total from rains in the preceding year was only 1.5 mc/mi², according to Figure 5; hence we have to expect about 3 mc/mi² to have been deposited prior to Operation Ivy by direct fallout, most reasonably from tropospheric debris. The total fired in

Nevada prior to this time which would not have fallen out in the immediate vicinity of the test site was approximately 200 KT for the Operations Tumbler-Snapper and Buster-Jangle together. If this were all deposited in the United States, it would amount to about 7.0 mc/mi^2. The test series Upshot-Knothole, with approximately 220–230 KT of distant fallout, and Teapot, with approximately 160–180 KT, should have added correspondingly to the 4.7 mc/mi^2 in October, 1953, and to the 3.0 mc/mi^2 of stratospheric fallout for the intervening period, for a total of perhaps 12 mc/mi^2 expected in the spring of 1956 in the United States.

The efficiency with which rain removes fallout from the air through which it passes is probably high. One knows, on simple physical grounds, that as little as 0.1 inch of rain will traverse at least 90 per cent of the air volume lying below the layer in which the rain originates, so that 90 per cent of all particles which can be swept up by a falling raindrop will be carried down by such quantities of rain. On the point as to whether fallout is likely to be deposited by rain, we note that G. H. Wilkening[13] showed that the decay products of the radioactive gas Rn which in themselves are isotopes of nongaseous elements are found affixed to particles of diameters between 20 and 800 angstrom units (0.002–0.080 μ)—a submicroscopic range not at all unlikely for the radioactive fallout stored in the stratosphere.[1] The velocity of fall for such particles would be very small and in this respect quite compatible with the long stratospheric storage times indicated by the Project Sunshine data. Blifford, Lockhart, and Rosenstock[14] studied the concentration of the Rn decay products in rainfall in the Washington, D.C., area and concluded that rain was the mechanism by which the particles containing these products were precipitated and that the average time the decay products spent in the air before being precipitated was only 15 days, approximately. O. Haxel and G. Schumann,[15] found this time in Heidelberg, Germany, to be about 4 days, and Damon and Kuroda[16] concluded that Blifford *et al.*, were correct in attributing the precipitation of the aerosol carrying the Rn decay products to rain, their conclusion being based in part on additional data that they had taken. The average time spent by water in the air was found by von Buttlar and Libby[4] to be between 5 and 14 days.

For these reasons it seems very likely that rainfall or snowfall carries down a major part of the fallout which comes from the stratosphere and probably is a very important mechanism for that part of the tropospheric fallout material which does not fall out in the first few hours or day or two after the detonations. Of course, the whole question can be settled by direct experiment in which a correlation between rainfall and total fallout is sought. The present data seem to favor the hypothesis. This conclusion and prediction seem to be borne out by Table 6, which presents the total Sr^{90} content of the top 2 inches of typical United States soils collected in October, 1955, and leached at room temperature for 30 minutes with 6 N HCl.

Table 7 shows data obtained in the Chicago laboratories on the Sr^{90} contents of rivers and lakes. It is clear that these are much lower than those of the rain from which they are derived. For example, from Figure 5 we should estimate that the average rain in the Chicago area in the summer of 1953 had a Sr^{90} content of about 7 dpm/gal. From Table 1 for the domestic pre-Castle soil contents and from Figures 4*a* and 4*b* for foreign pre-Castle soil contents, we estimate that the European

rains averaged about 3 dpm/gal. The four rivers, Mississippi, Mosel, Seine, and Danube, show less than 5 per cent of this; hence we conclude that the Sr^{90} in rain is removed by the soil before the water runs off to the rivers and lakes. This fact agrees, of course, with the sharp localization of the Sr^{90} in the top 2 inches of soil (cf. Tables 1, 2, and 3).

TABLE 6*

Sr^{90} FALLOUT ACCUMULATION IN TOP SOIL (0–2 INCHES) IN U.S. IN 1955
(Sampled September 23–October 20, 1955)

Station	Measured in Soil (dpm/Ft²)
La Guardia	310†
	350 ± 21
	550 ± 16
Binghamton	710 ± 16
Philadelphia	450 ± 19
Rochester	550 ± 20
Jacksonville	470 ± 20
Atlanta	530 ± 12
Detroit	640 ± 21
New Orleans	470 ± 14
Memphis	900 ± 20
Des Moines	540 ± 13
Rapid City	1070 ± 21
Seattle	400 ± 15
Boise	1160 ± 23
Albuquerque	270 ± 14
	290 ± 20
Grand Junction	280 ± 14
Salt Lake City	860 ± 18
Los Angeles	120 ± 12
Average	578
or	7.3 mc/mi²

(Probable additional Sr^{90} in lower layers and to be released by additional leaching probably will raise this about twofold.)

* Data by E. P. Hardy and R. S. Morse, of the Health and Safety Laboratory, New York Operations Office, personal communication.

† This datum was obtained by Dr. J. L. Kulp, Lamont Geological Observatory, Columbia University. The procedure was different from that of the New York Operations Office (personal communication).

TABLE 7

Sr^{90} CONTENT OF RIVER AND LAKE WATERS*

Location	Sr^{90} Content (dpm/Gal)	Location	Sr^{90} Content (dpm/Gal)
Lake Michigan, October 27, 1953	0.39 ± 0.08	Mosel River, Metz, September 7, 1953	0 ± 0.05
Mississippi River, Memphis, February 4, 1953	1.13 ± 0.16	Seine River, Nogent, September 8, 1953	0 ± 0.09
Mississippi River, St. Louis, April 17, 1953	<0.77 ± 0.18	Danube River, Ulm, September 12, 1953	0 ± 0.07

* See note to Table 2.

Examination of the data in Tables 2 and 3 on the Sr^{90} content of the exchangeable calcium in soils shows that there is a strong tendency for the lowest activity of Sr^{90} per unit weight of exchangeable Ca to occur in Ca-rich soils and vice versa, as would be expected, of course, if the fallout rate were uniform. This situation is displayed graphically for the pre-Castle soil data in Figure 6.

For years the Health and Safety Laboratory of the New York Operations Office of the Atomic Energy Commission (Mr. Merril Eisenbud, manager), with the co-

operation of the United States Weather Bureau, has been collecting fallout data[17] by use of gummed papers of 1-ft.^{2} area which are laid flat for a certain time out in the open away from buildings. After the exposure, the paper is folded and mailed to the New York Operations Office in an ordinary envelope. Samples thus can be collected cheaply, easily, and quickly from any populated area anywhere the postal service reaches. Most of the data so obtained have dealt with total fallout rather than with Sr^{90} specifically, but many analyses for Sr^{90} have been made since Operation Castle. These are presented later.

FIG. 6.—Top soil Sr^{90} concentration versus calcium content

The main question about the gummed-paper technique is its over-all efficiency of collection. In order to determine this, the Health and Safety Laboratory has conducted an extensive series of comparisons of the amounts of fallout by gummed paper and a 12-gallon pot with an 18-inch vertical cylindrical wall placed immediately beside the paper. Some of the data thus obtained are given in Table 8. From them we deduce a collection efficiency of 69 ± 9 per cent (but we use 63 per cent, since Mr. Eisenbud recommends this on the basis of more data and a better statistical treatment).

The data thus obtained for the post-Castle Sr^{90} fallout rate in the United States and South America are given in Table 9. These are combined with those for other areas, to give the world Sr^{90} fallout rates for September, October, November, and

TABLE 8

GUMMED PAPER COLLECTION EFFICIENCY
(Relative to 12-Gallon Pot [18-Inch Vertical Wall, 12-Inch Diameter; Cylindrical])

Month	Gummed Paper (dpm/Ft²/Mo)	Pot (dpm/Ft²/Mo)	Efficiency (Per Cent)	Reference
Mar., 1954	11.6	14	84	*
Apr., 1954	15.2	31	49	*
May, 1954	21.6	34	63	*
June, 1954	10.7	9.2	116	*
July, 1954	17.6	25	70	*
Aug., 1954	13.5	7.7	176	*
Sept., 1954	20.7	92	22	*
Oct., 1954	3.1	11	29	*
Nov., 1954	5.7	32	18	*
Jan., 1955	9.0	9.9	92	†
Feb., 1955	29.8	50.6	58	‡
Mar., 1955	210	150	140	‡
Apr., 1955	44.9	79.5	57	§
May, 1955	18.6	56.4	33	‖
June, 1955	12.4	51.7	24	‖
		Average	69 ± 9#	

* Interim Sunshine Report, NYOO Report NYO-4620, January 17, 1955.
† Sunshine Report for January and February, NYOO Report NYO-4643, April 21, 1955.
‡ Sunshine Report for March and April, NYOO Report NYO-4646, May 30, 1955.
§ Sunshine Report for May and June, NYOO Report NYO-4653, July 5, 1955.
‖ NYOO Report NYO-4623, January 18, 1955.
\# The figure of 63 per cent will be used for consistency. Mr. Merril Eisenbud (NYOO) recommends this on the basis of more data and a better statistical treatment.

TABLE 9

POST-CASTLE FALLOUT IN U.S. FROM GUMMED PAPERS
(Taken at 63 Per Cent Efficiency [cf. Table 8])

Month	Location	Sr^{90} Fallout Rate (Mc/Mi²/Yr)	Reference
Sept., 1954	Eastern U.S. (10 stations)	2.5 ± 0.2	*
Oct., 1954	Eastern U.S. (10 stations)	2.0 ± 0.4	*
Nov., 1954	Eastern U.S. (10 stations)	2.0 ± 0.4	*
Dec., 1954	Eastern U.S. (10 stations)	1.4 ± 0.2	†
Jan., 1955	Eastern U.S. (9 stations)	1.3 ± 0.2	†
Sept., 1954	Western U.S. (20 stations)	0.9 ± 0.2	‡
Sept., 1954	U.S. (38 stations)	0.92 ± 0.2	§
Oct., 1954	U.S. (38 stations)	0.79 ± 0.2	§
Nov., 1954	U.S. (38 stations)	0.95 ± 0.2	§
Dec., 1954	U.S. (37 stations)	0.71 ± 0.2	‖
Sept., 1954	South America (12 stations)	2.1 ± 0.2	‡
Oct., 1954	South America (12 stations)	1.6 ± 0.2	‡
Nov., 1954	South America (12 stations)	2.4 ± 0.2	‡

* Sunshine Report for March and April, NYOO Report NYO-4646, May 30, 1955.
† Sunshine Report for January and February, NYOO Report NYO-4643, April 21, 1955.
‡ Sunshine Report for May and June, NYOO Report NYO-4653, July 5, 1955.
§ Sunshine Report, August 1954, NYOO, HASL-S-2 (NYO-4656), November 12, 1954.
‖ Sunshine Report for July and August, NYOO Report NYO-4661, September 16, 1955.

December, 1954, presented in Table 10 and Figure 7. From these data we obtained these extremely important conclusions:

1. A Sr^{90} fallout probably derived from megaton weapons and nearly uniform over the world, except for local effects due to rainfall variations and to fallout from

submegaton weapons, seems clearly established. The fallout from the kiloton weapons lasts only a few weeks at most, since they involve only tropospheric

TABLE 10

World-Wide Sr^{90} Fallout Rate from Gummed Papers*
(Taken at 63 Per Cent Efficiency [cf. Table 8]; Mc/Mi²/Yr)

Area	Per Cent Total Earth's Area	No. Stations (Sept.)	Sept., 1954	Oct., 1954	Nov., 1954	Dec., 1954	Average
Arctic	6.5	5	1.2 ± 0.4	3.0 ± 0.4	1.4 ± 0.4	0.9 ± 0.4	1.6 ± 0.2
North Temperate	10.9	14	1.7 ± 0.24	0.68 ± 0.24	1.4 ± 0.24	0.6 ± 0.24	1.1 ± 0.12
Pacific	8.0	2	0 ± 0.6	0.6 ± 0.6	1.9 ± 0.6	1.2 ± 0.6	0.9 ± 0.3
U.S.	1.5	39	1.3 ± 1.4	0.9 ± 0.14	1.4 ± 0.14	0.9 ± 0.14	1.1 ± 0.07
North Tropic	18.5	8	0.7 ± 0.3	1.5 ± 0.3	0.7 ± 0.3	0.4 ± 0.3	0.8 ± 0.16
South Tropic	25.5	9	3.2 ± 0.3	2.4 ± 0.3	2.5 ± 0.3	0.6 ± 0.3	2.1 ± 0.15
				Average (area weighted)			1.5 ± 0.1

Area	Jan., 1955	Feb., 1955	Mar., 1955	Apr., 1955	May, 1955	June, 1955	July, 1955	Aug., 1955	Average
Arctic	0.32	1.2	0.52	0.62	2.0	1.5	1.6	0.82	1.07
North Temperate	1.0	0.55	1.1	1.1	2.9	1.9	2.5	1.4	1.57
Pacific (21 stations)	0.53	0.58	2.2	1.1	1.6	1.3	1.2	1.0	1.19
U.S.	0.86	0.86	1.4	2.3	4.0	2.8	2.9	1.4	2.07
North Tropic	0.71	0.70	1.2	0.78	1.5	0.80	0.87	0.50	0.88
South Tropic	1.5	1.3	0.83	0.40	1.0	1.5	0.60	0.72	0.98
South Temperate (4 stations)	1.1	0.46	0.9	0.40	0.38	1.9	1.1	1.4	0.74
Average (area weighted, except U.S. and North Temperate omitted because of Teapot)									0.95 ± 0.1

* Sunshine Report for July and August, NYOO Report NYO-4661, September 16, 1955, and personal communication from Dr. E. C. Plesset, Rand Corporation.

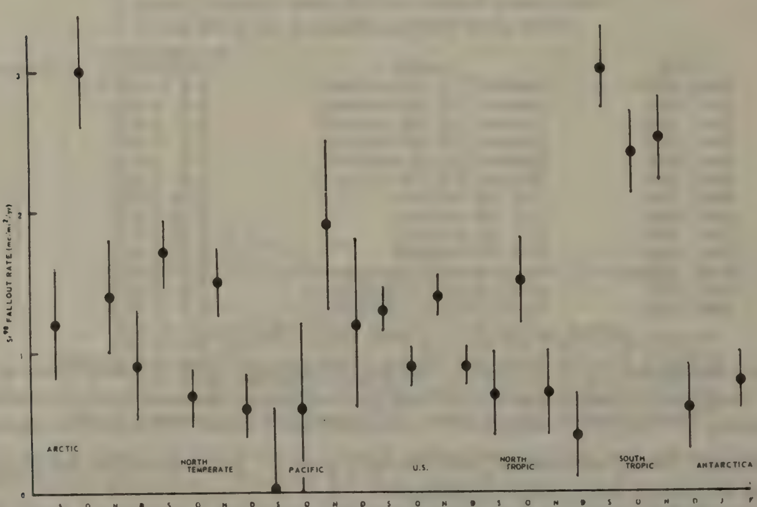


Fig. 7.—World-wide Sr^{90} fallout rates, September–December, 1954, (gummed paper at 63 per cent efficiency except antarctic value, which was snow).

storage, but widespread fallout is found to occur at least 1.7 years after a megaton test series.

2. This average world-wide Sr^{90} fallout rate in the fall of 1954 and the spring and summer of 1955 was 1.2 mc/mi²/yr (see Table 10).

Additional information available on the Sr^{90} distribution has been obtained by air filters operated at sea level, principally by the Naval Research Laboratory, measured in the Chicago Sunshine Laboratory, and by the Health and Safety Laboratory.[18] The surface samples collected at Washington, D.C., by the Naval Research Laboratory and measured at Chicago[8–12] are given in Table 11 and

TABLE 11

Air-Filter Data, Washington, D.C.
(Army Chemical Corps Type 5 Paper, 99 Per Cent Efficient Down to Few Tenths Micron; 75 Per Cent Efficient Down to 0.01 Micron)

Washington, D.C.	Sr^{90} (dpm/10^6 Ft³)	Equivalent* Fallout (Mc/Mi²/Yr)
April 5–8, 1953	18.6 ± 0.7	0.14
October 2–6, 1953	41.1 ± 3.0	0.3
October 6–9, 1953	30.5 ± 1.1	0.2
October 12–15, 1953	70.4 ± 12	0.5
April 3–5, 1954	91.0 ± 7	0.7
April 8–10, 1954	6.4 ± 0.2	0.05
April 10–12, 1954	258 ± 6	1.9
April 12–14, 1954	65.5 ± 4.6	0.5
April 15–17, 1954	11 ± 0.5	0.08
April 17–19, 1954	21 ± 0.6	0.16
April 29–May 1, 1954	32.2 ± 2.6	0.2
May 11–13, 1954	31.3 ± 2.2	0.2
May 24–26, 1954	216 ± 11	1.6
June 1–3, 1954	68.3 ± 4	0.5
July 16–17, 1954	73.5 ± 5.2	0.5
July 26–29, 1954	48 ± 3.9	0.36
November 1–3, 1954	120 ± 7	0.9
December 1–2, 1954	103 ± 4	0.8
January 3–4, 1955	281 ± 6	2.1
February 5–6, 1955	127 ± 5	0.9
February 10–12, 1955	241 ± 10	1.8
February 22–23, 1955	202 ± 11	1.5
March 3–4, 1955	270 ± 13	2.0
March 7–8, 1955	394 ± 20	2.9
March 13–14, 1955	267 ± 16	2.0
March 16–17, 1955	310 ± 15	2.3
March 22–23, 1955	393 ± 20	2.9

* 134 dpm/10^6 ft³ = 1 mc/mi²/yr (cf. text); (28300 ft³/ft² = 10^6 ft³/35.5 ft²).

Figure 8. In order to correlate these data with the fallout rate, we recall that, as remarked previously, any rain of 0.1 inch or more probably will thoroughly wash down the fallout in the air below the layer at which the rain originates. Examination of the weather data for the Washington, D.C., area in the period when the samples were taken shows that the average interval between rains was 6 ± 3 days. Therefore, the Sr^{90} content of surface air should correspond to fallout for this time on the average, and we would expect a fallout rate R (mc/mi²/yr) to correspond to a surface-air content of $R \times \frac{6}{365} \times 79 \times 41 \times 2.5$ dpm/10^6 ft³, since 79 dpm/ft² is equivalent to 1 mc/mi² and there are 41 ft²/10^6 ft³ below the tropopause on the average at Washington, D.C., and 2.5 is the average ratio of height of tropopause to rain-bearing layers. The resulting rate of 0.70 ± 0.2

mc/mi²/yr is definitely low, compared to the rain result of 2.3 ± 0.2 mc/mi²/yr for Chicago (Fig. 5) and of 1.5 ± 0.1 mc/mi²/yr for data involving gummed paper (Fig. 7 and Table 9). The uncertainty in the factor of 2.5 for the ratio of rain-bearing layer height is probably the principal uncertainty in the calculation of the fallout rate from air-filter data, though it may be that the perfect vertical mixing of the lower 40 per cent of the troposphere implicit in the calculation is an incorrect assumption, in that air right at the surface is cleaned to a considerable extent by surface contact with vegetation and water and soil and therefore has less fallout than the average for the lower troposphere.

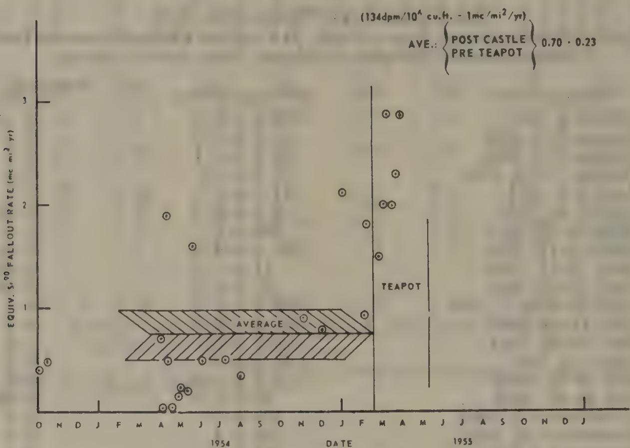


FIG. 8.—Sr^{90} in air, Washington, D.C.

From the data given on rates of fallout, we calculate the average stratospheric residence time, $\tau = 10 \pm 5$ years. The high-altitude data show a definite rise above the tropopause. This is strong confirmation for the stratospheric storage and dissemination mechanism.

II. DISCUSSION

A. PREDICTED Sr^{90} FALLOUT

The stratosphere reservoir of Sr^{90} immediately after Operation Castle had been completed was about 12 mc/mi². The fallout rate of Sr^{90} corresponds to an average storage time of 10 ± 5 years and essentially uniform world-wide dissemination. The radioactive half-life of Sr^{90} is 28 years, corresponding to an average life of 40 years. Therefore, the Sr^{90} fallout rate from tests up to and through the Castle series should be given by

$$R = \left(\frac{1}{10}\right) 12e^{-t(1/10+1/40)} \text{ mc/mi}^2\text{/yr}, \qquad (1)$$

where t is the time elapsed, in years, since May, 1954. This relation is given in Figure 9. From it, we can predict the tropospheric air content to be

$$A = \frac{RT \times 2.5 \times 79 \times 41}{365} \text{ dpm}/10^6 \text{ ft}^3, \tag{2}$$

where a period of T days elapses between the rains washing out the air mass concerned. Taking T to be 6 days on the average, the surface air should contain 134 R dpm/10^6 ft^3 in the middle latitudes.

The rainfall content will be 4 R dpm/gal for regions with an annual rainfall of 31.5 inches. The concentrations of Sr^{90} per unit volume for other annual precipitations are to be derived by inverse proportionately, e.g., in Antarctica, where the annual precipitation is about one-fourth of 31.5 inches, the Sr^{90} content of snow should be given by 16 R, or

$$A' = 16 \times 1.20e^{-t(1/10+1/40)} \text{ dpm/gal}. \tag{3}$$

In this connection, it is very important to note that regions of frequent rainfall very probably will receive more Sr^{90} fallout than will more arid regions.

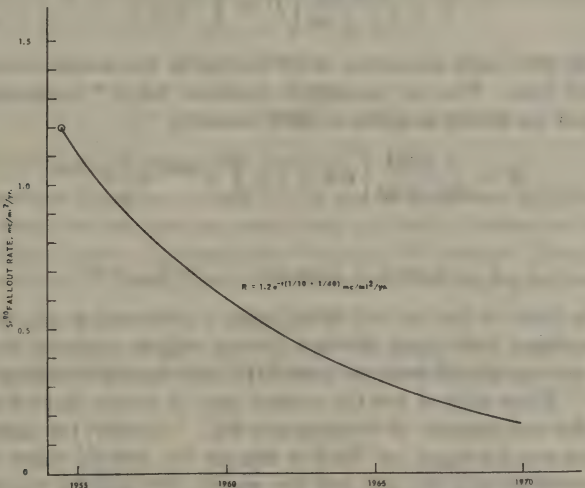


FIG. 9.—Predicted fallout rate for Sr^{90} formed prior to and during operation castle.

Of course, some fallout will be deposited by the surface winds blowing over the leaves of trees and grass. For example, the Naval Research Laboratory[10] has mounted an uncharged platinum screen vertically and held normal to the surface winds by a large vane. The deposition is by impact. Two weeks' total collections were made, and these gave up to 20 times as much as for gummed papers of the same area exposed for the same time in the same place. The screen was 80 mesh and probably passed about 0.5×10^6 ft.^{3} in the 2 weeks' exposure. From this it is clear that surface contact with fallout-laden tropospheric air must result in dep-

osition. Further evidence for this is seen in Table 1, where there is seen to be essentially no correlation between the Sr^{90} contents of soils and the crops of alfalfa grown on them. There obviously must have been very considerable direct dep osition on the surface of the plants. Also, the relatively low values for the Sr^{90} content of surface air found by the Naval Research Laboratory (Table 11), calculated with respect to the observed rain content, may very well be due to surface deposition by direct contact on tree leaves and grass.

The soil content will be the total of all fallout radioactivity, *less any natural weathering processes which serve to remove the fallout Sr^{90} from the chemically available form in which plants can assimilate it.* Neglecting this latter effect, although, as we shall see, there is reason to believe that such effects are operative in an important way, and taking the average for the exchangeable Ca content of soils over the world (cf. Tables 2 and 3), which is 8 gm/ft²/in. we calculate that the top 2.5 inches of soil, which in general holds nearly all the Sr^{90} (Tables 2 and 3), has some 20 gm of exchangeable Ca. Therefore, we predict that in the absence of curative weathering effects acting to remove Sr^{90} from contact with the biosphere, the average Sr^{90} concentration of exchangeable Ca, in MPC units, should be

$$S = \frac{0.0792}{2.2 \times 20}\left[P + \int_0^t R\,dt\right], \tag{4}$$

where P is the pre-Castle deposition of 0.8 mc/mi² in the middle latitudes and 0.2 mc/mi² world wide. Thus in the middle latitudes the Sr^{90} concentration of exchangeable soil Ca should be given in MPC units by

$$S = \frac{0.0792}{2.2 \times 20}\left[0.8 + 1.2\int_0^t e^{-t/10}\,dt\right]e^{-t/40} \tag{5}$$

or,

$$S = [0.0015 + 0.0213(1 - e^{-t/10})]e^{-t/40}. \tag{5'}$$

This result in terms of mc/mi² and MPC units is presented graphically in Figure 10. The maximum post-Castle Sr^{90} soil activity will be expected in about 1970. The present average should be about 0.005 MPC unit for soil with 20 gm. exchangeable Ca/ft². Those soils of low Ca content can, of course, have a much higher Sr^{90} content for unit amount of exchangeable Ca. Consider, for example, certain areas in Wales near Cardigan (cf. Table 3, Sample No. 54417), where the available Ca/ft² amounted to only 0.4 gm. and the specific Sr^{90} activity was found to be 0.097 MPC. For this area our analysis would predict a forty fold higher content than that given in Figure 10. This, of course, is reflected in higher contents for the bones of grazing animals.

The weathering processes which may operate to fix the fallout Sr^{90} and make it unavailable to the biosphere, such as the fixation in massive Ca deposits, are worthy of consideration. Only further investigation will reveal how important such processes are. The present Project Sunshine sampling program includes repeated sampling of given regions. The data so obtained should disclose any such trends. Some data already in hand seem to indicate such effects, but further confirmation is necessary.

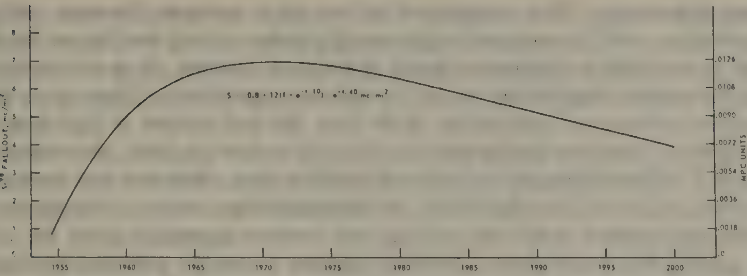


FIG. 10.—Predicted Sr^{90} in soil following Castle (Assumed Ca = 20 gms./ft²/2.5″)

In addition, palliative measures may prove effective. For example, Nervik, Kalkstein, and Libby[19] have shown that milk can be purified for radiostrontium by a treatment which may well prove to be quite practical and inexpensive.

B. BIOSPHERE CONTENT OF Sr^{90}

It seems clear that there will be discriminatory barriers of some magnitude operating at each of the stages of transfer from the soil into the biosphere. Taking these stages in general to be

(1) Soil → Plants,
(2) Plants → Animals,
(3) Animals (milk) → Humans,

we consider the three corresponding barriers.

1. *Soil-to-Plant Transfers.*—In Table 12 the Sr^{90} contents of plants are compared with those for the soils on which they grew at a variety of localities just before Operation Castle. It seems clear that, on the average, plants have about twice the specific Sr^{90} content relative to Ca that the supporting soils do. It seems

TABLE 12

PRE-CASTLE PLANT Sr^{90} CONTENTS
(Values Are Given in Terms of 1/1,000 MPC Units)

Location	Plant	Date	Reference	Plant Content	Content of Soil Which Grew Plant
U.S.	Alfalfa	(Average of Table 1)		8.9	4.7 ± 0.4
Turkey	Alfalfa	10–53	*	2.16 ± 0.18	1.2 ± 0.1
Cuba	Tobacco	4–54	*	1.7 ± 0.2	(1.6)†
New Zealand	Forage	11–53	‡	1.17 ± 0.28	0.21
New Zealand	Forage	1–54	‡	0.84 ± 0.10	0.18
Chile	Forage	11–53	§	0.84 ± 0.02	0.33*

* E. A. Martell and W. F. Libby, Project Sunshine Bull. No. 10, January 10, 1955.

† Calculated from average pre-Castle deposition (Fig. 4*a*) for latitude of Cuba and assumed average Ca content of 20 gm/ft² in top 2.5 inches.

‡ E. A. Martell, Project Sunshine Bull. No. 10, Suppl. 1, March 1, 1955.

§ E. A. Martell, *ibid.*, Suppl. 2, June 1, 1955.

very likely that this is due in part to fallout occurring directly on the external plant surfaces. This is further borne out by the lack of detailed correlation in Table 1 between soil and alfalfa results for eleven middle western United States farms. From this result, we may calculate that about half the total Sr^{90} content of alfalfa is due to direct fallout, while for general forage it is probably an even

higher percentage. This comparison is not quite accurate, because, although almost all the Sr^{90} is contained in the top 2.5 inches of soil, the Ca, on the other hand, is available to the entire depth of the root system. It is also recognized that the vertical distribution of the Ca may be nonuniform. There is no reason to expect preferential assimilation of Sr from the soil relative to Ca; hence the only other explanation for the data in Table 12 is direct fallout. As remarked in Section I in the paragraphs on rain and air-filter data, there are two mechanisms for direct fallout of the ultra-finely divided particulate matter carrying the Sr^{90} in the stratospheric reservoir—rainout and contact deposition after the fallout has entered the troposphere. Rainout appears to be the principal mechanism, though it has been demonstrated by the Naval Research Laboratory,[20] as mentioned earlier, that an 80-mesh screen mounted vertically to prevailing surface winds can gather more fallout than falls out directly on the average by all mechanisms. It is not clear, however, to what degree foliage acts in this way, but the probability seems high that the effect is relatively minor and that rainout, followed by drying of raindrops, is the main way in which the foliage surfaces gather fallout.

On this basis we conclude that total fallout in arid regions should be appreciably lower than in areas with normal rainfall. This effect seems to be borne out by the data available now, though more definitive experiments are needed. It probably follows also that regions subject to seasonal rainfall rather than relatively uniform precipitation all year should show less fallout for the same total annual rainfall. Also, regions subject to frequent morning fogs may be particularly high in total fallout. The Sr^{90} probably will enter the troposphere at relatively uniform rates, but the chance of precipitation will depend strongly on the local weather.

Of course, rainfall is necessary to plant growths, so that plants are certain to gather some fallout. However, for regions of low rainfall where irrigation is used—such as the Imperial Valley in California—the fallout content of the crops should be particularly low, for, as shown in Table 7, rivers are nearly free of fallout, since the soil purifies the runoff water before it reaches the rivers. Similarly, reservoirs and lakes will be low relative to rain because of dilution and the importance of runoff water from surrounding watersheds in replacing evaporative and withdrawal losses. It is also well to note that the ordinary water purification processes are effective in removing an appreciable fraction of the radiostrontium.

The by-passing of soil entirely, which occurs in the direct fallout on plant surfaces, of course means that the retarding effects of high Ca contents in soil are inoperative, and cattle grazing on such foliage may show little correlation in the Sr^{90} contents with the soil Sr^{90} activity for this reason. This appears, from the data in Table 1, to be true in the United States Midwest.

It is clear, however, that washing may reduce the level of fallout externally carried by plants, though direct leaf absorption will be expected to occur rather rapidly for water-soluble fallout. For the megaton weapons fired in the Pacific, the bulk of the fallout resides on particles of CaO or $Ca(OH)_2$ or mixtures of CaO, $Ca(OH)_2$, and $CaCO_3$ made by the great heat of the fireball acting on the coral of the islands and sea floor in the firing areas.[21] A large amount also is carried on $NaCl$ particles. This material, therefore, should be quite water-soluble and should

be rapidly absorbed into the leaves. Washing therefore probably will not be particularly effective for the world-wide fallout, which derives from the Pacific tests. From weapons fired in the air, the particles probably will consist of less soluble oxides and therefore are more likely to wash off of plant surfaces before being absorbed.

Menzel,[22] of the United States Department of Agriculture, grew cowpeas on 42 American soils to which equal amounts of bomb debris had been added. Available Ca ranged from 0.7 to 48 milliequivalents of Ca/100 gm of soil. The Sr^{90}/Ca ratio of the plants was approximately inversely proportional to the available Ca in the soil over the full range of Ca availability. In another set of experiments on a particular type of soil (Evesboro) to which known amounts of Sr^{90} had been added at two carrier levels, the results listed in Table 13 were found. The distribution

TABLE 13

PLANT ASSIMILATION OF Sr^{90} FROM SOIL

Crop	(Sr/Ca) Soil (By Equivalents)	k_{Sr}	k_{Ba}
Barley	0.017	0.45	0.020
	0.0017	0.39	0.022
Buckwheat	0.017	0.49	0.023
	0.0017	0.43	0.028
Cowpeas	0.017	0.53	0.057
	0.0017	0.37	0.053

factor, k_{Sr}, defined as (Sr/Ca)plant/(Sr/Ca)soil, indicates the discrimination which the plant makes between Sr and Ca uptake. Similar tests were made for Ba.

By combining these data, Menzel concluded, as shown in Table 13, that the average Sr uptake from American soils was best fitted by a distribution factor of $k_{Sr} = 0.36$. This average probably will apply world-wide about as well.

2. *Plant-to-Animal Transfer.*—The Sr^{90} contained in grass and foliage eaten by grazing animals will be retained to an extent dependent on the metabolism of the animal. For example, for a 1-year-old steer[23] 30 per cent of the Sr^{90} fed orally was retained with essentially no discrimination relative to Ca. There appears to be a higher retention, approaching 90 per cent, for intravenous injection of young rats.[23] High Ca diets reduce the Sr^{90} uptake for rats, adult rats take up about 16 per cent of the ingested Sr^{90} on the same low Ca diet for which the young rats took up 73 per cent.[23]

Comar[24] has performed experiments on cows in which the Sr/Ca ratio in feed, blood, and milk was measured under equilibrium conditions. Typical relative values were 1.0, 0.37, and 0.13, respectively, thus indicating a relative lowering of the Sr^{90} content of the milk relative to the feed. This is borne out by direct observation on the fallout, as seen in Table 1, where for the Chicago milkshed the milk averaged 0.0014 MPC, while the alfalfa fed averaged 0.0089 MPC.

3. *Milk-to-Human Transfer.*—This ability of the cow to reduce the Sr^{90} in the milk relative to the feed is important as a barrier to human ingestion of this fallout radioactivity. With it in mind, we can expect that human Sr^{90} burdens should be 20 per cent or less (for older people) of the plant contents and about equal to the milk and cheese levels if the entire Ca in the body were assimilated at a given Sr^{90} content of the milk.

Since the Sr^{90} content of the whole biosphere is continually rising, about as shown in Figure 10, the average Sr^{90} content of milk should rise in a similar manner. Therefore, the intake of Sr^{90} by humans is steadily increasing, as shown in Figures 1*a*, 1*b*, 1*c*, and 1*d*. The data in Table 3 and Figures 1*c* and 1*d* show that the milk level at the time of Operation Castle averaged about 0.001 MPC, peaking in the middle latitudes, as did the soil assays (cf. Fig. 4*a*). This value shows a ratio of about 0.7 for the milk level to the average soil level for the top 2.5 inches of soil with 20 gm. of contained available Ca/ft². This high value probably is due to leaf pickup of fallout. Taking this as a general result, we predict the world values for milk and cheese at about 70 per cent of the soil values given in Figure 10, plus any local fallout. This would mean that we would expect average foreign milk and cheese samples to show about 0.004 MPC at the present time.

The human bone, of course, is formed during the growing process, so that the Sr^{90} content of children should be higher than for adults. The data[2, 8–12, 25] verify this prediction; however, for newborn babies there is less Sr^{90} than corresponds to the milk, showing the retarding effect of the mother's older Ca pool. Children seem to carry Sr^{90} approximately equal to the average level of the milk during the period of their lives. Adults decrease in Sr^{90} concentration with age as expected. It seems that the adults of the future will have Sr^{90} levels corresponding to the milk levels during their lifetime weighted according to their rate of growth. For example, the years from 10 to 18 will be most important for men and from 6 to 12 for women. Foreign children born now, according to Figure 10, should develop about 0.011 MPC unit during their lives. Children born now in the United States will develop a somewhat higher level, due to somewhat higher milk levels.

[1] "World-wide Effects of Atomic Weapons: Project Sunshine," Rand Corporation, R-251-AEC, August 6, 1953.

[2] W. R. Eckelmann and J. L. Kulp, Project Sunshine Bulletin, October 15, 1955.

[3] S. Kaufmann and W. F. Libby, "The Natural Distribution of Tritium," *Phys. Rev.*, **93**, 1337, 1954.

[4] H. von Buttlar and W. F. Libby, "Natural Distribution of Cosmic Ray Produced Tritium. II," *J. Inorg. and Nuclear Chem.*, **1**, 75, 1955.

[5] L. A. Currie, W. F. Libby, and R. Wolfgang, "Tritium Production by High Energy Protons," *Phys. Rev.*, **101**, 1557, 1956.

[6] F. Begemann and W. F. Libby, "Tritium Content of Surface Water, March 1954 to January 1956" (in press).

[7] Personal communication from Dr. Lester Machta, United States Weather Bureau.

[8] E. A. Martell and W. F. Libby, Project Sunshine Bulletin No. 10, January 10, 1955.

[9] E. A. Martell, Project Sunshine Bulletin No. 10, Suppl. 1, March 1, 1955.

[10] E. A. Martell, *ibid.*, Suppl. 2, June 1, 1955.

[11] E. A. Martell, *ibid.*, Suppl. 3, September 1, 1955.

[12] E. A. Martell, Project Sunshine Bulletin No. 11, December 1, 1955.

[13] G. H. Wilkening, "Natural Radioactivity as a Tracer in the Sorting of Aerosols According to Mobility," *Rev. Sci. Instr.*, **23**, 13, 1952.

[14] I. H. Blifford, L. B. Lockhart, Jr., and H. B. Rosenstock, "On the Natural Radioactivity in the Air," *J. Geophys. Research*, **57**, 499, 1952.

[15] O. Haxel and G. Schumann, "Selbstreinigung der Atmosphäre," *Z. Physik*, **142**, 127–132, 1955.

[16] P. E. Damon and P. K. Kuroda, "On the Natural Radioactivity of Rainfall," *Trans. Am. Geophys. Union*, **35**, 208, 1954.

[17] "World-wide Fallout from Operation Castle," NYOO Report NYO-4621, January 21, 1955.

[18] Project Sunshine: Progress from September 1953 to January 4, 1954, NYOO Report NYO-4571, January 8, 1954.

[19] W. E. Nervik, M. I. Kalkstein, and W. F. Libby, "Purification of Milk for Calcium and Strontium with Dowex X-50W Resin," University of California Radiation Laboratory Report No. UCRL-2674, August 12, 1954.

[20] I. H. Blifford, Jr., and H. B. Rosenstock, "Fallout Dosages at Washington, D.C.," Naval Research Laboratory Report NRL-4654, November 3, 1955; I. H. Blifford, Jr., L. B. Lockhart Jr., and R. A. Baus, "Relationship between the Air Concentration of Radioactive Fission Products and Fallout," Naval Research Laboratory Report NRL-4607, November 4, 1955; R. A. Baus, I. H. Blifford, Jr., and L. B. Lockhart, Jr., "Radioactivity of the Air," Naval Research Laboratory Report NRL-4509, March, 1955.

[21] "Fallout Symposium," AFSWP-895, January, 1955.

[22] R. G. Menzel and I. C. Brown, "Leaching of Fallout and Plant Uptake of Fallout," bimonthly report from United States Department of Agriculture, March–April, 1953.

[23] United States Atomic Energy Commission, Division of Biology and Medicine Report, No. 100A July, 1954.

[24] C. Comar, "Agricultural Research Program—Semiannual Progress Report for July 1–December 31, 1953," Report ORO-110, April, 1954, and C. L. Comar and R. H. Wasserman, "Radioisotopes in the Study of Mineral Metabolism," in *Progress in Nuclear Energy*, Vol. 5, ("Biological Sciences and Medicine" [London: Pergamon Press, Ltd., 1956]) (in press).

[25] H. L. Volchok and J. L. Kulp, Project Sunshine Annual Progress Report, March 15, 1955.

[26] Sunshine Report for May and June, NYOO Report NYO-4653, July 5, 1955.

[27] Sunshine Report for July and August, NYOO Report NYO-4661, September 16, 1955.

[28] Sunshine Report, August, 1954, NYOO, HASL-S-2 (NYO-4656), November 12, 1954.

[29] NYOO Report NYO-4623, January 18, 1955.

Reprinted from the Proceedings of the NATIONAL ACADEMY OF SCIENCES,
Vol. 42, No. 12, pp. 945–962. December, 1956.

CURRENT RESEARCH FINDINGS ON RADIOACTIVE FALLOUT

BY WILLARD F. LIBBY

UNITED STATES ATOMIC ENERGY COMMISSION, WASHINGTON, D.C.

Communicated October 17, 1956

I. INTRODUCTION

The radioactivity produced by the fission reaction, being due to a mixture of many different fission products, changes its characteristics continuously and rapidly following release by the bomb detonation. Thus the conditions of firing are of extreme importance in determining the fallout effects. The intensity of radiation is enormously greater soon after the detonations, decreasing about tenfold for every seven fold increase in age. Since the time required for ingestion into the body is long, ingestion is unlikely for the shorter-lived fission products, and therefore the principal hazards for close-in fallout are radiation exposures by gamma radiation of the whole body and by beta radiation on the skin.

In the longer times, weeks and months after the explosion, the ingestive hazards begin to become important. The most serious of these is the high-yield fission product, radioactive strontium (Sr^{90}), which because of its own radiation and those of its short-lived daughter, yttrium 90 (Y^{90}), and because of its chemical similarity to the bone-building element, calcium, finds itself deposited in bone structure. Other radioactivities produced would be as bad if they spent as long a time in the body or if their radioactive lifetimes were long enough or if they were produced in high yield. Strontium has all these characteristics. Hence for the fission products which have survived the first weeks, the most important fallout constituent and the one most seriously to be considered is Sr^{90}. Neither radiostrontium nor its yttrium daughter emits gamma radiation, but only beta radiation. After the first-year cesium 137 (Cs^{137}), with half-life of thirty-three years, is the principal source of the residual gamma radiation, and any gamma radiation exposures due to fission products which are more than one year old are due very largely to radioactive cesium. In fact, old fallout can be thought of as a mixture of roughly equal radiation intensities in millicuries of radioactive Sr^{90} and radioactive Cs^{137}. The other isotopes either constitute no ingestive hazard or fail to emit gamma radiation in appreciable intensity. Thus, the hazards of world-wide fallout reduce themselves largely to the ingestive hazard of radioactive Sr^{90} and the external exposure from radioactive Cs^{137}.

The mechanism by which atomic-weapon debris is disseminated leads to three kinds of fallout. First, there is the strictly *local fallout*, which is due to the return to earth of the larger particles in the fireball. These may have their origin in the dirt, the soil, or tower structures which are taken into the fireball and either wholly or partially vaporized. The fraction of the total which falls out locally depends very much upon the conditions of firing. The most serious factor is the degree of contact of the fireball with the surface; another is the nature of the surface. For example, soil appears to be much more effective than water in producing local fallout.

Experience has shown that an atomic device exploded on the surface distributes about 80 per cent of its fission products on the ground within a few hundred miles of the burst point. A somewhat larger percentage takes part in the close-in fallout from an underground burst, and a smaller percentage will be scavenged from a near-surface burst or tower shot.

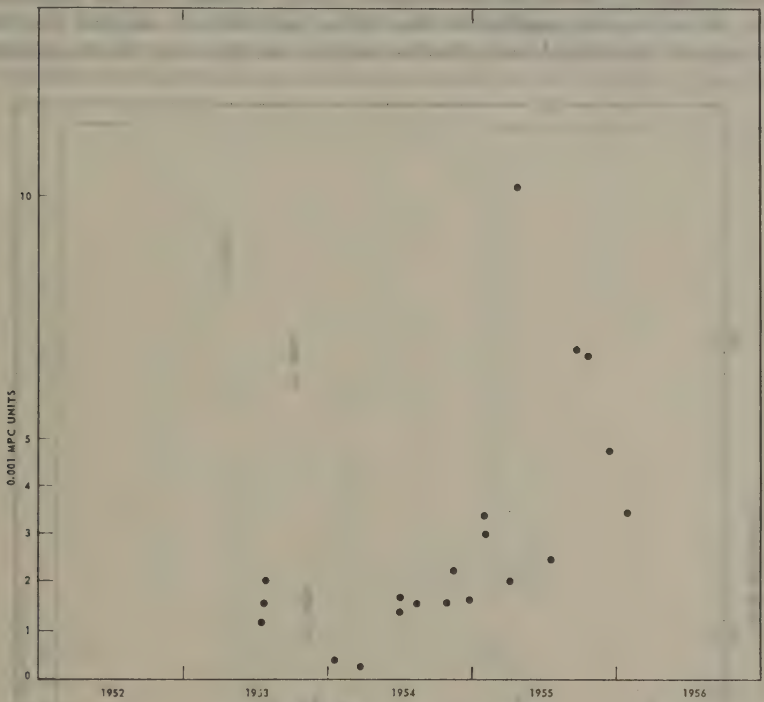


FIG. 1a.—Wisconsin cheese, Sr^{90} content

The tower shot is, in a sense, a special case of a surface burst, since the material of the tower itself is mixed with the fission products in the fireball to a greater or lesser degree, depending on the yield. Experience with tower shots indicates that even in cases where the fireball does not touch the ground a few per cent of the radioactive fission products come down as close-in fallout.

The fraction which takes part in the close-in fallout from a surface burst over deep ocean water appears to be somewhere between 20 and 50 per cent. This is less than the fraction of close-in fallout occurring from a corresponding surface burst over land, due to the evaporation of many of the drops before they reach the ground. Presumably this fraction is also affected by the prevailing humidity and temperature structure of the atmosphere through which the drops must fall. As the depth of the water is decreased, the point is reached where the fireball extends downward to the bottom and picks up bottom material. In such shallow water one would expect a higher percentage of close-in fallout than in deep water. Ex-

perience in the Pacific indicates that such is indeed the case and that in fact there would be very little difference in the fallout between a large-yield device in very shallow water and a true surface shot.[1]

The second type of fallout is the material which, though not coarse enough to fall of its own weight in the first few hours, is, nevertheless, left in the lower layer of the atmosphere, known as the *troposphere,* where ordinary weather phenomena occur. It is now well established that fallout particles are removed from this lower layer of the atmosphere in the first month or so; therefore, the tropospheric

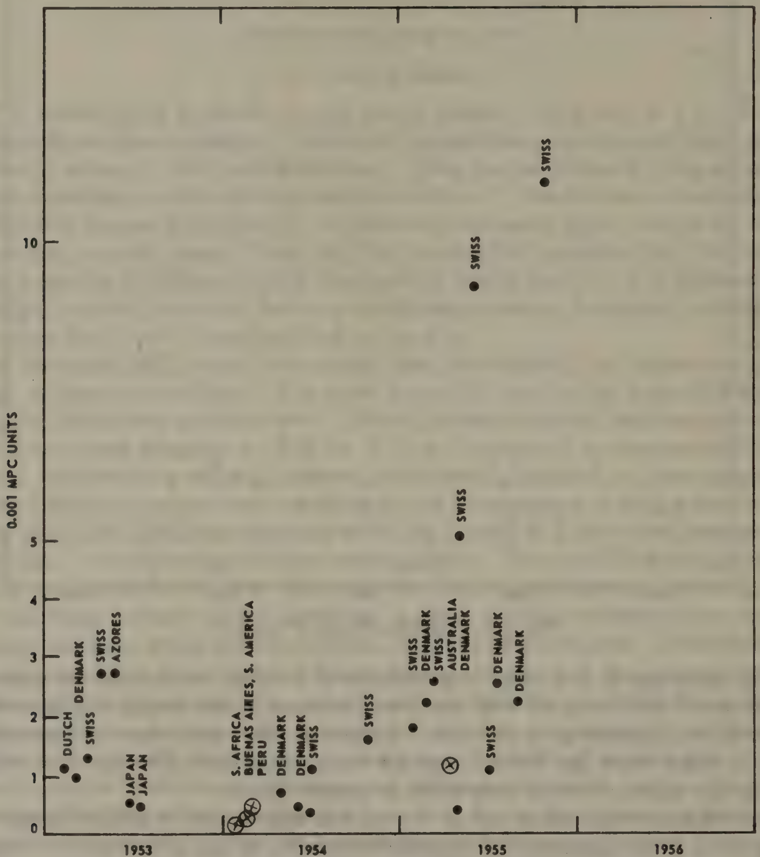


FIG. 1*b*.—Foreign cheese, Sr^{90} content (*crosses:* Southern Hemisphere)

fallout stays airborne at most a matter of a month or two before being deposited. Of course, in this time, it will in general move great distances, possibly even all the way around the earth; but, in general, it stays in the general latitude in which the explosion occurred. So the second type of fallout, the tropospheric world-wide fallout, produces a band of radioactivity in the general latitude of the firing site.

The fraction of the fallout in this category depends mainly on the bomb yield and the conditions of firing. A bomb which is fired on the ground produces a maximum of *local fallout*, naturally leaving less for the world-wide fallout of either the tropospheric or stratospheric variety. The bomb yield determines the division of the world-wide fallout between the two kinds of world-wide fallout. A general rough rule is that a 1-megaton bomb will produce clouds which push into the higher layer of the atmosphere, the stratosphere, before disseminating and that the clouds from bombs of less than 1 megaton will tend to stay mainly in the troposphere. Thus we see that a 500-kiloton weapon fired so its fireball did not touch the ground would

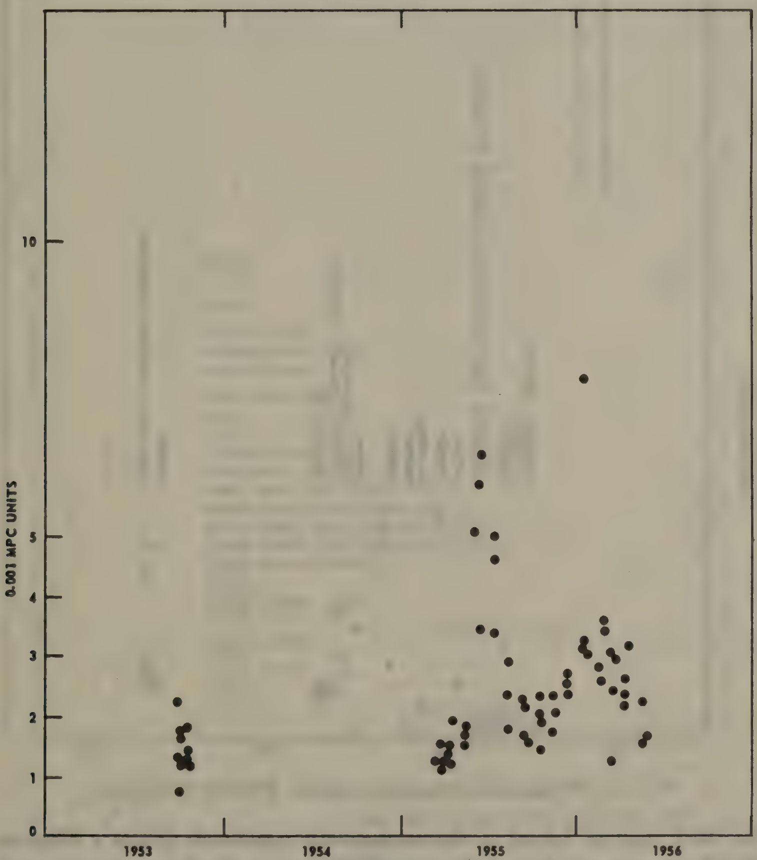


FIG. 1c.—Chicago Milk, Sr^{90} content

be expected to put the major part of its radioactivity in a band stretching around the world in the general latitude of the firing site. The distribution of the activity would be world wide and would be completed within the first month or two. Similarly, the same bomb fired in contact with the earth with ordinary soil would have a

large fraction—something like 80 per cent—of its fallout deposited within the first few hours within a few hundred miles of the test site, and the rest of the material would be spread in the tropospheric world-wide fallout pattern in a band around the earth in the same general latitude.

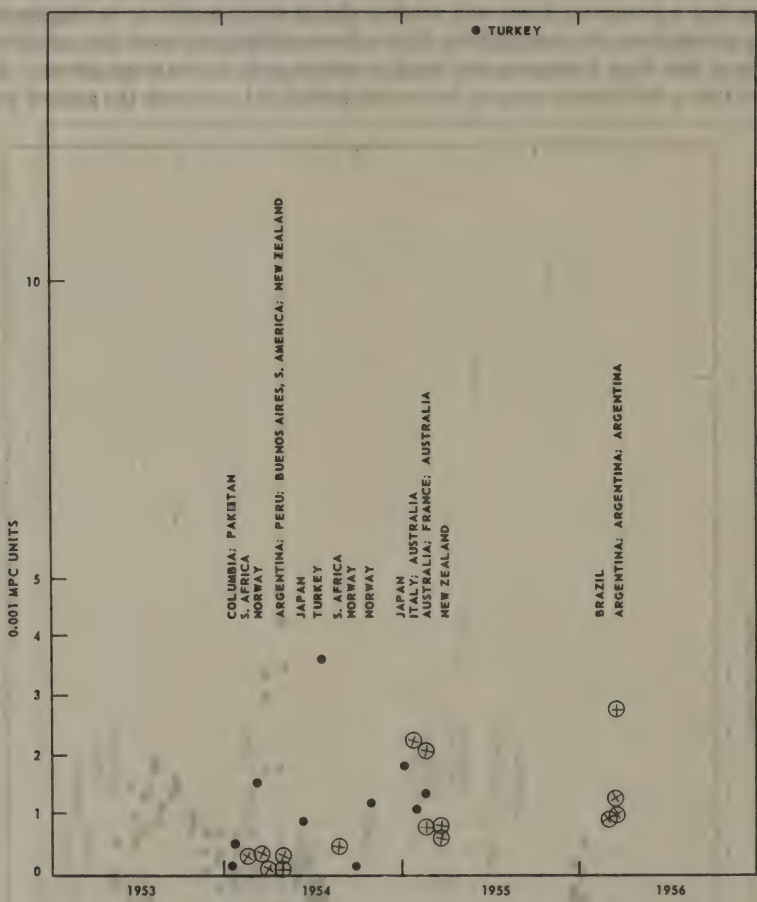


FIG. 1*d*.—Foreign milk, Sr^{90} content (*crosses:* Southern Hemisphere)

The third type of fallout is the *stratospheric world-wide fallout.* Weapons of yields of 1 megaton and greater thrust their radioactive clouds into the stratosphere, and the material which does not fall of its own weight within the first few hours is then very largely borne in the stratosphere for great lengths of time. An average time seems to be about ten years or somewhat less. A small amount of tropospheric world fallout is also produced, presumably due to a small fraction of particulate matter which is of just the right size to descend in a matter of weeks. The division into the two types, the *local* and the *stratospheric world-wide*, is very sharp and

FIG. 2*a*.—Human bone, Sr^{90} content

marked, however, and to a very considerable approximation one can say that the megaton weapons yield the bulk of their fallout in these two categories. A weapon involving 1 megaton of fission would, if fired in the air, place most of its radioactivity in the stratosphere, and this, in contrast to the tropospheric fallout, appears to be

FIG. 2*b*.—Human bone, Sr^{90} content, Lamont measurements

widely distributed latitudinally and does not descend through the tropopause into the troposphere until months and years—an average time of about ten years—have elapsed. The resultant pattern of fallout appears to be essentially in approximately uniform world-wide distribution. The long time spent in the stratosphere is proba-

bly due to the absence of such scavenging agents as rain or snow, so the particles either must fall out of their own weight or diffuse downward by slight eddy motion, either of these processes being of their very nature slow. After passing through the tropopause into the troposphere, they will be rained out rather quickly in a matter of days or weeks. Because of the long residence time in the air, this type of fallout is particularly harmless as a gamma-ray hazard, since only the Cs^{137} is left. The amount of Cs^{137} is about the same in millicuries as the Sr^{90}; thus 1 megaton of fission thus distributed throughout the stratosphere would yield about 1/2 mc/mi^2 of either Sr^{90} or Cs^{137}. Just as in the case of Sr^{90}, the rate of deposition of about 10 per cent of the reservoir per year corresponds to a stratospheric fallout rate of Cs^{137} of 0.05 mc/mi^2/yr in the beginning. This rate decreases to half, or 0.025 mc/mi^2/yr, at about seven years, as the stratospheric reservoir becomes depleted.

FIG. 2c.—Human stillborns (measurements by Chicago laboratories) (error less than 0.00005, unless otherwise indicated; parenthetical numbers are number of cases used for the average)

After the Castle test series was completed, there were about 24 megatons of fission in the stratosphere, corresponding to about 12 mc/mi^2 of Sr^{90} on the average and about the same amount of Cs^{137}. The subsequent stratospheric worldwide fallout rate appears to have been a little over 1 mc/mi^2/yr all over the world. In addition, of course, local fallout and tropospheric latitudinally localized worldwide fallout have occurred from subsequent weapons tests by the Russians and the Redwing series completed last summer at the Eniwetok Proving Grounds.

II. RESEARCH FINDINGS

A. Amount of Fallout and Expected Sr^{90} Body Burden from Weapons Fired to Date.—Since our last report,[2] further data on the actual magnitude of fallout in various places in the world and in various selected spots in the food chain have be-

come available. We shall summarize the results. Some human bone now contains radioactive strontium at levels of about one-thousandth of maximum permissible concentration (0.001 MPC; the MPC is 1 microcurie of Sr^{90} for the standard man and proportionally less for children, and is the maximum permissible concentration) in the northern latitudes where the bombs have been fired and the world-wide tropospheric fallout has occurred. There is evidence in the data on human material that age is a factor, e.g., older people who have had their calcium deposited

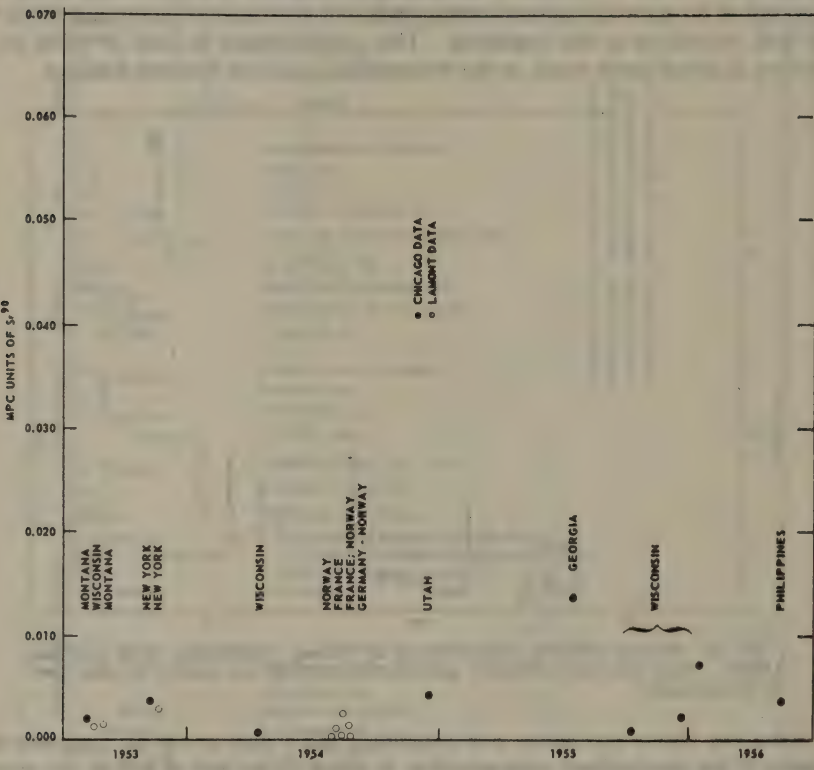


FIG. 3*a*.—Calf bones

prior to the weapons tests show lower concentrations, though in some instances exceptions to this rule are to be found. Lower levels are found in the Southern Hemisphere, where the major contamination is due solely to the world-wide stratospheric fallout, which in the northern latitudes of 10°–50° N. is generally less than one-half or one-quarter of the total fallout. The deposition in the human body seems roughly to parallel the levels of the total fallout. More data are necessary to validate this point fully.

At the end of 1955 the total deposition in the upper Midwest of the United States was some 13 mc/mi² of Sr^{90}. In the spring of 1956 this total rose to about 16 mc/mi².

Between May 5 and mid-July of this year, Operation Redwing was conducted at the Eniwetok Proving Grounds in the Pacific. Particular attention was paid to the fallout problem in this operation, and a major effect was made to produce a megaton-range weapon with an inherently smaller amount of fallout for a given energy release. This effort was successful. In addition, considerable attention

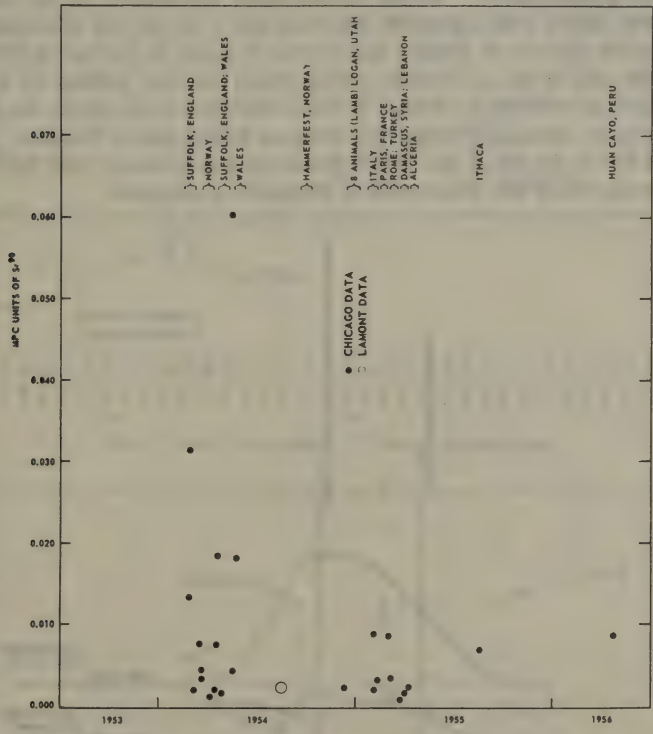


FIG. 3b.—Sheep bones

was paid to operational factors which would minimize world-wide fallout. Thus the total deposition in the stratosphere during this operation was held to a figure very considerably less than that present in the stratosphere before the operation. In fact, we estimate at the present time that the total stratospheric reservoir, counting all sources, is about the same as it was two years ago, i.e., 12 mc/mi² or Sr^{90}, or the equivalent of 24 megatons of fission, calculated as a uniform world-wide distribution. During the last two years the additional depositions in the stratosphere have amounted to about 6 megatons equivalent of fission products total, or 3 mc/mi² of Sr^{90} or Cs^{137}. This appears to have compensated approximately for the 10 per

cent per year of fallout and the 2.5 per cent per year of radioactive decay. In other words, the testing by all countries seems to have restored the stratospheric reservoir to approximately the 24-megaton value of two years ago.

The latitudinal tropospheric world-wide fallout, which is maximized by weapons of high yield which do not puncture into the stratosphere, is increasing. Several such weapons have been air-fired abroad in the last months. This material, for the reasons explained above, descends rather rapidly but all the way around the world in the same general latitude as the firing site. Thus, though it is difficult to estimate, it appears that this amounts to perhaps 5 additional mc/mi² of Sr^{90} and Cs^{137} in the United States. Adding to this about 1/2 mc/mi² for the world-wide tropospheric fallout from Operation Redwing and 1 mc/mi² for stratospheric fall-out, we would estimate at present that a total of about 22 mc/mi² or Sr^{90} is to be found in the soils of the midwestern United States and that perhaps 15–17 mc/mi² is the total to be expected for similar latitudes elsewhere in the world, the difference being due to our proximity to our own weapons testing site in Nevada. These 22 mc/mi² of Sr^{90} in the soil of the United States amount to about 0.040 MPC units in the top two inches of soil, where most of the fallout is absorbed.

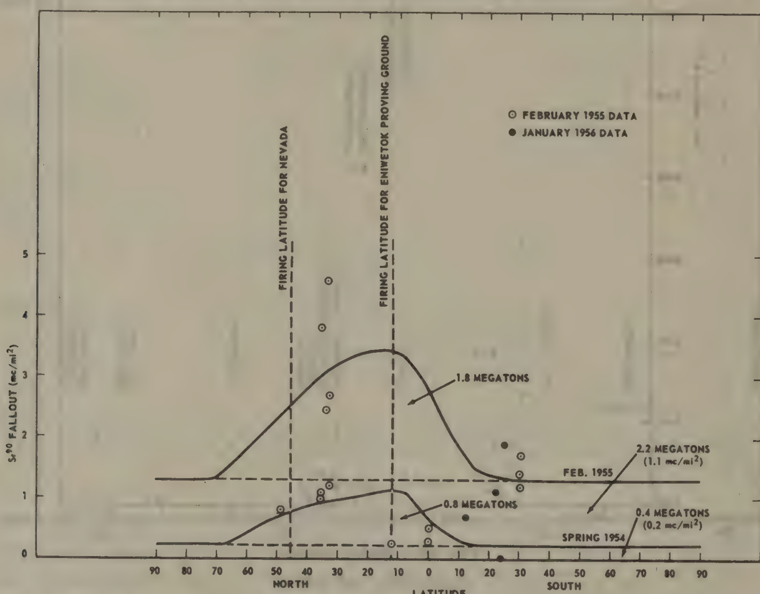


FIG. 4.—World-wide fallout, Sr^{90}, spring, 1955. Latitudinal distribution of foreign post-Castle Sr^{90} fallout (soil assays).

As of the present time, considering the latest human-bone and milkshed data, both domestic and foreign, together with the total fallout figures for corresponding periods, we find that the level of somewhat less than 0.001 MPC units now found in the bones of young children is to be compared with a total Sr^{90} fallout in the soil of about 12 times higher concentration. Additionally, laboratory data have shown that there is a threefold discrimination against strontium as compared to calcium in the assimilation by plants from the soil and that a further factor exists of about

eightfold discrimination against strontium relative to calcium in the excretion of strontium in milk as compared to the cow feed. Earlier,[2] it seemed reasonable to conclude that the human-body burden of Sr^{90} might well be as high as 70 per cent of the concentration in the top soil on which people live. The further evidence just

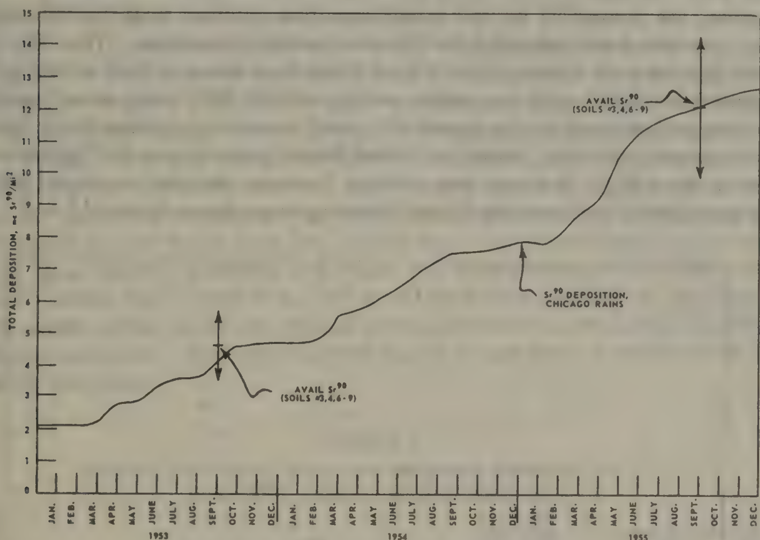


FIG. 5.—Sr^{90} fallout history, Chicago milkshed area

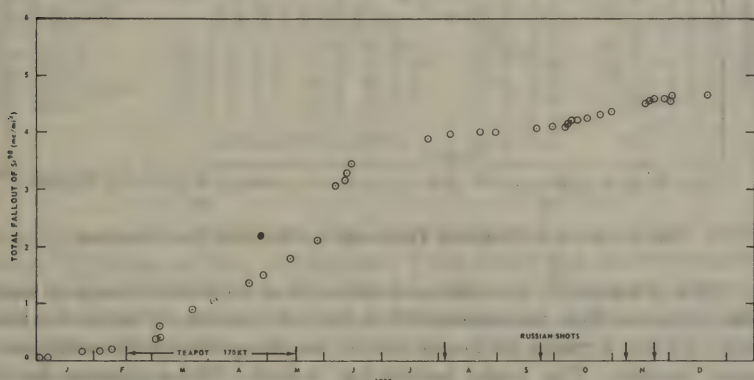


FIG. 6.—Total Sr^{90} fallout in Chicago in 1955 (Pot collection)

cited seems to indicate that this figure is much too high and possibly should be reduced to about 10 per cent. A strict application of the two discrimination factors described would give 4 per cent. Leaf retention of fallout which bypasses the soil causes the figure to be higher. Therefore, at the moment, we would expect that the body burden for children born now in America eventually would amount to be-

tween 0.004 MPC units, corresponding to 10 per cent of the top-soil concentration, and possibly a figure two or three times higher. The stratospheric deposition would be expected to continue at the expected rate, which at the present is about 1.2 mc/yr, so that some fifteen years from now, in the early 1970's a maximum additional total stratospheric fallout of about 6 mc/mi² will have occurred. In the meantime, the present 22 mc/mi² would have been reduced to 15 by radioactive decay, just about compensating for the stratospheric deposition. Thus the conclusion that the body burden in the United States from weapons fired to date would be about 0.004 MPC units, or possibly as high as 0.010 MPC units, seems justified. This level probably will not be exceeded in other countries unless particular factors of environment intervene, since the United States probably has the highest total fallout in the world. It seems very unlikely, however, that environmental factors could increase the level over the United States by more than a factor of 2 or 3.

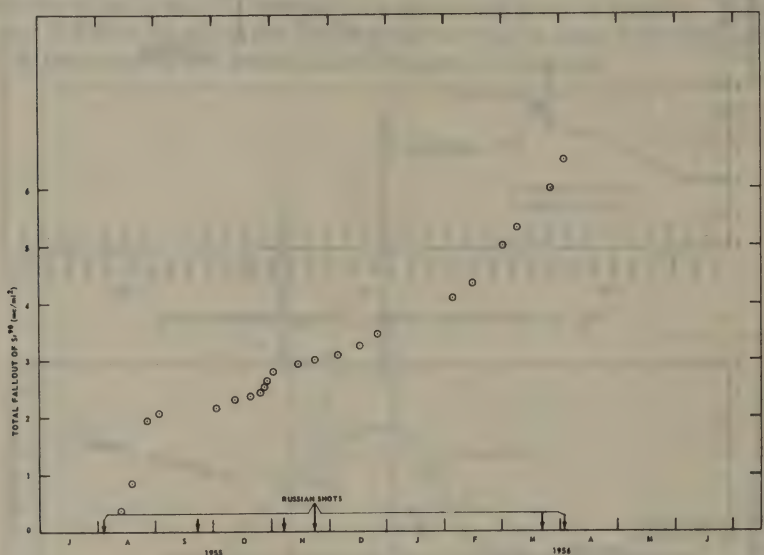


FIG. 7.—Total Sr^{90} fallout in Pittsburgh in 1955–1956 (Pot collection)

B. *Effect of Rainfall.*—As mentioned earlier, there is excellent reason to suppose that the deposition from the troposphere on the earth's surface is best accomplished by rain. By rain is meant not heavy rain but anything which involves the settling of water droplets. This might include fog or mist. The suggestion has been made[3] that the small size of the stratospheric fallout particles gives them a very high mobility due to molecular motion, since, in fact, they probably are almost molecular in dimensions. This high mobility of the particles makes it probable that direct contact of the fallout particles with water droplets will occur. One imagines on this theory that the tiny particles pass through the tropopause from the stratosphere and then meet water droplets in a cloud or mist or rain in the course of their rapid random motion due to collisions with the air molecules. Thus,

rather than the classical Langmuir mechanism of the rain sweeping out the air through which it falls by colliding with the particles themselves, the particles probably collide with the water droplets either before or during the rainstorm, probably most importantly before. It is clear from this mechanism that fog and mist may well be very effective and that a cloud probably gathers a considerable fraction of the fallout from the air in its bulk.

In any case, some experimental evidence has been found for the effect of rainfall on fallout by studying three particularly arid regions: the Imperial Valley in California at the town of Brawley and the western coast of South America at Antofagasta, Chile, and Lima, Peru. The soil in Brawley was sampled in January of this year and found to contain less than 0.6 mc/mi² of Sr^{90}. In order to realize the significance of this number, one should recall that we would have expected about 13 mc/mi² as an average figure for the United States. It is true that a considerable part of this is from the Nevada tests—the depositions of which occur mainly in an easterly direction and might well miss southern California—but it certainly seems that at least 8 mc/mi² would have been expected in the Imperial Valley under normal conditions such as prevail elsewhere in the United States and in Europe and Asia. Thus the observed fallout is not over a few per cent of that expected normally.

TABLE 1

BIOSPHERE Sr^{90} ASSAY, WISCONSIN MILKSHED, SEPTEMBER 30, 1955
(0.001 MPC Units unless Otherwise Stated)

Farm	Soil 0″–2″	Soil 2″–6″	Total Sr^{90} (Mc/Mi²) 1955 Value	Total Sr^{90} (Mc/Mi²) 1953 Value	Alfalfa 1955 Value	Alfalfa 1953 Value
Holcomb, Wisconsin	26.7 ± 1.0	3.8 ± 0.14	14.8	5.1	19.2 ± 1.0	8.3
Premo, Wisconsin*	15.0 ± 0.5 (0″–6″)		10.6	3.8	25.5 ± 1.3	4.1
Kurpeski, Illinois*	12.2 ± 0.4 (0″–6.5″)		10.4	4.0	7.05 ± 0.33	7.4
Austin, Illinois	49.9 ± 1.3	9.6 ± 0.4	16.5	4.7	38.0 ± 2.0	5.0
McKee, Illinois	9.8 ± 0.4	0.99 ± 0.04	10.6	6.3	30.5 ± 1.7	14.8
Van Winkle, Illinois	65.1 ± 2.6	10.0 ± 0.4	9.4	3.8	5.76 ± 0.29	5.0
Carver, Illinois	64.5 ± 1.3	14.0 ± 0.7	8.9	3.3	2.73 ± 0.18	2.3
	Average Sr^{90} (mc/mi²)		12.0	4.5		

* Had been plowed to 6″ depth.

In addition to the soil samples, tests were made on the vegetation grown at Brawley, California, as well. As expected, it was found that the level was lower. Lettuce samples collected from this region at the same time as the soil samples showed 0.0004 MPC; broccoli, 0.00025 MPC; green peas, 0.00134 MPC; alfalfa, 0.0021 MPC. These values all are much lower than those for the midwestern United States (shown in Table 2).

The rainfall data for Brawley are as follows: In 1955 the rainfall totaled 1.70 inches, 1.3 inches having occurred in January of that year and 9 months having had no registered rainfall at all. In 1953 the annual total rainfall was only a trace, this trace having occurred in February.

At Antofagasta, Chile, where it has never been known to rain, except possibly on one occasion, we find 0.02 mc/mi² of Sr^{90} in January, 1956, when the general deposition for this latitude was apparently a little over 2 mc/mi.² In other words

about 1 per cent of the fallout expected was found, and 0.02 is hardly larger than the experimental error of measurement.

TABLE 2

1955 Domestic Soil Samples

Location	Lab No.	Date Sample Taken	Depth of Sample (Inches)	Ca Extracted (Electrodialysis or NH_4Ac)	Calc. Ca (Gm/Ft²)	Sr^{90} Content (0.001 MPC)	Total Sr^{90} (Mc/Mi²)	
Winnebago Co., Ill., Swanson Farm, Site No. 3, Carrington silt loam	551503	9/30/55	0–8	NH_4Ac	82.8	6.83 ± 0.08	9.8	
Rock Co., Wis., Holcomb Farm, Site No. 4, Carrington silt loam	551500	9/30/55	0–2	NH_4Ac	15.0	26.7 ± 1.0	11.4	14.8
Rock Co., Wis., Holcomb Farm, site No. 4, Carrington silt loam	551501	9/30/55	2–6	NH_4Ac	31.8	3.81 ± 0.14	3.4	
Columbia Co., Wis. Premo Farm, Site No. 6, Miami silt loam	551502	9/30/55	0–6	NH_4Ac	25.5	15.0 ± 0.5	10.6	
McHenry Co., Ill., Kurpeski Farm, Site, No. 7	551496	9/30/55	0–6.5	NH_4Ac	30.9	12.2 ± 0.4	10.4	
McHenry Co., Ill., Austin Farm, Site No. 8, Miami silt loam	551504	9/30/55	0–2	NH_4Ac	6.98	49.9 ± 1.3	12.0	16.5
McHenry Co., Ill., Austin Farm, Site No. 8, Miami silt loam	551505	9/30/55	2–6	NH_4Ac	7.8	9.6 ± 0.4	4.5	
McHenry Co., Ill., McKee Farm, Site No. 9, Drummer silt-clay loam	551448	9/30/55	0–2	NH_4Ac	31.2	9.8 ± 0.4	8.5	10.6
McHenry Co., Ill., McKee Farm, Site No. 9, Drummer silt-clay loam	551499	9/30/55	2–6	NH_4Ac	78.4	0.99 ± 0.04	2.1	
Will Co., Ill., Van Winkle Farm, Site No. 11, Plainfield sand	551508	9/30/55	0–2	NH_4Ac	4.4	65.1 ± 2.6	8.0	9.4
Will Co., Ill., Van Winkle Farm, Site No. 11, Plainfield sand	551509	9/30/55	2–6	NH_4Ac	5.1	10.0 ± 0.4	1.4	
Will Co., Ill., Carver Farm, Site No. 12, Plainfield sand	551510	9/30/55	0–2	NH_4Ac	3.7	64.5 ± 1.3	6.7	8.9
Will Co., Ill., Carver Farm, Site No. 12, Plainfield sand	551511	9/30/55	2–6	NH_4Ac	5.6	14.0 ± 0.7	2.2	
Brawley, Calif. (in Imperial Valley)	56316	1/5/56	0–6	Electrodialysis	54.9	≤0.4	≤0.6	

Annual rainfall = 2.57 inches

In Lima, Peru, the total fallout of Sr^{90} in January, 1956, was 0.7 mc/mi². The annual precipitation in Lima averages only 1.89 inches, though there is a considerable amount of ground fog and mist.

It seems clear from these results and the reasonableness of the mechanism of deposition advanced by Mr. Greenfield that there is valid reason to believe that world-wide fallout is small in the absence of precipitation. Also, it is clear from this mechanism that the fallout should not be strictly proportional to total rainfall. Frequent light rains or mists would be expected to be more efficient than occasional heavy rains. The importance of rain is only to be revealed by a study of desert areas and a careful investigation of the scavenging mechanism itself. This work may well prove to be of considerable importance in meteorology, as well as in fallout studies. One should note that the local fallout due to larger particles which descend in the first hours probably does not need rain to precipitate it and occurs in the absence of the precipitation of moisture, although rain may well be able to increase even this fallout.

TABLE 3

Sr^{90} CONTENT OF FOREIGN SOILS AFTER CASTLE

Lat./Long.	Location	Lab. No.	Date Sample Taken	Depth of Sample (Inches)	Sunshine Units	Calc. Exch. Ca (gm/Ft^2) (Ammonium Acetate)	(Electrodialysis)	Total Sr^{90} (Mc/Mi^2)
33° N/36° E.	Damascus, Syria	55590	2/55	0–4	1.40 ± 0.10	62.8	31.7	1.2
49° N/2° E.	Paris, France	55614	2/55	0–4	0.69 ± 0.05	66.2	41.6	0.8
36° N/139° E.	Tokyo, Japan	55644	2/55	0–4	5.86 ± 0.29	18.9	6.1	1.0
0°/27° W.	Dakar, French W. Africa	55645	2/55	0–4	3.71 ± 0.14	4.4	4.5	0.5
0°/27° W.	Dakar, French W. Africa	55646	2/55	0–4	9.31 ± 0.74	1.3	1.1	0.3
36° N/2° E.	Algiers, Algeria	55647	2/55	0–4	1.20 ± 0.08	60.0	32.6	1.1
36° N/2° E.	Algiers, Algeria	55648	2/55	0–4	2.90 ± 0.20	53.8	47.1	3.8
25° S/57° W.	Asunción, Paraguay	56450	1/56	0–6	11.3 ± 0.75	6.1	6.2	2.0
22° S/46° W.	São Paulo, Brazil	56448	1/56	0–6	3.04 ± 0.27	21.0	14.2	1.2
30° S/30° E.	Durban, Natal, S. Africa	55777	2/55	0–4	4.43 ± 0.19	12.6	13.7	1.7
30° S/30° E.	Durban, Natal, S. Africa	55778	2/55	0–4	15.0 ± 0.08	3.3	3.4	1.4
12° N/45° E.	Aden, Saudi Arabia*	55787	2/55	0–4	0.69 ± 0.09	107.2	12.5	0.24
33° N/40° E.	Ankara, Turkey	55878	2/55	0–4	1.9	96.6	51.6	2.7
34° N/36° E.	Beirut, Lebanon	55591	2/55	0–4	3.2	63.9	52.6	4.6
34° N/36° E.	Terbol, Lebanon	55592	2/55	0–4	1.8	110.7	46.9	2.4
24° S/70° W.	Antofagasta, Chile	56447	1/56	0–½	0.44 ± 0.04	5.2	1.7	0.02
12° S/80° W.	Lima, Peru	56456	1/56	0–6	0.60 ± 0.04	63.9	42.7	0.7
30° S/115° E.	Perth, Australia	55839	2/55	0–4	14.7 ± 1.1	2.8	3.0	1.2

* Exchangeable Ca by isotopic exchange method for this sample was 19.0 gm/ft^2.

The importance of precipitation as a scavenging mechanism raises the possibility that different regions will be subjected to varying intensities of fallout, depending upon the weather conditions. It will be important to test whether this is so and whether it is a major effect in populated areas. We have evidence showing that extreme aridity greatly reduces the long-range or world-wide fallout, as explained above. The evidence to date does not indicate that it is a major effect for normal climates, in the sense that it does not appear to amount to more than a factor of 2. Regions in which people live normally have enough precipitation so that differences in precipitation appear not to affect the fallout by more than such a factor. Careful study of the data appended and the previously released data[2, 4, 5]

have failed to reveal any more serious deviation. To summarize, desert regions with little or no precipitation, or with only very minimum precipitation, apparently have minimum long-range, world-wide fallout; but other regions do not show that the fallout is proportional to the total precipitation, nor should it be expected to be so; however, detailed conditions related to frequency of precipitation might well be important. The data to date do not reveal deviations from the general average by more than about a factor of 2, and, in fact, they seem to indicate a smaller deviation than this. There is some evidence that certain areas have had more fallout than one might expect on the model described above and on previous occasions. In particular, there are reports that certain areas in England show higher levels, but the deviations appear to be considerably less than twofold.

More important, probably, than the variations in total fallout due to weather conditions is the effect of calcium in the soil in reducing the rate of assimilation of radioactive strontium by plants. The plants assimilate strontium because it is chemically similar to calcium and, since their appetite for calcium is limited, a larger calcium content in the soil dilutes the strontium so that a smaller fraction is assimilated. This effect might amount to a factor of 5 in the human-body assimilation of radiostrontium in regions with very low calcium content in the top soil.

C. *Direct Measurement of the Stratospheric Fallout Content.*—Direct measurement by means of high-flying balloons has shown that the stratosphere does indeed have about the fallout anticipated in it.[6] In addition, it has been found in these measurements that the radiocesium, Cs^{137}, occurs at about the same level as Sr^{90} in millicurie units, indicating that there has been no serious fractionation of the two fission products by the fallout mechanism. It further points out that the sampling of the stratosphere is a practical matter and that measurements can be made of the stratospheric content of radioactive fallout. Such data should greatly assist the whole study of fallout.

D. *Radiocesium Assays for the Biosphere.*—Recently a technique for measuring Cs^{137} in biosphere samples, particularly in human bodies, has been developed by Mr. L. D. Marinelli, of the Argonne National Laboratory.[7] It has been found that about four-millionths of one millicurie is present in an average adult. This corresponds rather well to the expected amount, considering the short residence time of radiocesium in the body, which is about 3 months, and the expected precipitation rate, which is taken to be equal, as a first approximation, to that of radiostrontium. The radiocesium, of course, constitutes no hazard, amounting in radiation dosage to a small fraction of the amount present in the blood in the form of that received from the ordinary potassium present in the body. Potassium is naturally radioactive. It is interesting further evidence, however, that the general model of fallout set forth is consistent with the data and essentially correct.

III. PLANS

It is clear that the peoples of the world are extremely interested in radioactive fallout because of the bearing that the new phenomenology of the nuclear age has on everyone's life. For this reason we must understand radioactive fallout in all its intricacies. It is to be hoped that the study will be a co-operative, international one. The United Nations Scientific Committee on Effects of Atomic Radiation offers an ideal forum for the discussion and consideration of the problem. From

these deliberations will come further suggestions, ideas, appraisals, and statement of the problem. The methods developed in this country for measurement and all the data collected are available to everyone. It is our hope and intention that this problem, like others of the atomic age, will come to be generally understood.

Fallout is normally considered an aspect of atomic warfare and nuclear armament. There is some similarity, however, between the weapons fallout and the hazard from a reactor accident, in which radioactive products would be disseminated over a limited area but would never reach the stratosphere or undergo anything like the world-wide tropospheric dissemination. As it has so often been observed in the past, so it is again true in this instance, that a new fact of nature is likely to have its beneficent as well as its somber and frightening aspects. As we learn about the way the world-wide fallout particle, probably as tiny as a virus molecule, wends its way from the stratosphere through the tropopause into the troposphere and, within a few weeks, collides with a water droplet and thus is brought to the earth's surface by rain, we shall learn more about the circulation of the atmosphere, about the way in which rain is formed, and about the questions which will naturally arise more and more frequently as the world's population increases world-wide pollution of the atmosphere not only with fission products but with the other by-products of our new technological age.

[1] W. W. Kellogg, R. R. Rapp, and S. M. Greenfield, "Close-in Fallout," P-822-AEC, March 12, 1956.

[2] W. F. Libby, "Radioactive Strontium Fallout," these PROCEEDINGS, 42, 365, 1956.

[3] Stanley Greenfield, Rand Corporation.

[4] John H. Harley, Edward P. Hardy, Jr., George A. Welford, Ira B. Whitney, and Merril Eisenbud, "Summary of Analytical Results from the HASL Strontium Program to June 1956," NYO-4751, August 31, 1956.

[5] Project Sunshine Bulletins Nos. 11 and 12, Enrico Fermi Institute for Nuclear Studies University of Chicago, December 1, 1955, and August 1, 1956.

[6] A. P. Hardy and S. Tarras, "General Mills High Altitude Balloon Filter Samples," Memorandum, New York Operations Office, July 2, 1956.

[7] "Gamma-Ray Activity of Contemporary Man," *Science*, 124, 122, July 20, 1956.

Isotopes in Meteorology

Remarks Prepared by Dr. Willard F. Libby, Commissioner United States Atomic Energy Commission, for delivery before the American Meteorological Society Chicago, Ill., Wednesday, March 20, 1957

A. STRATOSPHERIC MIXING

The existence of winds in the stratosphere together with the long residence times for fine particulates revealed by the fallout studies, make it seem possible that worldwide mixing may occur in the stratosphere in contrast to the troposphere. To establish or disprove this conclusion definitely and to measure the mixing rates would be possible with various radioactive isotopic tracers. The most immediately available are the bomb fallout products, in particular Sr90 and Cs137. This program is underway as Project Sunshine principally, with fallout collections being made by soil sampling and open pots at various places in both hemispheres as well as by means of a program of direct stratospheric sampling with air filters. From this work our present indications are that stratospheric mixing occurs in one or two years and that the over-all storage times for both Sr90 and Cs137 are both about ten years.

These conclusions will either become firmer or else be replaced by a more complicated mechanism corresponding to incomplete stratospheric mixing as Project Sunshine proceeds into its fifth year, and we probably will not be too optimistic if we expect that this study will definitely establish the mixing rate (and possibly the pattern), the storage time, and the general applicability of the single residence time to all stratospheric components which are not subject to commanding and dominating gravity fall. In other words, Sunshine may well teach us whether the whole concept of rapid stratospheric mixing relative to rates of removal into the troposphere is valid and whether these removal rates are the same for gases and particulate matter too fine to fall at any important rate, i. e., below 1 micron. This program will be prosecuted because of its commanding importance for fallout and meteorologists can expect that the help they may get may be appreciable.

It probably would be well to anticipate certain of these results so as to begin planning future experiments on this basis. For example, certain samples can be collected during the International Geophysical Year which would be important if the tentative conclusions that the stratospheric storage time is about ten years and the stratospheric mixing is essentially complete in two years, and that both apply generally to non-falling constituents, all are correct.

There are various types of particulate substances which may originate in the stratosphere and which should, therefore, be constantly fed into rainfall by the slow stratospheric feeding mechanism, whatever it be. Among these is meteoric dust. The fine meteoric matter which is too small to fall by gravity, i. e., O.1 micron or smaller, should blow about in the stratosphere establishing a more or less uniform concentration and finally descend through the tropopause and be precipitated by rain just as in the case of radioactive fallout. It might be wise to take filter samples of stratospheric air to establish the concentration of this material and if some technique for distinguishing this dust from ordinary dust can be devised, it would be interesting to establish the concentration in ordinary rain. It seems likely that the amount is adequate for detection in view of the quantities of meteoric material found in deep sea sediments.

Another type of material which may be descending from the stratosphere is the dust thrown into the stratosphere by megaton sized nuclear explosions. This material probably can be distinguished from meteoric dust by its chemical properties. For example, material from the South Pacific is mainly either calcium carbonate, calcium hydroxide, or sodium chloride, the various solid substances other than ice and, of course, the bomb materials themselves which are derivable from the surface material which could contribute to the fireballs.

It has been suggested recently by Dr. Philip Abelson that the type of mechanism described by Professor Urey and Dr. Stanley Miller might generate amino acids in the atmosphere by the photochemical action of the ultraviolet light from the sun in generating free radicals which could react to form these all important molecules. If this be true, it may be that rain contains amino acids from this source. It, of course, will be difficult to distinguish these molecules from earth dust and the amino acids from the ordinary living material. It may be possible to do this by using the fact that the ordinary amino acids are optically active whereas the postulated stratospheric materials presumably

would not be and thus could be distinguished in principal, at least. Of course, direct stratospheric samples should reveal these materials in much less contaminated form so if search in rain samples fails to reveal them stratospheric air samples may.

Natural tritium produced by the cosmic rays, or, as recently suggested by Dr. Arnold and Dr. Feld, possibly assimilated directly from the sun, is naturally produced or acquired at the highest rates in the highest layers of the stratosphere. The cosmic ray primary radiations interact with a mean free path of about 1/10 of the atmosphere, so although there is appreciable probability of production below the tropopause, the main part of the cosmic ray interactions with the aid occurs in the stratosphere. Since it is extremely likely that tritium, being an isotope of hydrogen, will burn to water, we essentially have a source of radioactive water in the stratosphere. The recent results from fallout suggesting that the stratospheric storage time for fine particulate matter is of the order of ten years, make it appear likely that a considerable fraction of all the natural tritium resides in the stratosphere, and that, in fact, it may spend a considerable part of its average radioactive life of 18 years there and so disintegrate there in considerable degree to form He^3 before entering the troposphere and the earth's water system. The ratio of the moisture content of the stratosphere to that of the troposphere is so small that if taken together with the stratospheric storage factor mentioned, it leads one to expect that the tritium concentration of stratospheric moisture may well be as much as 100,000-fold above that for ordinary surface water. This ratio of the stratospheric concentration to the tropospheric concentration will, in itself, give a more or less direct measure of the storage time. The sampling of the stratosphere for moisture must, of course, be done with balloons, aircraft or rockets, and air filters which will collect the moisture. It is not necessary apparently to know the volume of the air which is sampled, so the problem is one of the simplest of the stratospheric sampling problems, and it seems that it should be relatively simple to do. It would be important to sample at various latitudes to determine whether the expected uniformity with respect to latitude actually exists. It also would be very valuable to have data versus altitude.

Perhaps a word should be said about the short residence time of the bomb tritium in the stratosphere. Why is it possible that natural tritium have a residence time of perhaps ten years in the stratosphere whereas at least a major part of the bomb tritium, which certainly was pushed into the stratosphere with the bomb cloud, was observed by Dr. Begemann and myself to spend only something like 40 days in the atmosphere? The reason probably is that the bomb tritium is contained in relatively large ice crystals. The amount of water in the fireball is so large that when the fireball is pushed into the stratophere and cooled, relatively large ice crystals form. The proof of this lies in the appearance of the cloud, for the cloud as actually seen in its white mushroom outline in the stratosphere is visible mainly because of ice crystals or supercooled water droplets. In any case, the material which is pushed into the stratosphere is of sufficient mass so that gravity fall will cause it to leave the stratosphere relatively quickly. It is the material which does not settle by gravity that we expect has the ten year residence time. Probably any bomb fired in the troposphere will have enough moisture from the air so that it will form large enough ice crystals to cause the tritium to fall out relatively quickly. The stratospheric water samples need not amount to more than a few grams for the concentration should be high enough so isotopic enrichment would not be required. I am sure that Dr. Begemann would be pleased to measure these samples if anyone can obtain them. We will make efforts on our own part, but it is our hope that flights being made for other purposes can be used to obtain these moisture samples.

B. TROPOSPHERIC MIXING

In contrast to the stratosphere, the tropospheric residence times for particulate matter seem to be relatively short. Work with radon decay products done by Haxel in Germany and by Blifford and Damen and other people in this country, all agree that whatever the fate of the non-volatile radioactive decay products of the four day half-life radioactive noble gas radon emitted by uranium, the materials stay in the atmosphere only a fwe days before being precipitated out, probably mainly with water droplets. This is the same story as for the fallout particles. All the evidence known apparently indicates that there is some type of mechanism by which troposheric air is periodically cleansed in a matter of a few days, or at most a month or so.

It has been known for a long time that the rain and snow are good scavengers for atmospheric contaminants, and the clearing of the atmosphere following precipitation is recognized by everyone as a fact. It is less well known that this holds true even for the finest material. The new evidence for this is that the fine material which resides in the stratosphere for periods of years is, in fact, carried down by water droplets when it enters the troposphere in a relatively short time. This agrees with the radon decay products evidence for it was shown by Werkman and his coworkers that the particles which collect the radon decay products are in general microscopic, probably of the order of a few hundred angstroms in diameter. What can this mechanism be?

It is very well known that a falling raindrop can hardly pick up any particle so small that its inertia will not prevent its flowing around the raindrop as the air molecules do. Therefore, it has been something of a mystery as to how the fine particulate matter which carries the world-wide fallout in the stratosphere could be precipitated in the form of rain, as was shown rather conclusively to be the case by the fact that fallout is observed to be minimal in desert regions (though it is by no means proportional to total rainfall in other areas). The answer seems to have been given by Dr. Stanley Greenfield, who pointed out that the Brownian motion, that is, the violent random motion due to collisions with the air colecules, gives these tiny particles a considerable probability of colliding with the droplets in clouds. Therefore, he suggests that any cloud, mist or dew is an excellent medium for scavenging fine particulate matter and placing it, of course, in the rain which is formed subsequently from clouds. From this theory, which seems to be so explanatory of much of the information on moisture scavenging of fine particulate matter, we would expect that the efficiency with which fine particles entering the troposphere from the stratosphere are carried down will depend not so much on the total rainfall or total moisture precipitated as on the average lifetime of a particle as determined by the probability of its colliding with a smaller water droplet in a cloud, mist or fog. In other words, a foggy climate, or one given to morning dews, or one with frequent light rains, may well be more effective in this precipitation mechanism than one with infrequent heavy rains. The fallout evidence seems to agree with this, but further work with the Greenfield theory in mind ought to establish the matter clearly.

Of considerable more importance for meteorology, it seems likely that nucleation nuclei will in themselves have the same fate that these fine particles we speak of suffer, and that the fallout pattern should be the pattern for the cleansing of the air of stratospheric nucleation nuclei which are so important in the formation of ice crystals. The study of this problem of the history of fine particulate matter in the troposphere ought to be of real value to meteorology.

Thus, we have a model for the complete removal of particulate matter from the troposphere by water drops since the mixing up to the tropopause seems to occur in a matter of weeks, and this residence time for the particulate matter appears to be about the same as the residence time for the average water molecule in the troposphere. It appears that the air in the bottom rain-bearing layer of the troposphere is cleared in about 1 week while the troposphere as a whole requires 3 weeks or a month on the average.

Of course, these results are incomplete and it is necessary that many of the features be tested by further experimentation. For example, we should find whether it is really true that the air in a cloud is clear of fallout, and that the air in a fog is free of condensing nuclei and of virus molecules and all other forms of particulate matter which are fine enough for the Greenfield mechanism to operate. Some of these experiments could well be conducted in the course of the International Geophysical Year.

In contrast to the stratosphere, it seems that the short residence time in the troposphere makes latitudinal mixing essentially impossible. Thus, we find that fallout which never reaches the stratosphere or which falls out of the stratosphere quickly does not spread itself across the equator latitudinally. Most of the fallout from the testing in the Northern Hemisphere being tropospheric in character has occurred in the same general ranges of latitudes as the test sites occupy, and to a rough approximation it is true that all of the fallout in the Southern Hemisphere is due to stratospheric material disseminated by the stratospheric mixing mechanism. Therefore, it should consist of radioactivities which are necessarily old and, therefore, should not contain short-lived radioactive fission products because of the long stratospheric residence time. This point needs experimental testing. It is not a difficult measurement and the results should be available soon.

Insofar as these conclusions and predictions are borne out by further observation, we can expect that particulate contamination of the tropospheric air by industrial sources and others will restrict itself largely to the general region of the heavily populated areas in the Northern Hemisphere, and that any effects of such sources of particulate matter on the local weather should not pass themselves across the equator. It would be extremely interesting to observe whether the density of nucleation nuclei for ice crystal formation is appreciably lower in the Southern Hemisphere, as it might be according to the above notions.

C. MIXING OF THE STRATOSPHERE WITH THE TROPOSPHERE

The mechanism by which stratospheric material eventually finds its way into the troposphere can be elucidated in part by the use of isotopes. For example, if the stratospheric residence time for fine particulate matter and gaseous materials should prove to be identical there would be no doubt any longer that the mixing is one of air masses rather than the falling out of particles or the removal of fine material by some other mechanism. We might for a moment consider a specific mechanism for the removal of matter from the stratosphere into the troposphere and vice versa—a mechanism which consists of a mixing caused by or associated with the seasonal change in the height of the tropopause. This rise and fall of the dividing layer between the troposphere and stratosphere will constitute a type of pumping action. The shifting of the boundary occurs periodically and while the tropopause is near its higher levels, the characteristic rapid mixing of the troposphere takes the air which was formerly in the lower layers of the stratosphere and mixes with what was formerly in the troposphere; and during the subsequent time when the tropopause is at its lowest level, tropospheric air will be mixed similarly with the stratosphere. Now if the oscillation is assumed to occur annually and perhaps one-tenth or one-twentieth of the atmosphere lies between the two extreme locations of the tropopause, it would appear that ten to twenty years would be the expected average time for mixing. This is so reasonable in terms of fallout observation that it may just be that this relatively simple mechanism is the correct one. It was first mentioned to me by Professor James Arnold of Princeton University, but I am sure that meteorologists have thought about it for a long time. This model would predict that the mixing does not occur at exactly the same rate at all points on the earth's surface and the longer residence time in the stratosphere and the magnitude of the stratopheric winds would have to be depended upon to accomplish the latitudinal mixing.

Assuming this mechanism, one could say that the noble gases would establish a steady concentration in the atmosphere which in the case of radioactive materials would be determined by their radioactive half-life and the mixing time, since rain and water cannot scavenge and precipitate them. There is evidence for this already in the case of radon. The ratio of the abundance in the stratosphere to the abundance in the troposphere of the radioactive noble gas radon which has its origin at the earth's surface is known to be essentially zero. By the time the radon can be transported into the stratosphere there is litle if any left because of the four-day half-life for the radioactive decay. Air filters in the stratosphere show little radon decay products. This fact shows that the general model has some applicability to gases.

Gases generated in the stratosphere such as molecular hydrogen containing tritium should establish a concentration ratio between the stratosphere and troposphere which would be determined again by the stratospheric residence time and the radioactive half-life. If, for example, we assume that tritium is generated only in the stratosphere and that the molecular hydrogen has a negligible rate of combustion to water after being formed (an experimental fact in the laboratory unless a spark occurs); one calculates that the ratio of the tritium concentration in the form of molecular hydrogen in tropospheric air to that in the stratosphere should be slightly less than one-half, the half-life for the radiotactive decay of tritium being 12.26 years. This calculation also assumes that the escape of molecular hydrogen from the atmosphere into interplanetary space occurs at a rate which is negligible compared to the rates of combustion and the rates of mixing with the tropospheric air.

A check can be made on the assumption that the rate of combustion of molecular hydrogen in the stratosphere is small compared to the rate of mixing with the tropesphere by observing the ratio of the concentration of molecular hydrogen in the stratospheric air to that in the troposphere. This is so because

93299°—57—pt. 2——33

it is probably a valid assumption that molecular hydrogen is generated solely in the stratosphere by the action on water vapor of the ultraviolet radiations from the sun. If the combustion lifetime is long compared to the general stratospheric residence time for stratospheric matter, the compositions in the two layers will be the same. This observation would be important to make. It would require the collection of considerable amounts of stratospheric air in order that the hydrogen content could be determined. Similar analyses have been made on the noble gas content of air as a function of altitude.

D. SUMMARY

In conclusion, it seems likely that isotopes can contribute appreciably to meteorology both in the new techniques and data they can furnish as well as in interesting students of other disciplines in the problems of meteorology. Meteorology is such a broad subject that the possibilities for applications of specialized techniques and knowledge are large, ranging all the way from biology to pure astrophysics.

Isotopes have been observed many times to have a catalytic effect in encouraging the mixing of disciplines in science. Physicists and chemists have intermingled for years in the pursuit of nuclear science and the long history of biology is remarkable for the important contributions made by men from other disciplines. If isotopes can help catalyze this intermingling, this service alone will in itself constitute a considerable contribution to meteorology.

We have made only brief references this evening to the cosmic ray radioactivities. The reason is that these have been discussed previously in considerable detail by others, but we should not leave the subject without referring to the fact that in addition to radiocarbon and tritium, two beryllium isotopes and a chlorine, a sulfur, and two phosphorous isotopes all have been reported to be produced by the cosmic rays as they bombard the atmosphere. These give additional opportunities for meteorological observation similar in kind to those discussed in some detail tonight.

There are many other potential applications of isotopes. The possibilities of artificial labeling of air masses by the use of radioactive isotopes are important, and the maximum information has not yet been obtained from fallout material. So many other possibilities exist that meteorologists should earnestly consider the applications of nuclear techniques to their field. In many instances they will find the Atomic Energy Commission very interested in the research.

Remarks prepared by Dr. Willard F. Libby, Commissioner, United States Atomic Energy Commission, for delivery before the University of New Hampshire Distinguished Lecture Series, Durham, N. H., April 11, 1957

Radioactive Fallout From Nuclear Tests

FALLOUT FROM TESTS

There is a great deal we do not know about the precise effect of radiation on the human body, but we do know that the effect of radioactive fallout from nuclear tests is not, nor is it likely ever to be, the danger to the human race in this generation or in later generations which many people have been led to believe.

Long before nuclear weapons were even thought of, in fact, ever since people have lived on this planet, they have been subject to radiation from cosmic rays and from the radioactive material in the crust of the earth. In recent years has been added radiation from the use of X-rays, from luminous devices, etc.

Let us compare radiation from test fallout with radiation from some of the other sources which have been with us through all times so that we may better evaluate fallout as an element of danger. Cosmic rays, which come from outer space, have their radiation effect progressively diluted as they pass through the atmosphere surrounding the earth. Thus, the person living in Denver, Colorado, at an altitude of about 5,000 feet receives a dosage of cosmic rays approaching double that of a person who lives at sea level.

At the present time, the radiation dosage to bone from the most worrysome part of radioactive fallout, which is Strontium-90, is about the same as what

the Denver resident would receive as additional radiation from cosmic rays if he moved from Denver proper about 200 feet up on the mountainside, or to the resident at sea level if he moved from the beach to the top of a hill 300 feet high.

More exactly, and using the measure commonly used for radiation exposure, the roentgen, which is an amount equal in the chest to six to 10 chest X-rays—at sea level in our latitudes we receive 37 thousandths of one roentgen each year from cosmic rays, while people living at Denver, Colorado, at an altitude of 5,000 feet receive 60 thousandths of a roentgen annually. Now, in order to compare fallout we must put all items in the same terms.

FALLOUT AND RADIOACTIVE STRONTIUM

At the present time, the radiation effect on human bones from radioactive strontium, the most worrisome part of radioactive fallout, is about 1.5 thousandths of a roentgen annually—about one-fifteenth of the difference between the cosmic ray dosages at sea level, and at 5,000 feet elevation, or equal to the extra cosmic rays at the extra height of about three hundred feet.

Radioactive strontium does not occur naturally but is produced by nuclear reactions. It also has a natural tendency to stay in our bones and thus to irradiate them. How much does it amount to? As just stated, the most recent data show that children in the United States have about 1.5 thousandths of a roentgen annually of extra bone exposure from the radiation from radioactive strontium. Adults have much less because their bones grow more slowly. This country has somewhat higher rates than elsewhere because of our tests in Nevada.

The effects of irradiation of bone are bone tumor and leukemia. How much hazard does the bomb test radioactive strontium constitute? This is a difficult question to answer with complete certainty for the amounts which cause these effects are not well known, but we do know that the dosage is much less than the extra cosmic ray dosage at 5,000 feet altitude as compared to sea level. We have examined the vital statistics on these dread illnesses to see whether there is a higher rate for populations living at 5,000 feet than for those living at sea level in order to get from these data some direct notion about the magnitude of the effect. There is no evidence of any observable effect of the extra radiation in these numbers. The average annual incidence of bone cancer at both levels was about 2.8 per 100,000 people in 1947, and for leukemia, the average was about 7. The actual numbers were:

Occurrence of bone cancer and leukemia

[New cases per year per 100,000 population]

	Bone cancer	Leukemia
Denver	2.4	6.4
New Orleans	2.8	6.9
San Francisco	2.9	10.3

Figures obtained from National Institutes of Health, Department of Health, Education, and Welfare.

Since excessive doses of radioactive strontium are known with certainty to cause both bone cancer and leukemia in animals, we must not deprecate or casually dismiss the possible results of a widespread, but low intensity, effect in causing these disabilities. However, by using normal experience insofar as it is applicable, we can orient ourselves with respect to this new factor in our environment—much as we do for other results of our modern way of life. We cannot see any effect of a radiation dosage fifteen times the present test fallout dosage in large populations, but the number of cases of bone cancer and leukemia each year, even in the large cities, is small so our data may not be too significant. However, these figures do point to a definite tangible evidence of a margin of safety.

Perhaps more pertinent, though less direct, is the result of animal experimentation and the rare human experience on the effects of radiation in causing bone tumors and leukemia. The official tolerance limit for people working in atomic energy plants and with radioactivity professionally is 2,000 times the present bone content for children in the United States, and about 10,000 times that in

adults. However, it has been recommended by high scientific authorities that the tolerable amounts of radioactive strontium for the population in general should be only one-tenth of those acceptable for workers who of course expose themselves voluntarily. On this basis the present level in children is 1/200 and, in adults, about 1/1000 of this maximum permissible concentration for large groups of people. Perhaps a word of explanation of these tolerance or maximum permissible concentration limits would be helpful. When scientists speak of "risk" or "hazard," they do not use the words in the same sense that most laymen regard them. Scientists try to be precise; they measure such things almost to the limits of the finite; therefore, "risk" means possible effects far beyond the range of the probable or detectable. The maximum permissible limits do not mean that above those limits one encounters trouble—but rather that perhaps only a ten-fold larger concentration would give effects which would be definitely detectable.

WHAT ABOUT THE FALLOUT STILL IN THE STRATOSPHERE?

If all the radioactive fallout which still is airborne should come down suddenly, there would be about one-third more total radioactive strontium on the ground than we now have here in the United States. It falls so slowly, however, that there is expected to be relatively little increase over the present amount deposited—the extra fallout just about compensating for the naural radioactive decay of the radiostrontium already deposited. (The radioactive decay occurs at the rate of 50 percent every 28 years.) The ground level will remain about constant for the next ten years and then drop off at the rate of about 2.5 percent per year due to radioactive decay uncompensated by further fallout from the upper atmosphere. Therefore, in the United States the present level is about as much as we shall ever have from tests already fired.

WHAT ABOUT POSSIBLE GENETIC EFFECTS?

Radiostrontium is not the only fallout product of nuclear bombs. In fact, there are dozens of others but, for one reason or another, these fail to accumulate inside the body as radiostrontium does. However, certain of these other fallout materials do emit penetrating radiation which can irradiate the body from the outside and thus possibly can have effects on the health and can produce genetic mutations. In fact, for early fresh fallout close to the site of a nuclear explosion, this radiation of the body from the outside is the principal hazard, and it is only later on that radiostrontium becomes important. There is one short-lived form of radioactive iodine which is assimilated into the thyroid gland so it is not quite correct to say that no internal irradiation occurs from early fallout, but the external radiation certainly is the main hazard with which the United States Government is concerned in planning protection against enemy nuclear bombs. Later on and far away from the test site, months and years afterwards, there still is some external radiation which does amount to a detectable total and it is this radiation which raises questions.

We are continually being bombarded by radiations not only from the cosmic rays from outer space—the cosmic rays mentioned above—but from the earth and even from our own bodies. Following our previous tactic of comparing these new fallout effects with normal experience insofar as it is justified, we now compare these external radiation exposures with those normally encountered. The external radiation doses from test fallout have averaged between 1 and 5 thousandths of one roentgen per year during the last three or four years. Now, this is to be compared with a normal dosage from ourselves and our environment of 150 thousandths of a roentgen per year. Therefore, we conclude that the radiation dose from test fallout is relatively very small, but the question remains: "Small as it is, does it have genetic and health effects?"

The direct experience which we have had on health effects is reassuring. We do not see that the much larger dosages received by people living in the higher altitudes or in localities which are particularly radioactive have had noticeably bad effects—the effects expected are cancer and shortening of life. However, it may be that small effects do occur which because of their smallness are difficult to see. The only definite thing we can be certain of is that test fallout is very small as compared to natural dosages and, therefore, we know that the effects must not be very far outside of normal experience. One environment can differ from another in natural radiation intensity by much more than the total fallout. For example, a brick or concrete house can easily have enough natural radioactive material in the walls to give up to 40 thousandths of a roentgen per year more exposure than a wooden house—this is between 8 and 40 times the

annual dosage from test fallout. The genetic effects are much more difficult to consider since they will show up only in later generations and, most importantly, only when both parents have been affected either directly or through affected parents. But by study of animals and plants we know that radiation does produce genetic changes—we even irradiate plant seeds in order to speed up the rate at which new forms appear so superior new plants can be produced by selection of the few desirable ones and cultivating them in the way Luther Burbank developed so many useful new plants using just those naturally occurring forms. However, most of the forms from the irradiated seeds have inferior properties and it is only a rare one that is a definite improvement on the original plant. Similar results are found with animals so we guess that human beings probably are subject to same type of effects. Therefore, we believe that there must be *some* genetic effects of test fallout radiation but, again, from our normal experience in which no effects of high altitudes versus low, or brick versus wooden houses, etc., have been observed, we know that the effect must be very small. The laboratory experiments on plants and animals agree with this, but do insist that there must be some very small effect, although it will be entirely undetectable from test fallout.

WHAT ABOUT FUTURE TESTS?

Continued testing at the same rate and in the same way as during the last five years will not increase the hazard on a straight additive basis since an equilibrium will be established between additions of radioactivity and radioactive decay. For Strontium–90, the maximum factor of increase possible is eight-fold and the external radiation exposure outside the immediate test area would behave similarly. In 1980 it would be four times the present and, in 2011, about six times, and so on until after a very long time it would approach the factor of eight.

The cause for real concern is not the deleterious effect of radiation resulting from weapons tests, but rather what would be the effect of the infinitely greater amount of radiation which would result from the massive use of nuclear weapons in warfare. Here we would be dealing with excessive radiation, not to all the people of the world as has been suggested, but quite probably to large numbers of people residing in areas of substantial contamination. With regard to the people so overexposed, there would be serious increases in the pathological effect of excess radiation, such as cancer and leukemia. There would, also, be the genetic effect which would manifest itself in the children and the children's children of such people.

In nature, there are mutations in plants, in animals, in human beings. Some mutations show superior characteristics and these make it possible for us to develop superior strains, but unfortunately most mutations are harmful rather than beneficial. For this reason and because their effect is not limited to one generation, the genetic effects of excessive radiation may be more important than the pathological effects.

If radiation—from weapons test fallout, from natural sources, from the normal use of X-rays—is all measured in quantities so minute as to have very small effects on either present or future generations, we should concentrate our concern on what would happen if the world should engage in a nuclear war, for therein lies the real danger to mankind.

It is not contended that there is no risk, however minute. But all life, and every minute of our day and night, is measured in terms of risk—40,000 highway deaths each year in this country, accidents in the home, etc. We make our choice: How much risk are we willing to take as payment for our pleasures (swimming at the seashore, for example), our comfort or our material progress? Here our choice seems much clearer. Are we willing to take this very small and rigidly controlled risk, or would we prefer to run the risk of annihilation which might result if we surrendered the weapons which are so essential to our freedom and our actual survival.

Radioactive Fallout

Remarks prepared by Dr. Willard F. Libby, Commissioner, United States Atomic Energy Commission, for delivery before the spring meeting of the American Physical Society, Washington, D. C., April 26, 1957

I. INTRODUCTION

The radioactivity produced by the fission reaction changes its characteristics continuously and rapidly following the explosion of an atomic weapon and the

conditions of firing are of extreme importance in determining the rate at which the radioactivity descends to earth. As a result there are in general three different kinds of radioactive fallout, the relative importance of which is determined by the nature of the weapon, principally its yield, and the conditions of firing. These three types are: First, *the local fallout*, which is insignificant unless the fireball touches or comes close to the ground, but which in case the fireball does touch the ground can amount to a major fraction, in some instances as much as 80 percent of the total debris. This type of fallout consists of radioactivity which is carried down by the larger particles. It consists largely of matter drawn up into the fireball from the surface which is either totally or partially vaporized. Under these conditions so much matter is vaporized by virtue of the fireball's touching the ground that the particle sizes formed in the freshly cooled vapor are large.

The second and third types of radioactive fallout are world-wide in nature and consist of finer material and are divided according to whether the material happens to lie in the lower part of the atmosphere, the troposphere, where rain and weather phenomena occur, or the higher part of the atmosphere, the stratosphere, which is free of such precipitating mechanisms. The *tropospheric fallout* occurs in a matter of two or three weeks or a month or so. It occurs largely as a result of rain and snow, and water precipitation in general, and falls in the general latitude of the test site. The *stratospheric fallout*, in contrast, takes years. We are not completely certain, but it appears that an average time of something like ten years, or perhaps somewhat less, is a reasonable figure, and during this time the distribution becomes nearly world-wide. When the stratospheric fallout manages finally to pass into the troposphere it is quickly removed by the same type of mechanism that brings down the world-wide tropospheric fallout, namely rain and moisture.

The precipitating mechanisms consist in general of the collision of the tiny particles with moisture droplets in clouds, together with the interception of particles by falling raindrops. The first mechanism was recently suggested by Dr. Greenfield in connection with Sunshine problems—the study of world-wide fallout is called Project Sunshine. In addition to the scavenging action of rains and fogs, there is definite evidence for a considerable probability of pick-up on direct contact of air with surfaces such as the leaves of grass and trees. Frequently, grasses are found to have higher strontium-90 content than would correspond to the soils in which they grow, and this is due undoubtedly to direct pick-up.

The dissemination of strontium-90 and all fallout is greatly dependent upon the firing conditions. There is every evidence that important factors include not only contact of the fireball with the surface, but the nature of the surface, whether it be land or water and the type of soil and the composition of the water, whether fresh or sea water. Also, the height to which the fireball rises is important, in particular the height relative to the tropopause, the dividing layer between the troposphere and the stratosphere. Yield is the main consideration here. A rough rule is that megaton weapons push through the tropopause into the stratosphere, and kiloton weapons stay below the tropopause in the troposphere.

Thus, we see immediately that kiloton weapons deposit their fission products much more quickly than do megaton weapons. Of course this is of less importance in so far as the long-lived fission products, such as strontium–90 and cesium–137, are concerned, but it is of more importance for the shorter-lived fission products. As a general rule, an air-fired kiloton weapon will deposit its radioactive fallout in a period of between two weeks and one month on the average after the detonation, whereas an air-fired megaton weapon will deposit its radioactive fallout over many years—on the average about ten years. Thus, the effects which are due to the short-lived fission products are larger for a given amount of fission energy release in kiloton weapons than they are for air-fired megaton weapons. Considering the average age of the kiloton fission products to be 1 month, the external gamma ray exposure from one megaton of fission fired as say 50 bombs of 20 kilotons each would be 30 times that for a single bomb giving one megaton of fission energy—if both were fired well up in the air. The fission products from the small bombs fired in Nevada would fall in the latitudes 10°N to 60°N in about one month, while the larger bomb

would give fallout over essentially the whole earth in about 10 years. For strontium–90 effects there is relatively little difference per unit fission yield since even to the residence time in the stratosphere is small compared to the 28 year half-life of radioactive strontium and the 27 year half-life of radioactive cesium, which is produced at slightly higher yield than strontium–90, and which appears to be disseminated in about the same way.

The content of radiostrontium and radiocesium in the stratosphere is by direct measurement shown to be roughly the same though the radiocesium is somewhat higher possibly due to the slightly higher fission yield. The content of radiocesium in rainwater is comparable to that of strontium–90. Also, the content of radiocesium in the human body as measured by Marinelli at Argonne and Anderson and Langham at Los Alamos agrees well with the fact that it has an average residence time in the human body of about five months as compared to many years for strontium–90. The radiocesium data are very interesting because of their bearing on the fallout dissemination mechanism and the confidence with which we can establish the probable future behavior of radioactive strontium. The data confirm previous suggestions as to the dissemination mechanism, that is, we find that radiocesium fallout except of the local variety is carried down very largely in the form of moisture droplets and that there is some direct pick-up by leaves and grass on surfaces. It is captured and held tightly by the top two inches of most soils, so the water which falls and runs off in the form of rivers is clean by the time it has drained a short distance through soil. All of this is very similar to the radiostrontium behavior.

The plants pick the strontium–90, and radiocesium to a lesser extent, out of the soil and also off of their leaves and take it into their systems. There appears to be a discrimination mechanism which operates in most plants so that the strontium–90 content of the plant is considerably less relative to its calicum content than in the case of the soil. On the average, the discrimination factor between the top soil and plants against strontium relative to calcium seems to be about 1.4. When the cows eat grass they further discriminate by about a factor of 7 in making milk so there is an overall protection factor for strontium–90 from the top soil to milk of about 1.4×7 or 10. Also, there is a further discrimination factor against strontium relative to calcium in the human body. This factor is not known too well, but is known definitely to be at least as large as 2 and is thought possibly to be as high as 8. Researches are now in progress to settle this. Therefore, there is a series of protective factors which makes the concentration of radiostrontium derived from milk relative to calcium in human bone not over 1/20 and possibly as little as 1/80 of that in the top soil. Of course, it should be pointed out that there is a considerable part of the fallout which is picked up directly on the leaves and to this the factor of 1.4 does not apply, so for this fraction of the fallout the protective factor may be reduced to 14. Since milk is the source of most of our calcium, this means that the actual ratio of radiostrontium concentration in new human bones relative to that in the top soil should approach these numbers.

It must be realized that though only a small part of the calicum is derived from vegetables and meat, a similar calculation must be made for this portion and the total average ratio obtained. It seems that the meat-vegetable overall discrimination factor is about 10 so if 20 percent of the calcium is derived from such sources on the average the average overall factor will be between 1/13 and 1/30. The experimental data on new human bone in children appear to give a smaller figure, 1/60, as mentioned later.

A matter of importance in connection with the amount of strontium–90 which one would expect to be deposited in human bone as a result of atomic weapon detonation is the calcium concentration in the top soil. Since calcium is so similar to strontium, it seems very likely, and the evidence confirms this, that high available calcium content of the soil will reduce the probability of strontium–90 being taken up into the plants. Of course this probably does not have nearly as great an effect on the uptake of the material which is picked up directly on the leaves. We might expect therefore that soils which are particularly low in calicum might show higher strontium–90 contents for the grasses grown on them. This is, in fact, so, and sheep and goats and cattle feeding on such pasture display a higher strontium–90 bone content.

How such calcium deficiencies in the soil should affect the strontium-90 uptake by the human population is a most important question. One sees immediately that food distribution systems are such that the food supply is derived from large areas, and that there is consequently a sharp reduction in the sensitivity of the

human population to calcium deficiencies in local soils. This is brought out particularly well by the data on the radium contents of human bones and their obvious lack of strong dependence on the radium contents of local waters. But for people who consistently drink milk from cows grazing on such ground there should be a definite effect on the amount of radio-strontium uptake and the effect should be proportional to the radiostrontium content of the milk. So the question resolves itself largely into "What are the strontium-90 contents of the foods people in such regions actually consume?" We find on inspection of the food eating habits and calculation of the strontium-90 intake relative to calcium, that the increase in average strontium-90 concentration of the food due to the low calcium content of the particular soils can hardly be more than five-fold for a soil-calcium deficiency of 50-fold. That is, whereas normal soil carried about 20 grams of available calcium in the top 2.5 inches, a region with soil of only .4 grams per square foot would produce a human body burden equilibrium of about five times that which the normal soil would produce.

In order to understand the hazard of radiostrontium, which is generally agreed to be the most hazardous of the long-lived fission products, we try to establish the maximum permissible concentration both for occupational workers and for the population in general. These numbers have been set at 1 microcurie and .1 microcurie for the standard man, respectively. That is, an occupational worker may carry 1 microcurie of strontium-90 in his body, whereas the general public should not have over .1 of a microcurie of strontium-90 in the average standard adult. This last figure corresponds to a concentration of 100 micromicrocuries per gram of body calcium or what we call 100 Sunshine Units, that is, 1 micromicrocurie of strontium-90 per gram of body calcium is defined as 1 Sunshine Unit.

Now, we must try to see in some other way how our normal experiences can be brought to bear on the question: "How dangerous is atomic weapons testing from the pont of view of radioactive fallout?" At the present time we have in our bodies about .1 or .2 of a Sunshine Unit and children have about one-half of a Sunshine Unit. In a few minutes I will speak about the question of the variation from these average values, but assuming at the moment that these are the values, what is the threat or the hazard from these quantities? Obviously, they are much smaller than the 100 Sunshine Unit tolerance figure mentioned above. To obtain a comparison with normal experience, let us consider the fact that we know in a general way the magnitude of the radiation levels to which we are normally subjected by the cosmic rays, potassium in our own bodies, and the uranium, thorium and potassium in the ground and in our surroundings. We know these qantities amount to something like 150 milliroentgens per year for an average person in this latitude. But we also know that there are considerable variations with conditions.

For example, a person living in a brick house may very well get 25 to 50 milliroentgens per year more than one living in a wooden house, because of the natural radioactivity of the bricks. It is also very well known that whereas at sea level in this latitude the cosmic ray dosage is 37 milliroentgens per year, at 5,000 feet altitude as in Denver, Colorado, the dosage from cosmic rays is 60 milliroentgens per year, or a difference of 23 milliroentgens per year. What is this in terms of strontium-90 body burden?

First, we must consider what part of the natural radiation, if any, is similar to the radiation of strontium-90 in biological effect so we can say without doubt and hesitancy that the physiological effects, whatever they are, will be the same for the same energy absorbed. Fortunately, the cosmic rays seem to fit this bill. In other words, we are at liberty to compare the cosmic ray radiation dosages with the dosages from radiostrontium in our bone structure. The reason this is permissible is that the ionization density along the tracks of the mu-mesons which are the principal cosmic ray components at sea level and at altitudes of 5,000 feet are nearly the same as those of the yttrium-90 beta rays, the principal radiation which radiostrontium emits; that is, radiostrontium has a radioactive daughter, yttrium-90, which emits a very energetic beta ray and the ionization density along the track of this radiation is very similar to that of the mu-mesons of the cosmic rays and their disintegration electrons, and it is generally accepted by health physicists and radiobiologists that radiations of the same ionization density have very similar, if not identical biological effects for the same energy absorbed. The high energy of the yttrium-90 gives it an average distance of penetration in tissue of 2 millimeters so any effect of local non-uniformity of deposition of strontium-90 in the bone is removed. The cosmic ray exposure is, of course, uniform throughout the bone structure. Therefore, we can equate cosmic ray dosage

with strontium-90 dosage and thus it is possible for us to say that the difference between one altitude and another is equal in effect, other effects being equal, to a certain number of Sunshine Units in bone. Now to follow this thought through, 1 Sunshine Unit is equal to 3 milliroentgens per year. Therefore, the difference in annual cosmic ray radiation dosage between Washington, D. C., or any place at sea level in this latitude, and Denver, Colorado, is equal to 8 Sunshine Units, that is, 16 times the present body burden of equilibrium bone or bone near equilibrium as we see it in young children who are growing now.

Therefore, we must examine whether anything in our experience indicates that these differences are significant in terms of the occurrence of the principal effects expected of radiostrontium, namely leukemia and bone cancer. Now of course when one looks for such vital statistics, one finds that they are very hard to acquire. However, the National Institutes of Health and the Department of Health, Education and Welfare, have given us statistics for the occurrence of leukemia and bone cancer for the year 1947 for the three cities, New Orleans, San Francisco and Denver. They are shown in Table I.

TABLE I.—*Occurrence of bone cancer and leukemia*

[New cases per year per 100,000 population]

	Bone cancer	Leukemia
Denver	2.4	6.4
New Orleans	2.8	6.9
San Francisco	2.9	10.3

It is clear from this table there is no obvious effect of altitude, and it is also clear that there are other factors which are noticeably more important than cosmic ray dosage. Of course there may still be a considerable effect of altitude hidden in large fluctuations caused by other factors, which presumably are largely unknown and we cannot say that this proves anything. It does, however, give us some assurance from normal experience that the effect of eight Sunshine Units will not cause a detectable increase in bone cancer or leukemia.

This fits well with the laboratory data on animals and the limited experience on humans with radium. That is, 1 microcurie being 1,000 Sunshine Units, is still considered to be pretty safe on the basis of the laboratory data. It is set as a tolerance for occupational workers and it is therefore reasonable that eight Sunshine Units should give an effect so small as to be very, very difficult to detect. It is, I think, helpful for us, however, to realize that the present body burden of strontium–90 in new bone from the weapons tests that have occurred in the past is equal to the increase in cosmic ray intensity that goes with an increase of some 400 feet in altitude, a very small fraction of the difference in cosmic radiation intensity between Denver and sea level. Therefore, at the same time that we consider the possible effects of strontium–90 from such concentrations, we may deduce from our everyday ordinary experience limits on the effects to be expected. None of the evidence on the occurrence of bone cancer or leukemia as a function of altitude has given us any reason to believe that the present tolerance limits are in any way in error. The present body burdens in new bones are small compared to these limits.

Separate from the strontium–90 effects are the effects of general gamma radiation, the radiation that is received mainly from outside the human body, and which comes mainly from the very young fission products in the local fallout area, but which can come in smallest part from radiocesium accumulating on the ground in the case of the stratospheric fallout, or more importantly, from the shorter-lived fission products deposited by the tropospheric fallout. Of course, weapons tests are so conducted as to avoid exposures to local fallout, so our present discussion of the effects of weapons will be restricted to the much smaller gamma ray doses from the offsite tropospheric and stratospherice types of fallout. In time of war, of course, it would be the local fallout which would be of more direct concern, next to blast and thermal effects, and it is to this aspect of fallout which FCDA addresses itself in the main. In regard to nuclear tests, we have to study the effects on human genetics and the possible effects of such doses of radiation on health. Let us again apply the criterion of normal human experience to this. Measurements have shown that the general average intensity of fallout gamma rays from tests is 1 to 5 milliroentgens per year. Now

the general magnitude of the effects to be expected from this can be compared with the natural radiation intensity. We find, as mentioned earlier, that such things as living in a brick house, instead of a wooden house can amount to as much as 25 to 50 milliroentgens extra dosage per year, that there are certain areas in the world where the average dose in this country of 150 milliroentgens per year is exceeded by ten-fold, that people living on granitic rock as compared to those living on sedimentary rock receive about 70 milliroentgens per year more dosage due to the higher content of uranium and thorium in these rocks and that people living at higher altitudes have a higher natural cosmic ray dosage. Also, of course, we know that medical uses of X-rays can be considerably larger than any of these fallout dosages.

We do have experience and valid evidence that the somatic effects other than cancer and leukemia, that is, the effects of radiation on ordinary human health, require dosages which are very much larger, of the order of 25 to 50 roentgen units in order to be observed as changes in the blood and 100 to 200 roentgens for injury symptoms; whereas the dosages we are speaking of from test fallout are about one hundred thousand fold smaller.

As for genetic effects, these are extremely difficult to evaluate, since there is so little known about human genetics. But judging from experience with plants, insects, animals, and lower organisms, there is every reason to expect some genetic effects of radiation. The question is how much radiation is required for a given level of effect. There are a certain number of mutations in every new human generation. Are these largely induced by natural radiation or are they mainly of chemical, or rather biochemical origin, or both? From a chemical point of view, it seems likely that not all the spontaneous mutations in the human or any other species are caused by radiation effects, because it seems likely that radiation acts in inducing mutations mainly via molecules which it generates in the human cell, and that the mutations are caused by these chemicals and therefore in a sense are chemical in nature. Now if this be so, and the radiation induced mutations are nearly always caused by chemicals which are produced in the first instance by radiation, then chemicals themselves which are not produced by radiation but have other origins, can cause mutations, so it seems likely that a major part of the natural or spontaneous mutations in any species is not radiation induced. This point is an important one to settle, for the reason that we have to compare the effects of fallout radiation with the fraction of the natural spontaneous mutations which is due to the radiation we are normally subjected to. In other words, if the normal mutations are all due to radiation, then the effects of the additional radiation from general test fallout, or from other sources of radiation such as atomic power, or the medical uses of isotopes and X-ray, will be larger. It seems likely, and many genetic authorities agree on genetic grounds with this conclusion, that a major portion of the spontaneous mutations of the human species is not due to radiation but due to other causes. Therefore, a fraction of the spontaneous mutations in the human species is taken as being due to irradiation. Now, what this fraction is, it is difficult to say, but Professor H. J. Muller has estimated that this might be 10 percent. Therefore, one estimates the 150 milliroentgens per year from natural radiation now causes about 10 percent of the spontaneous mutations, and therefore, that the test fallout if continued indefinitely will, at the present level of about 1 to 5 milliroentgens per year, cause an increase in the natural spontaneous mutation rate of something like $\frac{1}{50}$ of ten percent, or 0.2 of a percent of the spontaneous mutations. In the extreme, if it should prove that all of the spontaneous mutation rate is radiation induced despite the chemical arguments, the effect would be ten times as great, or two percent. Dr. Dunning of the Division of Biology and Medicine of the AEC estimated 1.4 percent in 1955 on similar assumptions. (The Scientific Monthly *81*, 265–December 1955.) This effect is one which is comparable to moving to a slightly different locality and is much less serious than changing from one house to another or doing any of a dozen things. The only important point is that genetic effects show only if large numbers of people are subjected to them. Therefore, we would expect that the effects of large populations changing their environment, such as living at a higher altitude, or living in a region of naturally higher radioactivity, should cause genetic effects, if test fallout does so. An examination of vital records should be made to test for such effects and the Atomic Energy Commission is doing so as best it can. The United Nations Scientific Committee on the Effects of Atomic Radiation has been comparing the data on natural background dosages, and it is hoped that this study will be continued and that the search will be made for observable effects of variations in the natural background dosage, for it is certain that any effects

due to gamma rays from fallout must be already present in much larger measure due to the natural dosage.

II. VARIATION IN INDIVIDUAL STRONTIUM-90 BURDENS

What is the likelihood that even though the average strontium-90 content be well within tolerance limits, that a few individuals should exceed tolerance limits, that a few individuals should exceed tolerance limits? Let us consider first the case which will ultimately hold, the situation of complete equilibrium with the environment in so far as the strontium-90 burden is concerned. The only way we can make judgments about the expected individual variations from the mean concentration is by direct experiment on human body composition, not only for strontium-90 but for other analogous constituents. Most of the recent data on the strontium-90 body burden are from odd bits of bone removed during surgical operations, but fortunately we have actual data for the strontium-90 content of the entire bodies of some several dozen stillborn children [1] in the city of Chicago in the year 1953. A strenuous effort is now being made on the Sunshine Project to continue this series and also to check the human bone data by analyses of complete skeletons. We present the distribution of the strontium-90 data for the stillborn children in Figure 1. Data for the occurrence of ordinary non-radioactive strontium in human bones also have been published.[2] These obviously refer to the full steady state condition and are obviously at least as nearly in equilibrium with the environment as the fallout radioactive strontium ever will be. These data are presented in Figure 2. The occurrence of radium in the human body also has been used since it is chemically similar to both calcium and strontium, and therefore is a bone seeker and because it is obviously also in steady state equilibrium. The data used were those by Palmer and Queen [3] in Figure 3. And, finally, we use the recent data on occurrence of normal potassium in human bodies as determined by Anderson and Langham [4] at the Los Alamos Scientific Laboratory as presented in Figure 4. All of these data show a normal frequency distribution as indicated by the theoretical curves. The respective widths of the curves (standard deviations) are 36 percent for radiostrontium, 40 percent for normal strontium, 40 percent for radium, and 18 percent for natural radiopotassium. It is completely clear from these data that they agree with one another in general shape and that the magnitude of the distribution of the strontium-90 contents of the Chicago stillborn babies was not in any way anomalous. Therefore, we shall take the distribution curve for radiostrontium to be the same as for the normal strontium data. The occurrence of non-radioactive normal ordinary strontium in the bones should certainly tell us what the equilibrium distribution will be for radioactive strontium, and from it we should be able to learn the points about distribution which we cannot yet learn in any detail from the radioactive strontium itself. Turekian and Kulp noted in their study of normal strontium in human bone that in a given region the deviation from the average was about 34 percent of the average, that is, for human bone from the regions Colorado, Texas, Cologne, Bonn, Venezuela, Chile, Vancouver, China, and India. In each instance the ratio of the standard deviation from the mean itself was taken and the average calculated to obtain 34 percent. Therefore, we take 34 percent as the expected standard deviation from the mean *for a given locality* for the eventual strontium-90 equilibrium burden in human bones.

With this result we can, assuming a normal error curve shape of the distribution of probabilities, answer the immediate question: What is the probability of an individual exceeding the tolerance even though the mean does not? On the basis of this analysis we find that at steady state and in equilibrium the variation from the mean will constitute an error curve with a shape corresponding to the standard deviation, being ⅓ of the mean. Therefore, at steady state among people living in a given locality, only one person in about 700 will have more than twice the average strontium-90 burden, and the chances of anyone having as much as three times the normal burden will be about one in twenty million.

Now what about the non-equilibrium distribution, when the strontium-90 is finding its way into the biological system? Obviously, the burden will be much

---

[1] W. F. Libby, "Radioactive Strontium Fallout," Proc. Nat. Acad. Sci. *42*, No. 6, 365–390 (1956); University of Chicago, Project Sunshine Bulletin No. 12, August 1, 1956.
[2] K. K. Turekian and J. L. Kulp, Science *124*, 405 (1956).
[3] Hanford Report, HW–31242.
[4] E. C. Anderson, R. L. Schuch, W. R. Fisher, and W. Langham, "Potassium and Cesium Radioactivity in People and Foodstuffs." (In press.)

lower here, but the deviation from the mean will probably be much higher percentagewise, particularly in adults where most of the bone has been deposited before strontium-90 was produced. The present strontium-90 content of adults depends very much on the growth rate and the metabolic activity of the various bones in the given individual's body which happens to be sampled. However, the specific concentration of the strontium-90 deposited will not exceed that in new bone developed entirely in the present biological environment, i. e., the local concentration in adult bone will not exceed that for the whole bone in young children, whose total bodies are composed of the mixture of strontium-90 and calcium which now is present in food. Since the present ratio for children to adults is about four to one for average total strontium-90 content, the factor of concentration in adults' active bone regions may be as much as four-fold greater than the whole body average. Thus the apparent spread for random bone samples taken from adults should be very large compared to the true equilibrium spread for these reasons. As equilibrium is approached, however, the spread must decrease very, very markedly.

The data on human bones indicate a very wide scatter, but it seems extremely clear that the variation is a reflection of the fact that the main skeleton of adult individuals is not in equilibrium with the present food supply, and that the variations reflect the different rates at which the various bones in the bodies of individuals are coming into equilibrium with the food supply in the general biological environment. A study of whole skeletons taken from one given locality which is now under way as a part of Project Sunshine will clarify the point about the variations among individuals in their rate of coming into equilibrium with the general biological environment. This study is under way in Dr. Kulp's laboratory.

It should appear from these studies that the variation from the mean of adults will be larger than the factor of one-third which apparently is normal for the types of equilibrium distribution considered above. It is, of course, very important to establish the truth of this prediction clearly. However, the general agreement in shape of the distribution curves for such widely different materials as normal potassium in whole bodies, radium, and normal elementary strontium in fragmentary human bone, and actual fallout radioactive strontium in the whole bodies of stillborn children, give us good reason to believe that there is nothing extraordinary in the distribution of radiostrontium in human bone.

III. VARIATION OF THE STRONTIUM-90 BODY BURDEN WITH LOCALITY

Most important of the causes of variation of the strontium-90 content of individuals with locality is, of course, the amount of fallout in a given region. The general rules about the intensity of fallout have been described above. For air-fired megaton weapons our present indication is that the fallout is almost world-wide and for reasons of simplicity and in the absence of better information at the present time, we work on the model that this is a uniform distribution, over the entire world, of material that falls from the stratosphere. Further evidence and data on this point are rapidly being collected which will undoubtedly settle the stratospheric horizontal mixing question.

At the present itme, the general latitudes in the Northern Hemisphere which are betwen 10° and 60° North have the highest strontium-90 content. In the United States, which because of our proximity to the Nevada Test Site has unusually high fallout, there are at the present time about 25 millicuries per square mile of strontium-90. For average soil this means a concentration in the top soil of about 50 Sunshine Units. With the factors of discrimination mentioned above, this means that an equilibrium body burden between 1.7 Sunshine Units and 3.9 Sunshine Units is to be expected. Actually, the present body burden in young children indicates that the lower value is probably more realistic. The present body burden in children—about 0.5 Sunshine Units—probably was derived from an average strontium-90 content in the top soil of something like 15 millicuries per square mile, or about 25 to 30 Sunshine Units during the time the strontium-90 was being acquired. Thus we find that the experimental value for the ratio between the body burden of young children and the average concentration in the top soil is about 50 to 1; rather closer to the higher range of the laboratory results than to the lowest range.

Table II contains the latest data for the total strontium-90 fallout as measured in U. S. soils, and Figure 5 displays these data graphically.

The Northern part of the United States has about 20 to 30 millicuries of strontium-90 per square mile, the Southern States are somewhat lower. The low figure of 7 millicuries per square mile for Grand Junction, Colorado, is probably due to local climatic and sample site conditions.

TABLE II.—*Health and safety laboratory 1956 survey of United States soils for Strontium-90—Samples taken between Oct. 8 and 13, 1956—Strontium extracted with 6N HC1 at room temperature—Replicates represent individual soil aliquots taken after sampling and air drying—Each error term represents 1 standard deviation due to counting error*

Sampling site	Depth	d/m/gm soil	mc/mi²	mc/mi²	
				Ave.	Total
Albuquerque, N. Mex.	0–2″	0.078±0.001	7.5±0.1	7.3	
		0.075±0.001	7.2±0.1		
	2–10½″	0.008±0.002	4.4±0.9	3.4	11
		0.005±0.002	2.4±0.8		
Atlanta, Ga.	0–2″	0.35 ±0.007	14.0±0.3	15.0	
		0.42 ±0.009	16.0±0.4		
	2–6″	0.018±0.004	2.8±0.6	3.0	18
		0.021±0.003	3.3±0.5		
Binghamton, N. Y.	0–2″	0.32 ±0.007	17 ±0.4	18.0	
		0.35 ±0.007	18 ±0.4		
	2–6″	0.019±0.003	4.4±0.8	4.8	23
		0.023±0.005	5.2±1.1		
Boise, Idaho	0–2″	0.23 ±0.006	20.0±0.6	22.0	
		0.26 ±0.006	23.0±0.6		
	2–6″	0.012±0.002	3.1±0.6	3.5	26
		0.015±0.002	4.0±0.6		
Des Moines, Iowa	0–2″	0.31 ±0.007	23.0±0.5	23.0	
		0.31 ±0.007	23.0±0.5		
	2–6″	0.028±0.002	7.6±0.7	7.1	30
		0.024±0.003	6.6±0.7		
Detroit, Mich.	0–2″	0.26 ±0.006	20.0±0.5	20.0	
		0.27 ±0.006	20.0±0.5		
	2–6″	0.038±0.003	7.3±0.5	7.8	28
		0.044±0.003	8.4±0.6		
Grand Junction, Colo.	0–2″	0.10 ±0.001	7.8±0.1	7.0	
		0.091±0.001	7.1±0.1		
		0.11 ±0.019	8.2±1.4		
		0.070±0.013	5.1±1.0		
	2–10½″	≤0.002	≤0.45	≤0.48	7
		≤0.002	≤0.51		
Jacksonville, Fla.	0–2″	0.11 ±0.009	7.3±0.6	7.3	
	2–6″	0.013±0.004	2.7±0.9	3.4	11
		0.020±0.005	4.0±1.0		
Los Angeles, Calif.	0–2″	0.12 ±0.008	6.9±0.5	7.5	
		0.14 ±0.009	8.0±0.5		
	2–7″	0.009±0.002	3.3±0.9	2.8	10
		0.006±0.002	2.2±0.7		
Memphis, Tenn.	0–2″	0.27 ±0.006	15.0±0.4	15.0	
		0.26 ±0.006	15.0±0.4		
	2–6″	0.028±0.003	6.5±0.7	6.6	22
		0.029±0.003	6.6±0.7		
New Orleans, La.	0–2″	0.24 ±0.006	8.8±0.2	8.6	
		0.22 ±0.006	8.3±0.2		
	2–6″	0.009±0.002	3.3±0.9	2.8	11
		0.006±0.002	2.2±0.7		
New York, N. Y.	0–2″	0.21 ±0.006	10.0±0.3	12.0	
		0.29 ±0.007	14.0±0.3		
	2–6″	0.072±0.004	14.0±0.8	14.0	26
		0.068±0.004	14.0±0.8		
Philadelphia, Pa.	0–2″	0.17 ±0.005	12.0±0.4	12.0	
		0.16 ±0.005	11.0±0.4		
	2–6″	0.029±0.003	7.3±0.8	6.8	19
		0.026±0.003	6.4±0.7		
Rapid City, S. Dak.	0–2″	0.29 ±0.006	20.0±0.4	22.0	
		0.34 ±0.006	23.0±0.4		
	2–6″	0.053±0.004	12.0±1.0	11.0	33
		0.045±0.003	10.0±0.7		
Rochester, N. Y.	0–2″	0.22 ±0.006	16.0±0.4	16.0	
	2–6″	0.013±0.004	2.5±0.4	2.5	19
		0.013±0.002	2.5±0.4		
Salt Lake City, Utah	0–2″	0.32 ±0.007	22.0±0.5	22.0	
		0.33 ±0.007	23.0±0.5		
		0.31 ±0.007	22.0±0.5		
	2–8″	0.016±0.002	5.7±0.7	5.8	28
		0.016±0.002	5.9±0.8		
Seattle, Wash.	0–2″	0.46 ±0.011	17.0±0.4	17.0	
		0.44 ±0.010	16.0±0.4		
	2–6″	0.051±0.007	9.4±1.2	9.5	27
		0.052±0.004	9.6±0.7		

The differential rates at which the fallout has been occurring probably are best measured by the so-called "pot collection" method. A bucket with vertical walls of appreciable height is placed out in the open and allowed to collect the total fallout for a given period including the rain, snow, dust, etc. The bucket is left out whether it has rained or not and covers the total fallout for a given period. Figures 6 and 7 give the curves so obtained for New York and Pittsburgh areas together with the estimated errors of measurement. It is interesting to note the changes in slope and to correlate them with the occurrence of test activitis and the relatively short-lived troposheric fallout. The minimum slopes which appear during quiet periods when no one is testing are the stratospheric fallout of which we have spoken and these slopes when we have enough pots operating all over the world will, when taken together with the results of the measurements of the amounts of radiostrontium and radiocesium in the stratosphere, give an accurate value for the stratospheric residence time and settle the mixing question.

In addition to the intensity of fallout, the question of the fraction of the radiostrontium, and, for tropospheric fallout, the radioiodine of eight-day half-life, that is in assimilable form is an important one. So far most fallout strontium appears to be completely water soluble and therefore most assimilable, though continued tests on this point should be made. Direct leaf pick-up of course promotes assimilation of the strontium because the plant differentiation against strontium when it assimilates it from soil thus is avoided. Another factor is, of course, the concentration of available calcium in the soil. By available calcium we mean calcium which is available to plants and not the total calcium in the soil. It is known that soils which are high in available calcium produce plants of lower radioactive strontium content; that is, the radioactive strontium to calcium ratio in the plant is lower as a direct consequence of the lower concentration of radiostrontium in the available soil calcium. In addition, as mentioned previously, plants tend to prefer calcium to strontium with a discrimination factor of about 1.4. Sheep which grow in certain areas of Wales have shown concentrations in their bones approaching 150 Sunshine Units, while sheep and cattle growing in the U. S. have hardly ever exceeded one-fifth of this. The Welsh soil in certain areas is very low in calcium and as a result grows grasses of high radiostrontium content. Of course, it is clear that fertilization with calcium will immediately relieve this difficulty, but in the absence of such fertilization, the question is: How serious is the effect of calcium deficiency in promoting strontium-90 pick-up through the food chain?

As was remarked earlier, there is an averaging which occurs in food distribution systems and calcium deficient soils are naturally rather poor producers and as a consequence the weight of the food so produced is less than for a good well fertilized, well balanced soil. This factor reduces the flow into the general food system of material of exceptionally high strontium-90 content. It therefore will probably be sufficient to consider the radiostrontium of milk, since milk is the main source of calcium, in order to test for the radiostrontium content of the food in given areas. Direct measurements have shown that a factor of five encompasses the total variation due to all factors including calcium deficiencies in acid soils.

The general intake must depend on the food distribution pattern and the relatively small fluctuation in milk contents must reflect this. The number of individuals who rely totally on the food output of soil of very low calcium content is very small indeed, but it must be true that these individuals if they grew up on such a provincial, isolated farm would have as much as ten to 50 times the normal average strontium-90 content. The normal calcium concentration in soils in the United States is about 20 grams per square foot for the top 2.5 inches and about the poorest soil known has about 0.4 gram available calcium per square foot for the top 2.5 inches—a deficiency factor of 50.

It is clear from a detailed examination made by the author for people living in calcium deficient areas with normal food distribution patterns, that a factor of 5 is about as large an effect as can be expected from a fifty-fold deficiency of calcium in the soil. The food from outside of the calcium deficient area reduces by a factor of about ten the increase in strontium-90 pick-up rate which would be expected from the calcium deficiency in the soil if people lived entirely off the soil for their whole growing period of 20 years or so.

The food of lowest strontium-90 content is fish flesh, because of the great dilution the fallout receives by the hundred meters of sea water above the thermocline which rapidly mix with the fallout within a few hours or days. This means that

the specific concentration of radioactive strontium, or any other fallout constituent in sea water, is relatively very much lower than it would be in soil. For example, 100 meters of sea water has 370 grams of dissolved calcium per square foot as compared to the average of 20 grams per square foot for the top 2.5 inches of soil which absorbs and holds the fallout radiostrontium. Therefore, in principle sea food and fish are lowest among foods in content of radiostrontium fallout.

IV. EFFECTS OF CONTINUED TESTING AND GENERAL CONCLUSIONS

In summary, then, we see that the present body burden of strontium 90 from atomic weapons tests in the United States corresponds to the radiation dosage to the bones which would result from a few hundred feet increase in altitude, and the present vital statistics show no observable effect on the occurrence of bone cancer or leukemia of much larger changes in altitude. The tolerance figure of 100 Sunshine Units, or 0.1 of a microcurie for an average individual, or 100 micromicrocuries per gram of body calcium, that is recommended now is about two hundred times the present level for new bone in the U. S., and it will be exceeded by fallout from weapons tests in any foreseeable circumstances.

The distribution of strontium–90 burdens among individuals *for a given locality* will be a normal error curve with a standard deviation of about one-third of the average concentration. This means that about one individual in 300 will have more than twice the normal average value for a given locality, and that about 1 in several million will have three times the average value.

The effect of locality is more important, however, particularly in the effect of calcium deficiency in the soils. Careful consideration of this question indicates that there will be very few individuals who show a strontium–90 content which is strictly inversely proportional to the available calcium concentration of the soil in their region. This is due to the fact that food distribution systems automatically average over a wide area and people assimilate their calcium slowly. Most people drink milk and eat cheese and other calcium-bearing foods from a rather wide area, and this effect reduces by an estimated factor of ten the potential effect of calcium deficiency in the local soils.

On the basis of laboratory experiments the human body concentration of strontium–90 at equilibrium will be between 13 and 30 times less than that in the top soil. The present data indicate that the higher figure is closer to the truth, and so we will be conservative in taking the figure of 20 for this ratio. Therefore, the present burden of 50 Sunshine Units in the top soil of the United States may eventually lead to as much as 2.5 Sunshine Units in the human bones, but more likely will lead to about 1.7.

Of course, as testing continues and more fallout occurs, the levels will rise. The strontium-90 that still resides in the stratosphere at the present will fall out according to our expectations at a rate which just about compensates for the decay of the material already deposited, so that no great additional increase from this source is to be expected from weapons fired in the past. If the testing should continue at about the same rate as it has averaged over the last five years, then we should at equilibrium, after an infinite time, approach a level of 8 times the present rate, since the average life of strontium-90 is 40 years. This assumes that the future testing will be conducted so as to give in each future five-year period the same as the last five have. And so we would expect in the United States at that time an average human strontium-90 concentration of 20 Sunshine Units with the conservative factor of 20 between the top soil concentration and the concentration in human bone, or 5 Sunshine Units if the factor of 80 is used. In other words, in the United States something between 5 and 20 Sunshine Units would be the equilibrium concentration of human bones if testing continued indefinitely at the average rate of the past five years. This level would be approached only after a few decades. After 28 years the level would be half of this equilibrium value, and after another 28 years, 56 years total, from an arbitrary beginning which we have set as 1952, we would expect in the year 2008 three-fourths of the equilibrium figures. So somewhere between 4 and 15 Sunshine Units of strontium-90 in human bones in the United States might result from the present type of testing being continued for the next 50 years.

In those certain areas in the world where the soil is low in calcium, this level might go five-fold higher. At the present rate of testing we might indeed approach the figure of 100 Sunshine Units, the tolerance limit for large populations, at the beginning of the 21st century for these certain limited regions in

the world. The observed conditions in these regions could be relieved, however, by fertilization of the soil with calcium, either calcium nitrate or lime being used, as appropriate from other considerations.

The Sunshine Project continues to study the problems of world-wide fallout—the stratospheric inventory of radiostrontium and radiocesium, the occurrence of these isotopes in the soils and water and the biosphere all over the earth, the biological effects at certain levels of contamination with strontium-90, and to a lesser degree with cesium-137, and the possible genetic effects of the low gamma ray dosages associated with world-wide fallout from atomic tests. All of these are studied not only with the point in mind of devising methods of protection against atomic warfare, but also with the thought of possible application in the remote event of industrial accidents which may happen in connection with certain of the peaceful uses of atomic energy, particularly atomic power. Certainly an understanding of the basic principles of world-wide fallout is applicable to the control and safe-handling of isotopes. All of this is done in collaboration with the United Nations Scientific Committee on the Effects of Atomic Radiation, and it is to be hoped that as the data appear all of the countries in the world will join together in this international effort to understand better the effects of the great new fact in life, the nuclear atom.

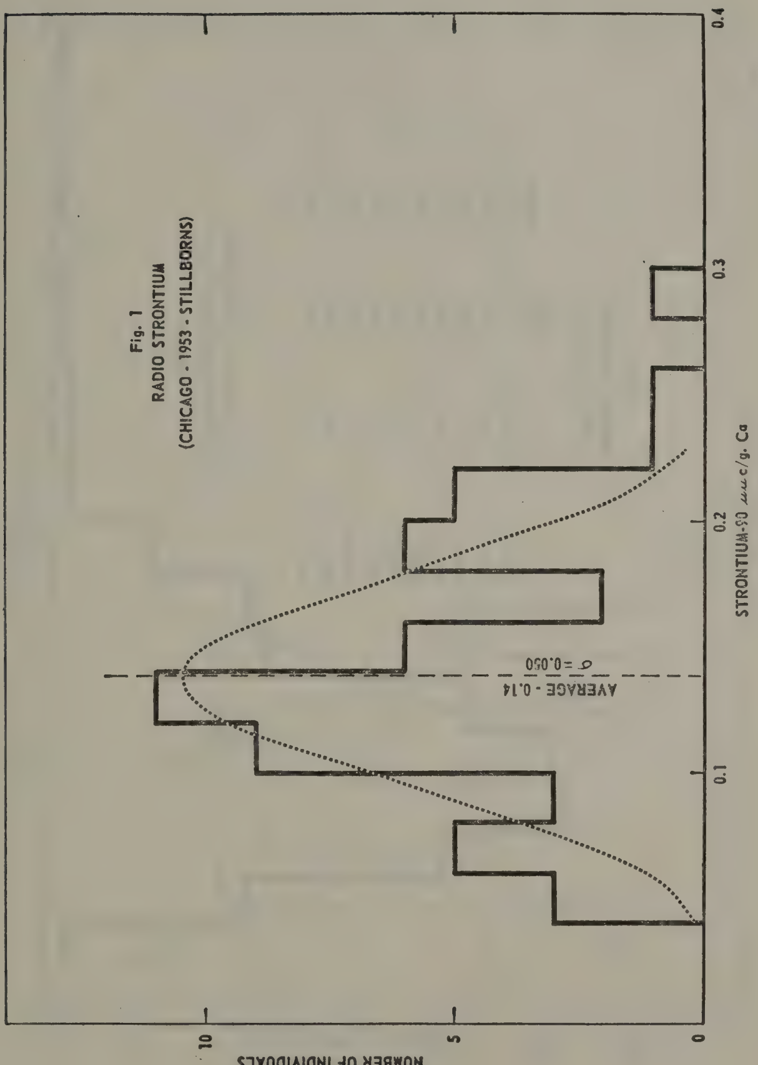


Fig. 1
RADIO STRONTIUM
(CHICAGO - 1953 - STILLBORNS)

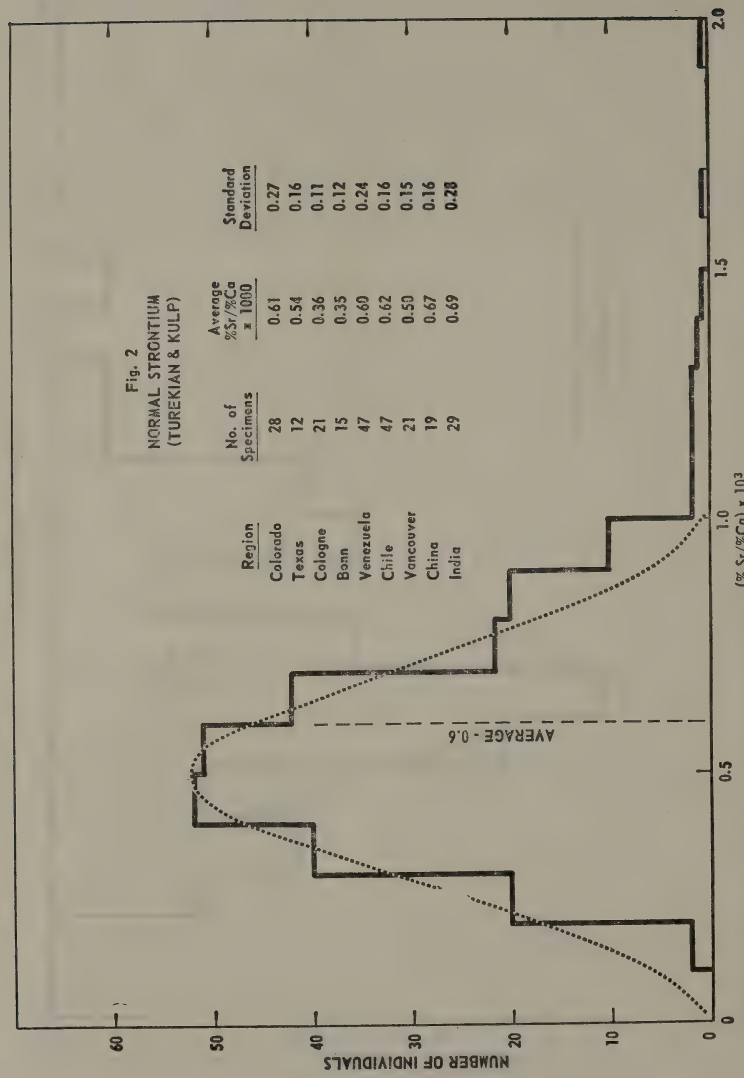


Fig. 2
NORMAL STRONTIUM
(TUREKIAN & KULP)

Region	No. of Specimens	Average %Sr/%Ca x 1000	Standard Deviation
Colorado	28	0.61	0.27
Texas	12	0.54	0.16
Cologne	21	0.36	0.11
Bonn	15	0.35	0.12
Venezuela	47	0.60	0.24
Chile	47	0.62	0.16
Vancouver	21	0.50	0.15
China	19	0.67	0.16
India	29	0.69	0.28

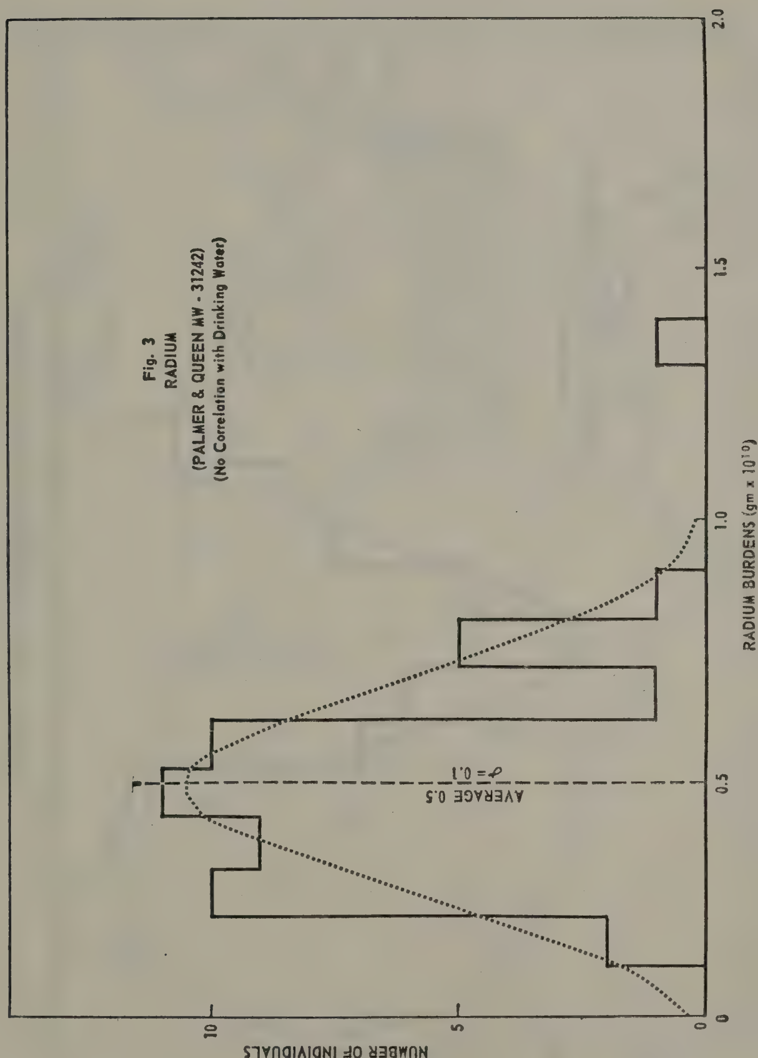


Fig. 3
RADIUM
(PALMER & QUEEN MW - 31242)
(No Correlation with Drinking Water)
AVERAGE 0.5
σ = 0.1
NUMBER OF INDIVIDUALS
0
5
10
RADIUM BURDENS (gm x 10^10)
0
0.5
1.0
1.5
2.0

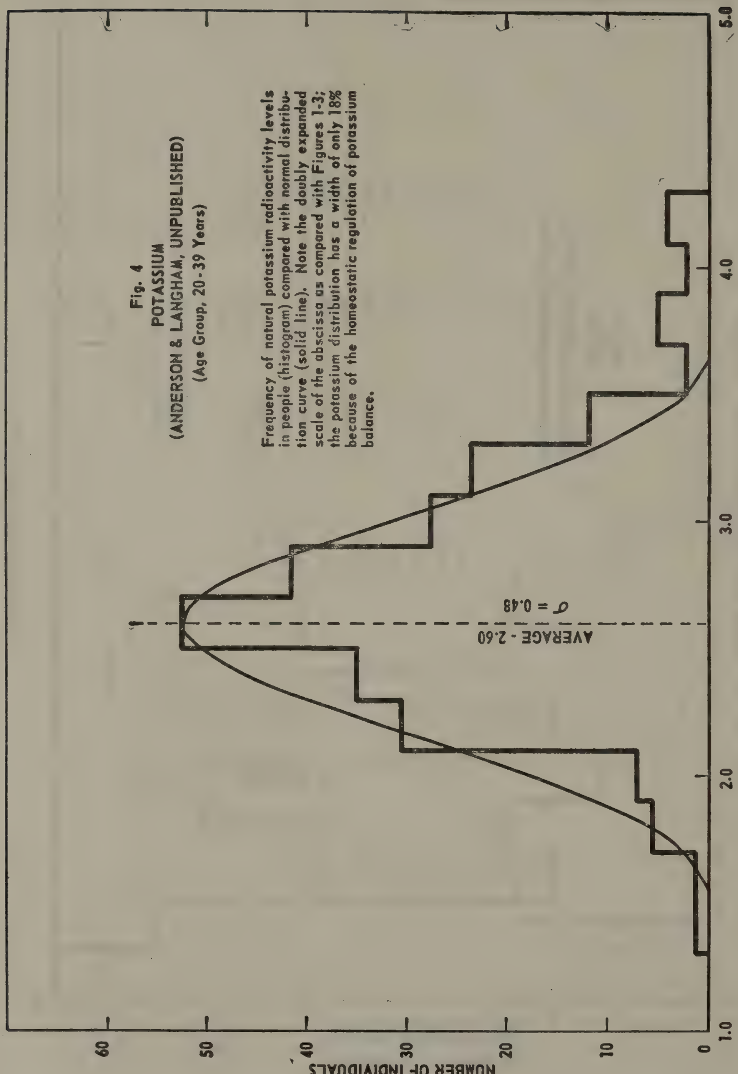
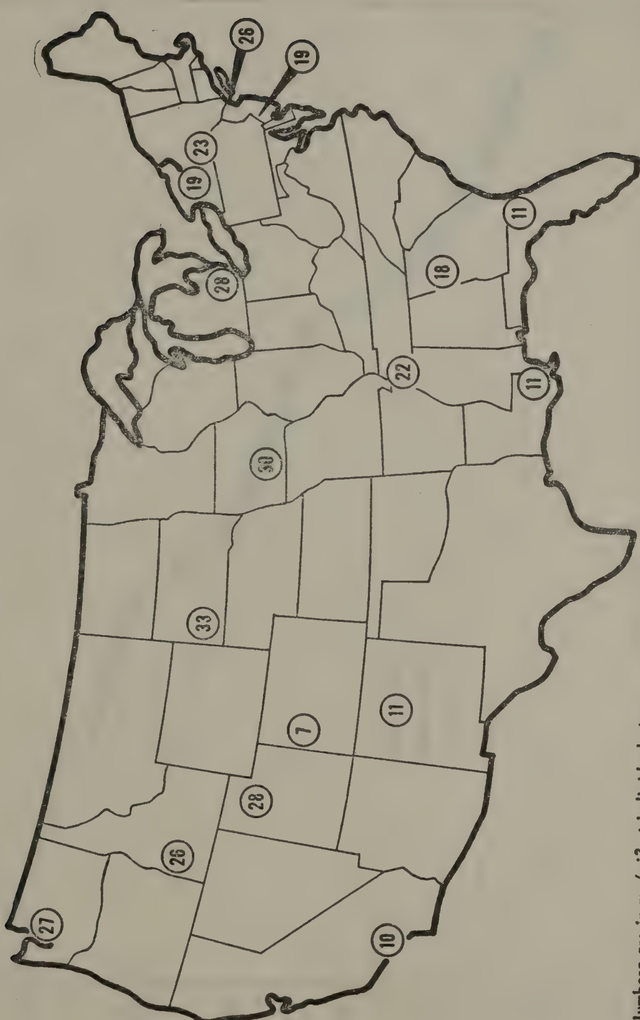


Fig. 4
POTASSIUM
(ANDERSON & LANGHAM, UNPUBLISHED)
(Age Group, 20-39 Years)

Frequency of natural potassium radioactivity levels in people (histogram) compared with normal distribution curve (solid line). Note the doubly expanded scale of the abscissa as compared with Figures 1-3; the potassium distribution has a width of only 18% because of the homeostatic regulation of potassium balance.

Fig. 5

Sr^{90} IN U. S. SOIL (HASL - OCT. 8, 1956) (HCl EXTRACTION METHOD)



Numbers are in mc/mi^2 at individual site.

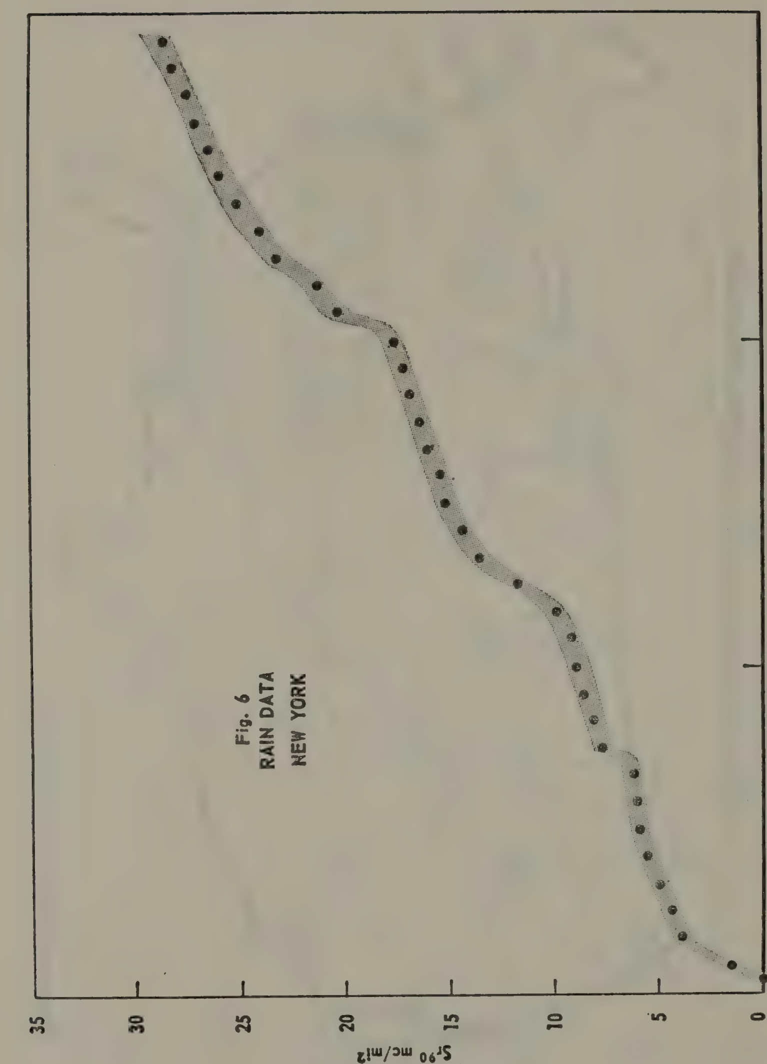


Fig. 6
RAIN DATA
NEW YORK

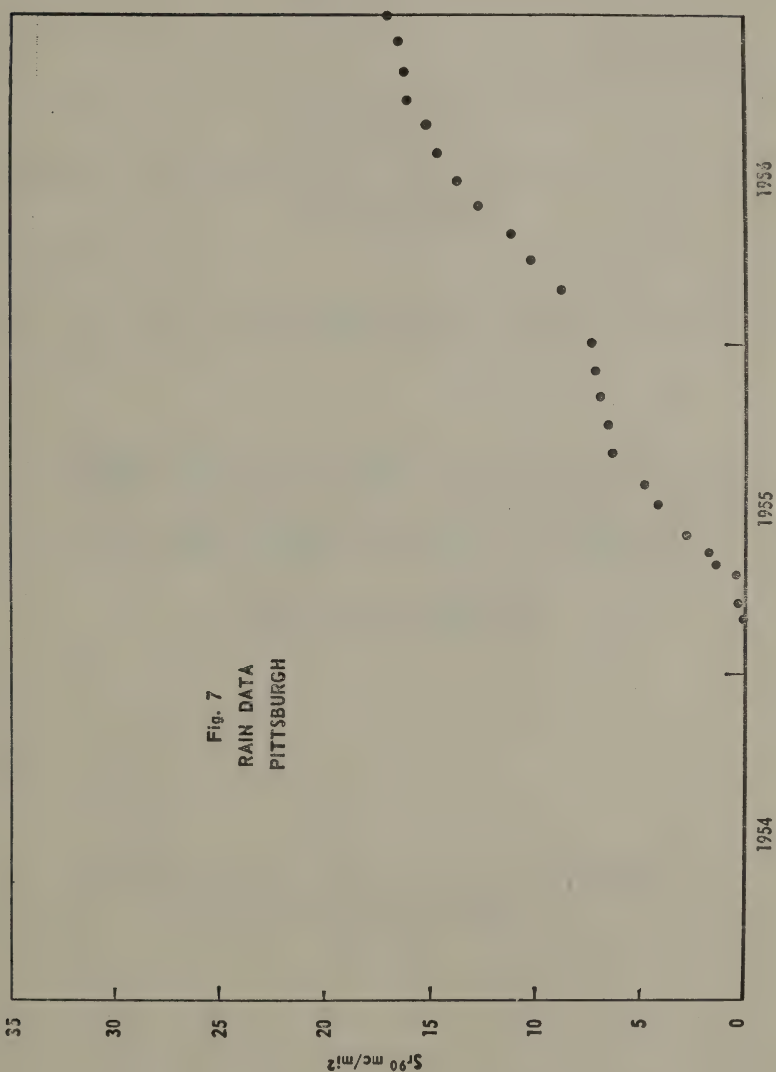


Fig. 7
RAIN DATA
PITTSBURGH

Appendix 2

British Statements, Reports, and Articles on Fallout and Radiation Effects

MEDICAL RESEARCH COUNCIL

THE HAZARDS TO MAN OF NUCLEAR AND ALLIED RADIATIONS

Presented by the Lord President of the Council to Parliament by Command of Her Majesty June 1956

93299 O—57—pt. 2——35

To The Most Honourable
THE MARQUESS OF SALISBURY, K.G., LORD PRESIDENT OF THE COUNCIL

My Lord,

On the 29th March, 1955, the Prime Minister, through you, requested the Medical Research Council to appoint an independent committee to report on the medical aspects of nuclear radiation, including the genetic aspects. The Council accordingly appointed a committee, under the chairmanship of Sir Harold Himsworth, and this body has now reported.

The report has been accepted by the Medical Research Council, and I have been authorised to transmit it to you with a view to its presentation to Parliament. It is the wish of the Council that, in so doing, I should express their high appreciation of the care, thought and ability which all members of the Committee have devoted so freely to their most difficult and important task.

I have the honour to be, my Lord,
Your Lordship's obedient Servant,

LIMERICK,
Chairman,
Medical Research Council.

June, 1956.

THE HAZARDS TO MAN OF NUCLEAR AND ALLIED RADIATIONS

A REPORT TO THE MEDICAL RESEARCH COUNCIL

CHAPTER I

INTRODUCTION

1. In accordance with the undertaking of the Prime Minister given in the House of Commons on the 29th March, 1955, the Medical Research Council, in April of that year, appointed us members of a committee, under the chairmanship of Sir Harold Himsworth, to review the existing scientific evidence on the medical aspects of nuclear and allied radiations, and we have unanimously signed this report.

2. We held our first meeting on the 3rd May, 1955, and decided to carry out our enquiry for the most part through two panels, one of which undertook to consider the effects of radiation on the health of the individual, and the other the possible genetic consequences of radiation to the population as a whole as well as to the individual and his descendants. Sir Ernest Rock Carling, Professor A. Haddow, Professor A. Bradford Hill, Dr. J. F. Loutit, Professor W. V. Mayneord, Dr. F. G. Spear, Professor Sir Lionel Whitby and Professor B. W. Windeyer have served on the former panel, which has met nine times. Professor A. Bradford Hill, Professor K. Mather, Professor P. B. Medawar, Professor J. S. Mitchell, Professor L. S. Penrose, Sir Edward Salisbury and Professor C. H. Waddington have served on the latter, which has met eleven times. In addition, Sir John Cockcroft and Professor J. R. Squire have served on the main committee. Both panels have worked under the chairmanship of Sir Harold Himsworth and all papers have been circulated to every member of the committee. More than seventy specially prepared papers have been considered, some of them written by scientists not serving on the committee, and we have drawn widely on the relevant published material. Groups to consider special problems have been appointed as the need arose. We have also taken into account relevant work carried out under the auspices of the Medical Research Council's Committee on Protection against Ionizing Radiations, and the recommendations and discussions of the International Commission on Radiological Protection. On the completion of the work of the panels, we have met together in full committee to consider our conclusions and to draw up this report. We have held four meetings of the whole committee during the period of our enquiry.

3. Throughout the course of our work, and in the preparation of this report, we have been greatly helped by our two scientific secretaries, Dr. W. M. Court Brown and Dr. T. C. Carter, of the scientific staff of the Medical Research Council. We are also very much indebted to Dr. R. H. L. Cohen and to Dr. Joan Faulkner, of the Council's headquarters staff, who have been responsible for co-ordinating the work of the panels and the various special enquiries that we have initiated.

4. The immediate occasion for the Government's request to the Medical Research Council to set up this Committee was the widespread public concern

about the long-term effects of nuclear weapon testing. This is only one aspect, however, of the much larger problem arising from the increasing use of ionizing radiations. It is already apparent that the future development of our civilisation is closely bound up with the exploitation of nuclear energy. At present, the potential hazards from its possible military uses overshadow in many people's minds the vast potentialities for good of this new source of power. The hazards to health are qualitatively the same, however, whether they arise from nuclear weapons or from the use of ionizing radiation for peaceful purposes. The difference is one of degree and intensity only. As with other sources of energy that man has harnessed to his service, the use of ionizing radiation necessarily entails risk ; but the risk is controllable within limits that he can accept. It is the purpose of this report to indicate the nature of the risks and the extent to which they can be controlled.

5. Our purpose has been to give as firm a guide as the evidence allows to informed opinion in the country as a whole, and more especially to those with whom lies the responsibility for practical decisions of policy. This has laid on us the duty of drawing more precise conclusions than we might wish to do on purely scientific grounds, and we feel bound to point out that in the course of our enquiry we have become increasingly aware of the impossibility, in the present state of knowledge, of coming to final conclusions on many questions of importance in the subject under study. Nevertheless, because of the many and urgent problems on which action cannot be delayed, we have felt it incumbent upon us to attempt to give guidance to the best of our ability. It is inevitable that, with the advance of knowledge, many of the views which we have expressed will come to require amendment, but we feel reasonably confident that the general picture which we have drawn is unlikely to be found grossly inaccurate in perspective or scope.

6. We wish to remind those who read this report that human populations have always been exposed to ionizing radiation, and that it is the scale and not the nature of the hazard which is new. Moreover, our remarks in many respects can be applied only to large populations living under conditions similar to those prevailing in this country. A technically advanced community, such as our own, is likely to be exposed to a greater risk from the industrial and medical uses of atomic energy. These risks have to be weighed against the established benefits derived from the use of ionizing radiation in industry and medicine, and against the benefits likely to be conferred in the future.

7. We have thought it helpful to the general reader to follow this introduction with a brief account of radiation and its mode of action on living cells (*Chapter II*). The types of radiation and the units in which they are measured are described, and the chapter concludes with an outline of the way in which radiation acts on living tissues.

8. In *Chapter III* consideration is given to the effects of radiation on the health of the exposed individual. A brief review of the available sources of information is followed by an account, first, of the clinical manifestations which may occur within a short time of exposure, and, secondly, of the effects which may appear a considerable time, perhaps many years, afterwards. The chapter includes a discussion of the evidence from a detailed study of the relationship between radiation dose and the incidence of leukaemia in patients suffering from a particular disease. This study was undertaken at our request by members of the Medical Research Council's staff, and we should like to thank Dr. W. M. Court Brown and Dr. R. Doll, who carried out the work, for the great help that they have given us. The full results of the enquiry are to be published separately and are summarised in Appendix B.

9. *Chapter IV* of the report opens with a short account of the biological processes controlling the hereditary constitution in human beings, and proceeds to a description of the way in which radiation might affect the genetic structure of human populations. An attempt is then made, in terms of the incidence of certain specific grossly harmful conditions, to assess the consequences for the individual and society of increasing the rate of mutation, and to define the levels of dose which might be expected to bring about such an increase.

10. In *Chapter V* the contributions made to the present level of radiation by naturally occurring radioactivity and by the medical, industrial and other uses of ionizing radiation are reviewed. Many new data have been obtained, and an investigation has been initiated at our request, and is still in progress, to establish more precisely the contribution from various diagnostic and therapeutic procedures (Appendix K). An assessment is then made of the results of contamination from the fall-out from atomic and thermonuclear test-explosions, and the chapter concludes with a brief description of the nature of nuclear warfare.

11. *Chapter VI* sets out our assessment of the hazards of ionizing radiation on the basis of the evidence put forward in the earlier chapters and proceeds to a discussion of the dangers from radiation in peace and war.

12. *Chapter VII* contains a summary of our report which is followed by a statement of our conclusions.

13. The highly technical nature of this report has made it necessary to devote some space in each chapter to a description of generally accepted scientific theory in terms comprehensible to the general reader. No attempt has been made to prepare a bibliography for these parts of the report. Where new material has been drawn upon or controversial topics are discussed, the evidence has been set out in greater detail in appendices, to which lists of selected references to published work have been attached.

14. It will be evident to any reader of this report that, at the present time, there are many large and serious gaps in our knowledge of the medical and biological effects of ionizing radiation. If the potentialities for good are to be exploited with confidence and safety, it is necessary that these gaps should be filled. Much research on many broad fronts will be required. Given the necessary facilities, there is no reason to doubt that the information can be obtained ; and we attach the greatest importance to the recommendations for future work that we have been invited to submit for the consideration of the Medical Research Council.

CHAPTER II

THE NATURE OF RADIATION AND ITS ACTION ON LIVING CELLS

Introduction

Discovery of X-rays

15. The transference of energy from sun to earth by radiant heat and light was already well recognised when in 1895 the discovery of X-rays revealed something quite novel—namely rays which had the power of penetrating normally opaque objects. The power of penetration varied in relation to different tissues of the body, and this variation enabled shadow pictures of internal structures to be seen, and so laid the foundation of the first great use of the new discovery. Almost by accident it came to be recognised in the next few years that part of the radiation was absorbed in the tissues, with the production of physico-chemical changes which could lead to biological damage.

Discovery of natural radioactivity

16. The production of X-rays was soon followed by the discovery of natural radioactivity. It was found that compounds of certain heavy elements in the earth's crust, such as uranium and radium, spontaneously emitted rays which had similar properties to X-radiation, although they were of different penetrating power. Later, rays were identified which reached the earth from outer space and these were named 'cosmic rays' *.

Disintegration of radioactive elements

17. The radiation emitted from radioactive elements is due to spontaneous disintegration of their atoms, with the production of one or more types of radiation and of a new element lighter in weight than the original one. A radioactive element, such as radium, may be regarded as a population of atoms, each of which has a length of life ending in spontaneous break-up of the atomic nucleus with emission of radiation, partly in the form of a stream of particles and partly as wave-propagated radiant energy. In this way the amount of radium gradually diminishes. While the moment of disintegration of any particular atom is unpredictable, the rate of decay of the population of atoms as a whole follows a strictly constant rule. Thus, any group of radium atoms decays to half its original number in a period of about 1,600 years. This period, called the half-life, varies widely for different radioactive substances but for each it is constant and characteristic. Sometimes the new element formed by atomic disintegration is itself unstable and a cascade of successive disintegrations occurs, each with the emission of one or more types of radiation, until finally a stable non-radioactive substance is formed.

Radioactive isotopes

18. After much pioneer work, dating from Rutherford's experiments as early as 1919, it was found possible in the nineteen-thirties to turn many normally stable elements into unstable versions of their original form, by treating their nuclei with suitably energetic radiations, and so to produce artificial radioactive substances. At first it was only possible to make these

* For a description of cosmic rays see paragraphs 192–193.

artificial radioactive substances under very special conditions and on a very small scale. The expansion of nuclear research during the last war, however, led to the development of the nuclear reactor, by means of which it became possible to create radioactive substances in very large quantities either directly or as by-products of the fission* processes in the reactor itself. Both natural and artificial radioactive substances are now commonly called radio-isotopes.

Types of Radiation and Units of Measurement

19. Several different kinds of penetrating radiation are known, of which the common types are the following:

alpha particles: These are the nuclei of helium atoms and are swiftly moving particles of high energy, carrying a positive electric charge. They have little power of penetration, passing into soft tissues for only small fractions of a millimetre, and irradiation of the body from outside with alpha particles is consequently of little significance. They may, however, affect living tissues when they arise from radioactive materials actually within the body, and, in sufficient quantity, they are then biologically very destructive.

beta particles: These are fast-moving energy-carrying particles (electrons) of very small mass with a negative charge. The amount of energy that they carry may vary considerably and their power of penetration will vary accordingly. In general, beta particles are more penetrating than alpha particles and can traverse distances of up to about a centimetre in soft tissues. For this reason they are valuable therapeutically, and radioactive substances emitting beta radiations are used for the destruction of superficial tumours. For the same reason heavy doses from outside the body can damage the superficial tissues and, if beta-emitting substances are ingested, destructive effects within the body may be produced.

gamma rays: These are electro-magnetic radiations of high energy emitted by atomic nuclei. Like alpha and beta particles they are produced in the process of natural or artificially induced atomic disintegration. Gamma rays have great penetrating powers in comparison with alpha and beta particles and the more energetic gamma rays can traverse the whole body with relatively little absorption. As a result, almost the whole thickness of the body may be irradiated by gamma radiations and this is a deciding factor in producing the general illnesses which may follow this type of irradiation. The properties of gamma rays are essentially similar to those of X-radiations but in general gamma-rays have an energy and penetrating power corresponding to the more penetrating X-rays produced at such extremely high voltages as several million volts.

X-rays: These are similar wave-propagated radiations, which are usually produced artificially by electrical machines and which are widely used both diagnostically and therapeutically in medical radiology. They vary considerably in their penetrating power, according to the electrical energy used in their production. The biological effects of X-rays are brought about by high-energy electrons, which are liberated in the tissues during the passage of the rays, so that the biological action of X-rays and beta particles is essentially the same.

neutrons: These are normal constituents of atomic nuclei but may be liberated with considerable energy. They carry no electric charge and

**Fission:* The splitting of the nucleus of a heavy atom into two roughly equal parts with the release of a large amount of energy.

are therefore not repelled by the charged nuclei of atoms, but enter into them to build up unstable structures which often disintegrate with the production of artificial radioactivity. Fast neutrons act chiefly by collision with the hydrogen of the water and of the other compounds which the tissues contain, the resulting 'recoil hydrogen nuclei' somewhat resembling alpha particles in their action. The initially fast neutrons are gradually slowed down in the tissues and may then bring about biological effects by interaction particularly with nitrogen. They may also be captured by hydrogen, thereby releasing energetic gamma rays.

Ionization

20. These several types of radiation vary not only in their powers of penetration but also in relation to the number of electrically charged atoms and molecules, called ions, that they leave in their tracks as they pass through tissues. For this reason they are collectively known as ionizing radiations. It is the production of electrically charged particles, or ions, which is mainly responsible for initiating the physico-chemical changes in living tissue that lead ultimately to the production of overt radiation damage. The efficiency of a given dose of radiations in producing biological effects can be related to the numbers of ions produced per unit length of track.

Intensity of dose and length of exposure

21. The biological effects of radiation are closely related to the dose, or quantity, of radiation received. An analogy can be drawn with the effects of ultra-violet light on the skin in producing sunburn. It is well known that these effects depend on two main factors, the brightness of the sunshine and the length of the exposure to it. Similarly, the effects of radiations such as gamma rays, X-rays or beta rays are determined by the same two factors, the intensity of the radiation and the period of exposure. Radiation may be regarded as consisting of small units of energy called 'quanta', and the intensity of the beam of a given kind of X- or gamma rays is simply a measure of how many such quanta are striking a particular area in a given time. The dose of radiation might therefore be described as the energy which is absorbed in the small mass of tissue upon which the radiation impinges. Living tissues, however, are not inert. After some types of damage by radiation, repair processes take place and the rate at which the dose of radiation is given becomes an important factor in determining the observed biological effect. Thus, if a dose of radiation is spread out over a very long time, for example many years, the response may be very much smaller than or even quite different from that which would occur if the same amount of radiation were given in a very short time. On the other hand, with some well known forms of biological damage produced by ionizing radiations, recovery does not occur. The production of gene mutation is perhaps the most important example of this latter type of change.

Measurement of dose: the roentgen

22. The difficulty of making satisfactory measurements of the dose of radiation has been overcome by making use of the changes of electrical conductivity which are brought about in air when ionizing radiations pass through it. This particular method of measurement is used, not only because it is technically convenient but also because the atomic composition of air or water is in this respect essentially similar to that of the body, the

absorption of X-rays taking account only of the kind of atoms present and not of their particular chemical combinations. The unit of dose which has hitherto been adopted internationally is called the roentgen,* which is abbreviated to the letter 'r'; for very small quantities of radiation the milliroentgen (0·001 r) is often used as the unit. The intensity of radiation to which we are ordinarily exposed from our natural surroundings is about 0·1 r per year.

Measurement of radioactivity : the curie

23. It has been seen that the biological effects of radiation depend upon the amount of energy which is absorbed in the tissues. At each radioactive disintegration, an atom emits a certain amount of energy in the form of high-speed particles and gamma rays. The total rate at which the tissues are irradiated therefore depends on how many atoms disintegrate per second. In considering the effects of radioactive materials actually within the tissues, use is accordingly made of units of radioactivity which depend on the number of atomic disintegrations per second. Based originally upon the rate of disintegration of radium, the unit of radioactivity is called the curie and represents the amount of an element in which $3{\cdot}7 \times 10^{10}$ disintegrations occur per second. This is too large an amount of radioactivity for most biological work and it is customary to measure the amounts of radioactivity in the body in microcuries (millionths of a curie). For some radioactive materials the maximum amounts which can be allowed in the body are of the order of only one microcurie or less ; but even this very small amount of material corresponds to many thousands of atomic disintegrations per second.

Relative biological efficiency

24. The destructiveness of the different types of radiation can also be expressed in relation to that of an equivalent amount, in terms of energy, of gamma rays. This measure is known as the relative biological efficiency (R.B.E.) and varies between different tissues.

Action on Living Tissues

25. The basis of the biological action of ionizing radiation is not fully understood. The consensus of opinion is that radiation acts primarily upon the cell and its constituents, and upon the complex chemical processes occurring in these, rather than upon the fluids in which the cell is bathed. It is thought that the processes associated with the formation of ions during the passage of radiation lead to changes in some of the highly organised molecular systems within the cell. These changes are probably brought about by highly reactive chemical intermediates liberated within the cell subsequent to the physical process of ionization.

Effect of radiation on organisms, tissues and cells

26. All living tissue is killed if exposed to large enough doses of radiation. Different types of organisms, tissues and cells, however, vary greatly in the amount of radiation which they can withstand. Among mammals the dose of X-rays to the whole body which will kill 50 per cent of an animal population varies from 200 to 1000 r—depending on the species ; for man it is thought to be between 400 and 500 r. There is also a wide variation in sensitivity between different animal tissues. For instance, in man the most sensitive tissues include the lymphatic glands, the epithelium of the small

* The roentgen shall be the quantity of X- or gamma radiation such that the associated corpuscular emission per 0.001293 gramme of air produces, in air, ions carrying 1 electrostatic unit of quantity of electricity of either sign.

bowel, and the precursors of the blood cells which are situated in the bone marrow, whereas adult nerve and muscle tissue are comparatively insensitive. Variations in sensitivity also occur at different stages in the life cycle of a cell; for example, cells about to divide are often more sensitive than those in the resting state.

Repair processes

27. At dose levels below those which damage tissue irretrievably, the situation is modified by processes of repair; but a distinction must be drawn between true recovery, in which the damaged cells return to normal form and function, and the replacement of injured cells by those coming from outside the field of radiation. The latter is the more conspicuous form of repair after heavy radiation damage in the higher animals and leads to the original tissue being replaced by simpler unspecialised material or scar tissue. Repair processes within the individual cell are little understood and still largely a matter of speculation, but they must play an important part after low doses.

CHAPTER III

THE EFFECTS OF RADIATION ON THE HEALTH OF THE INDIVIDUAL

Introduction

28. Experience of the effects of ionizing radiations has been accumulating with increasing rapidity since the benefits which they may produce in the treatment of malignant disease first began to be appreciated. This experience has been limited, in the main, to the effects produced by the relatively large doses which it is often necessary to give to the area of the body under treatment. More recently, knowledge of the effects of very large doses to the whole body has been obtained as a result of the atomic bomb explosions in Japan. In this chapter it will be necessary to draw heavily on the information from these two sources in considering both the acute and the long-term effects of exposure to radiation, but the reader must bear in mind that such information is only indirectly relevant to the circumstances of ordinary civilian life, since doses of this magnitude would only be conceivable in the immediate vicinity of an accident in a nuclear reactor.

29. There is much less information about the possible effects of chronic exposure to very low doses of radiation, such as those to which special groups of workers may be exposed in the course of their occupations. At a time like the present, when nuclear energy is being intensively developed for civil use, the importance of obtaining such information cannot be exaggerated. The investigation which we have sponsored on leukaemia was undertaken in an attempt to obtain information on the relationship between the size of the dose of radiation and the incidence of the disease among patients with ankylosing spondylitis, so that conclusions might be drawn about the effects of lower doses. The investigation must be regarded, however, as only the first step towards this goal.

Sources of Information

30. Information about the effects of radiation on man has been derived from four main sources: radiotherapeutic experience; occupational experience, including that from accidents; experience from atomic bomb explosions; and animal studies.

31. *Radiotherapeutic experience.* Both X-rays and the gamma rays of radium have been used for many years in the treatment of disease, mainly in the treatment of cancer. Observation of patients receiving radiotherapy has yielded information on the general effects of radiation and on the effects produced in different tissues; and the therapeutic use of radioactive isotopes has provided data on the effects of radioactivity within the body.

32. *Occupational experience.* Information on the occupational hazards of radiation has been obtained from studies of three groups of workers: medical radiological workers, painters of luminous dials for watches and clocks, and miners working radioactive ores in the Schneeberg mines in Saxony and in Joachimsthal. The experience of these three groups serves to illustrate three different forms of radiation hazard. The radiological workers were exposed mainly to external irradiation by X- and gamma rays, and some developed

skin damage leading to skin cancer, or bone-marrow damage leading to severe diseases of the blood. The luminous-dial painters ingested paint containing the naturally-occurring radioactive elements radium, mesothorium and radiothorium, which are retained within the skeleton, and some developed bone tumours. The miners of Joachimsthal and Schneeberg worked in an atmosphere containing high concentrations of the radioactive gas, radon, and many developed lung cancer. The study of these three different hazards has contributed greatly to our knowledge of the harmful effects of radiation, and has provided data for the formulation of safety standards.

33. *Atomic bomb experience.* The atomic bomb explosions over Hiroshima and Nagasaki brought widespread destruction to these cities. Blast and fire caused most of the casualties, but about 15 to 20 per cent were caused by the gamma and neutron radiations emitted during the explosions. In 1946 the United States established in Japan the Atomic Bomb Casualty Commission, which has studied the immediate and the long-term effects of radiation from the bombs on the populations of both cities; the findings have been of great value in expanding knowledge on this subject.

34. *Analogous effects produced in animals.* The discovery that X-rays could produce changes in human tissues led investigators to study the effects of radiation on animals. As a result, it was established that radiation produces effects in animals similar to those observed in man and it thus became possible to make an experimental approach to the problem of radiation hazards. The knowledge thus gained has been drawn on freely in this report.

Factors Affecting the Severity of Radiation Injury

35. The harmful effects of radiation can be divided into those developing within a few weeks of exposure and those developing some considerable time, perhaps many years, afterwards. Illnesses which develop within a few weeks are sudden in onset and run an acute course, whereas those occurring some years after exposure develop insidiously.

36. The severity of radiation injury in any particular instance is determined by the interplay of several factors: the type and dose of radiation received, the duration of the period of exposure, the extent and part of the body which has been irradiated, and also the age of the person exposed.

The dose of radiation received

37. If the dose of radiation is a large one and is received by the whole body in the space of a few minutes, a severe and possibly fatal illness is likely to develop within a few hours, and certainly within a few weeks, of exposure. Some of those who survive this early illness may die several years later from one of the delayed effects of radiation, such as anaemia or leukaemia. Exposure of the whole body to smaller doses of radiation, over a period of months or years, will not cause the early illness, but there may still be a slightly increased risk of death from the delayed effects in later years.

The extent of the body irradiated

38. On the other hand, if only a fraction of the whole body is irradiated, as in radiotherapy, immediate general effects are rare, although some patients may develop a mild form of the early illness, known as radiation sickness. It is often necessary to give a large dose locally and there may be local reaction in the irradiated area with temporary reddening of the skin or blistering similar to that which occurs in sunburn. Delayed local effects

that may occur in these patients are scarring, less commonly necrosis and rarely the later development of cancer in the irradiated tissues. It is now apparent that there may be delayed general effects, a small proportion of patients in later years developing anaemia or leukaemia.

The part of the body irradiated

39. Experience has shown that there is a difference in the general effects of radiation according to the part of the body which is irradiated. Even quite a large dose of radiation given to a portion of a limb will usually produce no general ill effects whereas a similar dose directed to an equally large volume of tissue in the upper abdomen, for example, may produce severe immediate illness.

The type of radiation

40. The severity of effect produced by radiation may also depend to some extent upon the type of radiation concerned, since radiations differ in their powers of penetration and in their destructive effects. For example, fast neutrons are about ten times more potent than X- or gamma rays in causing cataract in the lens of the eye, although these three forms of radiation differ very little in their capacity to cause the early acute form of illness.

The age of the individual exposed

41. It has long been known to radiotherapists that young children are more likely than adults to develop reactions after irradiation. Further evidence on this point comes from a recent report on the inhabitants of the Marshall Islands, who were exposed to radioactive fall-out after the thermonuclear test explosion in that area of the Pacific Ocean in the spring of 1954. A consistently greater fall in the number of white corpuscles in the blood occurred among children than among adults, and a similar age-difference in response was noted also in regard to loss of hair.

The frequency of radiation effects

42. Reasonably good estimates have been made of the numbers likely to develop the acute illness under varying conditions of whole-body irradiation. Thus, every member of a population receiving a single whole-body dose of 500 r of gamma-rays would become ill shortly afterwards; if the dose were 150 r, only about half would do so; and, if it were of the order of 50 r, sickness would be extremely rare. It is much more difficult to assess the proportion likely to suffer from the delayed effects; all that can be said with certainty is that it would be small.

The Early Effects of Exposure to Radiation

43. The following description of the effects of a single heavy dose of gamma rays to the whole body is based on observation of the bomb-victims in Nagasaki and Hiroshima. It must be repeated that, in peacetime, exposure at this level could result only from an accident which would rarely, if ever, occur and that, even then, only those in the immediate vicinity of the disaster would be affected.

Effects of heavy dosage

44. The first effect of exposure of the whole body to a heavy dose of gamma rays of the order of 500 r is a sensation of nausea developing suddenly and soon followed by vomiting and sometimes by diarrhoea. In some people, these symptoms develop within half an hour of exposure; in others, they may not appear for several hours. Usually, they disappear after two to three days. In a small proportion of cases, however, the symptoms

persist; vomiting and diarrhoea increase in intensity; exhaustion, fever, and perhaps delirium follow; and death may occur a week or so after exposure.

45. Those who recover from the phase of sickness and diarrhoea may feel fairly well, although examination of the blood will reveal a fall in the number of white cells. Between the second and fourth weeks, however, a new series of ailments, preceded by gradually increasing malaise, will appear in some of those exposed. The first sign of these developments is likely to be partial or complete loss of hair. Then, from about the third week onwards, small haemorrhages will be noticed in the skin and in the mucous membranes of the mouth, which will be associated with a tendency to bruise easily and to bleed from the gums. At the same time, ulcerations will develop in the mouth and throat, and similar ulceration occurring in the bowels will cause a renewal of the diarrhoea. Soon, the patient will be gravely ill, with complete loss of appetite, loss of weight, and sustained high fever. Feeding by mouth will become impossible, and healing wounds will break down and become infected.

46. At this stage, the number of red cells in the blood is below normal, and this anaemia will increase progressively until the fourth or fifth week after exposure. The fall in the number of white blood cells, noted during the first two days after exposure, will have progressed during the intervening symptomless period, and will by now be reaching its full extent. The changes in the blood-count seriously impair the ability to combat infection, and evidence from Nagasaki and Hiroshima shows that infections of all kinds were rife among the victims of the bomb. Many of those affected die at this stage and, in those who survive, recovery may be slow and convalescence prolonged; even when recovery appears to be established, death may occur suddenly from an infection which in a healthy person would have only trivial results.

Effects of lighter dosage

47. The radiation effects described above are the most severe which can follow a single whole-body dose of 500 r of gamma rays and still allow some hope of survival; but at least half of a population so exposed would die. With smaller doses, fewer people would develop symptoms and the illness would become correspondingly less severe; thus, with a dose of 100 r, not more than about 15 per cent of the exposed population would be affected, the illness would be comparatively mild, and very few, if any, would die.

Effects of exposure to 'fall-out' in the vicinity of an explosion

48. The radiations considered above have been those occurring within one minute of the detonation of a nuclear weapon. These radiations have been called the 'prompt' radiations to distinguish them from those emitted by the radioactive dust, or fall-out, which settles over a wide area in the vicinity of an explosion. The fall-out may itself be active enough to cause radiation illness of a type similar to that described above and, in addition, it may contaminate and damage the skin with which it comes in contact.

49. Following the firing of a thermonuclear weapon in the region of the Marshall Islands, the fall-out on one island was so heavy that it was compared to snow, and the inhabitants received an estimated average whole-body dose of 175 r. This fall-out did not cause any deaths, but it did produce a mild illness with early sickness and diarrhoea, a fall in the number of cells in the blood, loss of hair, and some ulceration of the skin contaminated by radioactive material. The skin lesions, caused largely by the higher local

dose of beta-radiation emitted by the fall-out, appeared about two weeks after exposure on those parts of the body which had not been protected by clothing, and took the form of spotted areas of increased pigmentation, from most of which the skin peeled off as if it had been sunburnt. In about 20 per cent of cases, deeper ulceration of the skin occurred but all wounds healed satisfactorily.

Relationship between dose and incidence of effects

50. For the purposes of assessing risk and defining standards of safety, it is necessary to know the nature of the relationship between the dose of radiation and the effect induced. This relationship may be a simple linear one in which the incidence of the particular disease increases strictly in proportion to the dose received, or it may be a curvilinear one in which, with each successive and equal increment in dose, the incidence increases not by an equal but by a progressively greater amount. All the evidence suggests that the relation between dosage and radiation effects occurring within a few weeks of exposure is of the latter type, and that the curve shows a 'threshold' level, implying that a certain quantity of radiation must be exceeded before these particular effects are produced.

The Delayed Effects of Exposure to Radiation

51. Delayed effects of radiation which have been observed locally in tissues heavily irradiated are atrophy and fibrosis of the skin and underlying soft tissues, and sudden breakdown or necrosis of tissues such as bone and cartilage. In rare instances cancer has subsequently developed in the damaged tissues. Cataract has occurred if the lens of the eye has been irradiated. The delayed general effects of radiation which are known, are the development of severe anaemias and leukaemia; in addition, evidence is beginning to accumulate from observations made on animals that irradiation may cause some shortening of the normal life-span. In our report we have dealt in considerable detail with leukaemia, because experience in Japan following the atomic bomb explosions in 1945, and the results of our own investigation on the incidence of leukaemia among irradiated patients, have provided more precise information on the effects of different levels of exposure than is available for any other of the delayed effects.

52. The knowledge that long-term effects may be produced by radiation is in itself an insufficient basis for assessing the risk that any of them will develop as a result of a particular dose. For this purpose, it is necessary to estimate, from national mortality statistics, the incidence of the condition in the absence of exposure to radiation additional to that from natural sources, and then to compare this figure with the incidence of the same condition in a population that has been exposed to radiation. If an increase is demonstrated, the frequency with which the condition develops at different levels of radiation dose must be determined, and the relationship between the dose and the incidence of the disease must be evaluated. Only then is it possible to assess the hazards, if any, associated with the different uses of radiation.

INDUCTION OF LEUKAEMIA

53. Leukaemia is a disease in which uncontrolled over-production of the white blood-cells occurs. It is at present invariably fatal, although some forms may run a protracted course over many years. Several kinds of leukaemia are described according to the type of cell mainly affected. Usually, there is an increase in the number of the affected cells in the blood, associated with the appearance of immature forms of the cell in question. In some cases, however, the numbers in the blood may fall below normal

through failure to liberate the cells from their site of formation in the bone marrow ; the disease is then known as aleukaemic leukaemia.

54. In many countries the death rates from leukaemia have shown a steady rise in recent years. In 1920 the crude annual death rate from this condition for both sexes in England and Wales, for example, was 11 per million persons ; in 1954 it was 49 per million. Some of this rise has undoubtedly been due to an improvement in diagnosis but it seems probable that this is not the whole explanation and that, for a reason as yet unknown, there has been a real increase in the national death rate from leukaemia.

55. It is known that leukaemia may be induced in animals as a result of exposure to radiation. Case reports have appeared from time to time of patients who have developed leukaemia after exposure to radiation for the treatment of various diseases, and there have also been a number of reports of radiologists dying from leukaemia. Such isolated reports do not of themselves prove that the relationship is one of cause and effect, but the matter has now been put beyond doubt by a series of recent observations on the incidence of leukaemia under conditions in which an estimate could be made of the degree of exposure to radiation.

Leukaemia following a single exposure : atomic bomb explosions

56. The most recent information, for which we have to thank the United States National Research Council, covers all cases of leukaemia recorded by the Atomic Bomb Casualty Commission in Nagasaki and Hiroshima between January, 1947, and August, 1955. Vital statistics allow an estimate to be made of the number of cases of leukaemia that would have been expected to occur over a similar period in a Japanese population not exposed to radiation from the bombs but otherwise comparable to the surviving populations of Nagasaki and Hiroshima. Calculations have been made for the combined totals of the survivors of the explosions in both cities (Appendix A).

57. During the eight years from 1947 to 1954, about 25 deaths from leukaemia would have been expected in an unexposed Japanese population of the same size and having the same age and sex distribution as the combined populations of survivors from both cities. Over the same period, however, 91 proven and 14 suspected cases have been recorded among those present at the time of the explosion and still resident in one or other city at the time of diagnosis. The difference between the expected and the observed number of cases is so great that it is most unlikely to be due to chance.

58. The difference between the numbers expected and those observed becomes even greater if the most heavily irradiated survivors are considered separately. Only for Hiroshima are adequate details available of the distances from the centre of the explosion at which the individual survivors had been exposed. In the absence of radiation, it is unlikely that even one case would have occurred among the number of survivors less than 1,000 metres distant, yet 15 cases have been found. Further, there is a much higher incidence among those who developed the early acute illness than among those who had, at the most, only mild symptoms.

59. An examination of the incidence of leukaemia in relation to the distance from the explosion has been made for the survivors in Hiroshima, where the concentric distribution of the radiation was not affected to the same degree as in Nagasaki by the irregular distribution of the radio-active fall-out. The dose from the prompt radiation decreases as the distance from the explosion increases. In survivors who were 2,000 metres distant

or more, the incidence during the period January, 1947, to August, 1955, was about 2 cases in every 10,000 persons. Among those between 1,500 and 2,000 metres distant, the incidence was about 3 to 4 cases per 10,000 persons, and for those at the shorter distances of between 1,000 and 1,500 metres and under 1,000 metres it was respectively about 28 and 128.

60. To make an accurate estimate of the relationship between the dose of radiation and the incidence of the disease, one would have to substitute doses expressed in roentgen units for the distances from the centre of the explosion. It has not been possible to obtain reliable estimates of these doses, which should include not only the contribution from the gamma rays but also that from the neutrons emitted by the explosion and that from the radioactive fall-out. Tentative estimates of the gamma ray dose received by people standing in the open can be made from the information published in 1950 by the United States authorities in 'The Effects of Atomic Weapons'. These estimates suggest that the dose at under 1,000 metres would not be less than 1,400r, and at 1,250 metres about 350r. At 1,750 metres it would be about 50r, and at 2,000 metres about 8r. As a dose of 1,400r or more would kill everyone exposed to it, survivors who were within 1,000 metres of the explosion must have been heavily protected. An unknown proportion of the survivors at all the other distances must also have been protected to some extent because they were either indoors or, if outside, shielded by buildings. For this reason, it is not possible to indicate with any great confidence the average levels of dose received by survivors at different distances from the bomb and, in view of the uncertainty about the actual doses received by the exposed population, one cannot infer with certainty whether the relationship between dose and the incidence of leukaemia is a curvilinear or a linear one.

61. For the Japanese cases which occurred up to the end of 1954, the average length of the period between exposure to the bomb and the first appearance of symptoms was about 6 years. It is clearly important to determine whether there has been any tendency for cases to occur less frequently in subsequent years. The morbidity rate has therefore been examined year by year in both Hiroshima and Nagasaki, and it has been found that the recorded incidence has remained approximately constant in Hiroshima in the period 1948 to 1954, and in Nagasaki in the period 1950 to 1954 (Appendix A). This finding suggests that there is no sharply-defined peak year of occurrence, but that with this type of exposure the incidence of leukaemia rises, after a variable latent period, and then remains approximately constant up to at least the ninth year.

Leukaemia following repeated exposures : radiotherapy

62. Before 1955, there had been a report of leukaemia developing in two patients given X-ray treatment for ankylosing spondylitis. In 1955, two further publications directed attention to this possibility, and another reported the occurrence of leukaemia in young children who had been given X-ray treatment to the chest in infancy for suspected enlargement of the thymus gland. In an attempt to obtain further evidence on the occurrence of leukaemia as a delayed effect of irradiation, and in particular on the relationship between the dose received and the incidence of the disease, we have sponsored a survey of patients treated for ankylosing spondylitis with radiation.

63. Ankylosing spondylitis is a disease which affects chiefly the joints of the spine, and to a less extent other joints, particularly those of the pelvis and the shoulders. It usually starts in early adult life and is about six times more frequent in men than in women. It causes severe pain and reduced mobility and, unless treatment is given, the affected joints may gradually lose their freedom of movement and the back become progressively stiffer. In

93299 O—57—pt. 2——36

severe cases all spinal movement is lost, chest expansion is greatly diminished, and the movements of other major joints restricted. The popular description, 'poker back', is a very apt one.

64. Some patients with this condition are benefited by X-ray treatment, which is given to relieve pain and increase mobility and which may permanently halt the progress of the disease. As treatment usually takes the form of irradiation of the whole spine in one course of radiotherapy, it involves exposing a large section of the body directly to the X-rays. In some patients one course of treatment does not suffice, and further courses have to be given, either to the spine or to the major joints, or to both. Indeed, this group of patients was chosen for our investigation because the treatment is so extensive that it more nearly approaches whole-body irradiation than that given for any other non-malignant condition.

65. An analysis has been made of the hospital records of between 13,000 and 14,000 patients, all of whom had been treated with X-rays at some time during the twenty-year period 1935 to 1954. Thirty-eight of these patients developed leukaemia, an incidence of only about one-third of one per cent; yet calculations based on the national death rates over the same period show that even this low incidence is about ten times greater than would have been expected in the absence of irradiation. The possibility of such a difference being due to chance is so remote that we shall ignore it.

66. Caution is necessary, however, in interpreting this finding. It is not possible to conclude immediately that the increased number of deaths from leukaemia is related to the X-ray treatment, in the way that the increased death rates among previously healthy people in Hiroshima and Nagasaki can be attributed to exposure to the radiations from the bombs. The possibility has to be considered that death from leukaemia would, even in the absence of treatment by irradiation, be a more frequent occurrence among sufferers from ankylosing spondylitis than among the normal population, or alternatively that ankylosing spondylitis in some way increases a patient's susceptibility to irradiation.

67. By courtesy of the Ministry of Pensions and National Insurance, it has been possible to examine the records of a group of about 400 male patients with ankylosing spondylitis who had never at any time been treated with X-rays. The fact that no increased incidence of leukaemia was found in this group suggests that ankylosing spondylitis does not of itself predispose a patient to the development of leukaemia. To confirm this point, it would be necessary to examine the records of a much larger group of unirradiated patients; X-ray treatment is, however, so widely used for ankylosing spondylitis that it may be difficult to do this.

68. Clear evidence was, however, found in our main investigation for the existence of a relationship between the dose of radiation and the incidence of leukaemia. The dose was estimated in two different ways, firstly by calculating the total amount of energy absorbed in the whole body, and secondly by calculating the dose of radiation received in certain parts of the bone marrow. The first method demonstrated a curvilinear relationship between the incidence of leukaemia and the radiation dose, whereas the second method resulted in a linear relationship. Fortunately, over the range of doses likely to be met with in ordinary civil conditions, the difference between the two results is negligible. The theoretical implications of the two possible relationships are, however, very different and important and point the way to considerable future research. The data upon which the findings are based are summarised in Appendix B.

69. The average length of time between the first exposure to X-rays and the diagnosis of leukaemia was about six years. This period cannot be directly compared with that observed in the Japanese cases, as many of the patients had had several courses of radiation before leukaemia was diagnosed, and it is not known which particular course was the effective one or whether all the courses may not, to some degree, have affected the development of the disease. Nevertheless, it may be concluded from both series of cases that the latent period for radiation-induced leukaemia is shorter than for radiation-induced cancers.

Leukaemia following chronic exposure

70. We have no precise knowledge of the incidence of leukaemia under conditions of chronic exposure. It has been reported that, relative to the numbers at risk, there are about nine times as many deaths from leukaemia among American radiologists as among other American physicians. This figure is based on a study of the obituary notices published in the Journal of the American Medical Association from 1929 to 1948, in which both the professional occupation and the cause of death are usually reported. In about a quarter of the notices, however, the cause of death was not reported and thus a bias may have been introduced into the results of the study. A review of all the published papers on this subject shows that there may well be an increased death rate from leukaemia among American physicians as a whole, compared with the general population, and in particular among American radiologists, but it is not possible to estimate the extent of the increase with any certainty.

General conclusions on the induction of leukaemia

71. The results of the investigations carried out by the Atomic Bomb Casualty Commission in Japan, and of our own study of the occurrence of leukaemia in patients with ankylosing spondylitis, leave no doubt that ionizing radiations can induce leukaemia in man, and that the average latent period between exposure and the development of the disease is only a few years. In neither of these situations were the conditions of exposure similar to those of persons engaged in work associated with a possible radiation hazard. Those exposed occupationally tend to receive radiation in small doses over long periods, and it is not yet known whether the dose-response relationship based on short periods of heavy exposure is directly applicable to such conditions.

INDUCTION OF CANCERS

72. The evidence for the induction of cancers by radiation consists chiefly of reports of the occurrence of cases under circumstances which make it reasonable to suppose that some at least were radiation-induced, and of the apparently increased frequency of a particular type of cancer, itself rare in the normal population, in persons exposed to heavy doses of radiation. Most of the information comes from the case-records of patients treated with radiotherapy and from those of workers in certain special occupations who in the past received very heavy doses of radiation in the course of their work. It is noteworthy that tumours following radiotherapy tend to develop in tissue already severely damaged by radiation, and that, compared with leukaemia, a much longer period—up to 20 years or more—usually elapses between the first exposure to radiation and the clinical appearance of the disease.

Cancer of the lung

73. The mines of Schneeberg and Joachimsthal are rich in a variety of ores and, since the latter part of the last century, pitchblende, an ore containing radium and other radioactive elements, has been extensively worked there. It had long been known that the miners were liable to die in middle-life from a respiratory disease locally named 'mountain sickness'. It is now recognised that this condition is one of cancer of the lung and it is generally accepted that there is a strong connexion between the excessive mortality from this disease and the high radioactive content of the air of the mines. Investigations have suggested that, up to 1939, nearly one-half of the miners who had died had contracted lung cancer.

74. The first decay-product of radium is a gas, radon, which in its turn disintegrates, giving rise to a series of products, all of which are solids. Radon, being a gas, diffuses through the rocks containing the radium ore, and escapes into the atmosphere of the mines. The inhalation of radon is known to constitute a serious hazard, and the International Commission on Radiological Protection has advised that the concentration of this gas in the inspired air should not exceed 0·0001 microcuries per litre. A series of measurements of the radon content of the air of the mines, made between 1924 and 1939, showed that the concentration of radon must then have been on the average about thirty times greater than the maximum permissible level since laid down. The serious hazard incurred in breathing such an atmosphere comes, not only from the radon itself but also from its solid daughter-products which, being attached to dust particles in the atmosphere, may be retained in the chest and may irradiate the tissues of the lungs for long periods.

75. The average latent period for the induction of lung cancer in these miners was about 17 years, and calculations have shown that the dose to the lungs during this period would have been equivalent to about 1,000 r. This calculation assumes that the radiation dose is spread evenly over the lungs, but it may well be that some areas of the lung, depending on the sizes of the radioactively-charged dust particles which are inhaled, may be subjected to doses of more than 10,000 r over a whole working life. It is consistent with other knowledge that tumours could be induced under these conditions, particularly when it is remembered that radium itself and many of its daughter-products emit alpha particles with high biological efficiency.

76. The only conditions in which an increased incidence of lung tumours has been observed in association with radiation are those in which there is an increased risk of inhaling radon and the other daughter-products of radium. In theory, however, the inhalation of radioactive material in particulate form, either as a result of fall-out from nuclear weapon explosions or in the vicinity of nuclear reactors, could lead to the accumulation of a high radiation dose within the lungs. Such particles would not be uniformly distributed within the lungs but would tend to aggregate on discrete small areas of the bronchi, which would thus be subjected to a high radiation dose, with the result that in the long run lung cancers might be produced in some people. In this country appropriate measures are always taken to eliminate the hazard in the vicinity of nuclear reactors, and it would be extremely unlikely to occur as a result of fall-out except in conditions of actual warfare. There is no evidence that external irradiation by X- or gamma rays can cause lung tumours in man.

Cancer of the bones and joints

77. Radium and the daughter-products of thorium, when assimilated into the body, tend to be held for long periods of time in the bones where, if

in sufficient concentration, they may give rise to local destruction and disease. A number of artificially produced radioactive isotopes, of which the most important are strontium and plutonium, also show this predilection for bone. Radioactive strontium exists in several forms, one, strontium 89, having a half-life of 53 days and another, strontium 90, of 28 years, while the half-life of plutonium 239 is about 24,000 years. A warning of the potential danger from these artificial elements is given by past experience of the effects of the natural elements radium, mesothorium and radiothorium after they have gained entrance to the body and become fixed in bone (Appendix N).

78. Our knowledge of these effects comes mainly from the case-records of former workers in the luminising industry and of a group of patients given radium compounds internally in the course of treatment. Stringent controls are now enforced in the luminising industry to protect the workers, and the prescription of radioactive substances for treatment has been controlled by legislation.

79. Since 1925 there have been many reports of illness and death occurring among a group of workers engaged in the painting of watch and clock dials with luminous paint, most of whom had been in the industry during the period from 1916 to 1924. Luminous paint is compounded of zinc sulphide and radium, and, formerly, varying mixtures of radium, mesothorium and radiothorium were also used. It was customary for dial painters to apply their paint with fine brushes, the points of which they 'tipped' between their lips before painting. In this way they swallowed radioactive material, some of which became lodged in the skeleton. If large amounts were swallowed, death sometimes occurred, within about three years, from severe anaemia, haemorrhages, and infections, particularly of the bones of the jaw. Those who had ingested smaller quantities of paint often developed cystic and necrotic changes in the bones which might cause 'rheumatic' pains or fractures. Occasionally, these changes progressed and cancer of the bones appeared. Such tumours usually developed more than fifteen years after the first exposure to the hazard.

80. Similar effects have occurred in patients given radium compounds internally for the treatment of mental disease or for various rheumatic and other affections, and in people who, for quasi-medicinal reasons, have consumed large amounts of 'radioactive water'. In animals strontium 90 has been shown to produce similar biological effects.

81. It is possible to estimate the amount of radium in the body of a living person, if there is good evidence that no other radioactive element is present in addition to the normal components of the body. Measurements carried out on those who have been exposed to unknown mixtures, such as luminous compounds, are difficult to interpret. So far, no person is known to have developed radiation-induced bone cancer who had less than 3·6 microcuries of radium in his body, unless either mesothorium or radiothorium was also present; the lowest radium content, in the presence of one or other of these elements, has been 0·52 microcurie at the time of appearance of the tumour. On the other hand, it seems certain that early non-cancerous cystic changes in bones have developed with a body-content of as little as 0·4 microcurie of radium alone. These amounts of radium are to be contrasted with the maximum permissible level for body radium, which, as laid down by the International Commission on Radiological Protection, is 0·1 microcurie.

82. Bone cancer has also been reported after the use of X-rays in the treatment of non-malignant bone tumours and some infections. Such cancers have occurred only after very heavy doses of radiation and have originated

in the area of the body treated. The risk of the development of bone cancer at the levels of X- or gamma radiation experienced under modern occupational conditions is insignificant.

Cancer of the skin

83. Cancer of the skin was the earliest form of radiation-induced tumour to be described in man. Radiation dermatitis of the hands, forearms and face was common among the early radiologists and radiological technicians, and cancer often occurred in the damaged skin. By 1911 no fewer than 54 cases had been described ; the occurrence of these tumours diminished as radiologists learned to take the necessary precautions.

84. Since the early part of the century, records have accumulated of the occurrence of skin cancers following X-ray or radium treatment. In some instances, these tumours have followed the injudicious use of X-rays for mild skin affections, or even for the removal of facial hair. The latent periods have usually been long, ranging in a recently reported series of 13 cases from 12 to 56 years, with an average of 33 years, Although it is usually impossible to make any accurate retrospective assessment of the doses of radiation received, the severity of damage to the skin suggests that, in these cases, they must have been of the order of several thousands of roentgens.

Cancer of the thyroid gland, the pharynx and the larynx

85. A number of cases of cancer of the thyroid gland have been reported among children, some years after they had been given X-ray treatment for conditions including suspected enlargement of the thymus gland, bronchitis, infected tonsils and adenoids, and enlarged glands in the neck. In many instances, the children were less than one year old when irradiated. In a series of cases irradiated for suspected enlargement of the thymus gland, the average latent period between irradiation and the establishment of the diagnosis of cancer of the thyroid gland was only about 7 years. Perhaps the most important feature of these cases is the comparatively small dose of radiation responsible for induction of the tumour, in contrast to the large doses associated with the induction of cancer in adults ; cancer of the thyroid gland has developed in a child after a recorded dose as low as 250 r. It is possible that hormonal factors may be involved in addition to the direct effect of irradiation.

86. A few reports have drawn attention to the development, many years later, of cancers of the pharynx and larynx in patients who have had X-ray treatment for such conditions as tuberculous glands of the neck. The latent period is long, averaging about 20 years, and periods of more than 30 years have been recorded. In most cases, the irradiation was given in the early days of radiotherapy and there is practically no information available about the size of the radiation doses that were employed.

EFFECTS ON THE BLOOD OTHER THAN LEUKAEMIA

87. Observations have shown that a fall in the numbers of red cells, white cells and platelets in the blood may occur in persons exposed to radiation in the course of their work. There is little direct information on the dose-response relationships, but it seems possible that, even with whole-body doses of gamma rays as low as 1 r per week, slight changes can occur in the white-cell count of especially susceptible people. Certainly, with doses much in excess of 1 r per week, a general depression occurs in the white blood cell count. A reduction in the numbers of red cells and platelets may occur at a later stage, and in some persons continued exposure may lead to severe degrees of anaemia.

Aplastic anaemia

88. If not detected in time, radiation-induced anaemias may endanger life, particularly when the red bone-marrow is itself so severely damaged that the red-cell deficiency cannot be made good by the production of new cells; this condition is known as 'aplastic anaemia'. The diagnosis is not easy to make, and the condition can easily be confused with aleukaemic leukaemia unless a full examination of the bone marrow is carried out. This diagnostic difficulty was encountered during the investigation of leukaemia among patients treated with X-rays for ankylosing spondylitis. Particular attention was paid to deaths reported as being due to aplastic anaemia but, when these cases were fully investigated, evidence was found that a number were, in fact, aleukaemic leukaemia; eventually, only four deaths could with any certainty be ascribed to aplastic anaemia out of a total of some 50 deaths from leukaemia, aplastic anaemia and allied diseases combined. Similarly, only six cases of aplastic anaemia were reported from Nagasaki, compared with over 40 cases of leukaemia in the same city. It seems clear, therefore, that aplastic anaemia is a rarer delayed effect of radiation than leukaemia.

INDUCTION OF CATARACT

89. The term 'cataract' implies an opacity in the normally transparent lens of the eye, varying from a tiny granule which does not cause any definite impairment of vision, and which may disappear, to a large plaque resulting in blindness. It has been known for some time that exposure of the eye to X-rays can lead to cataract formation, but the large doses which appear to be necessary for its induction are only likely to occur under very unusual conditions. For all practical purposes, therefore, the production of cataract by X-rays is not an occupational hazard, although it was discovered in 1948 that the condition had developed among a group of physicists exposed to neutron irradiation during the operation of a cyclotron.

90. In the following year there were reports from Japan of an increased incidence of cataract in the populations of Hiroshima and Nagasaki. The extent of the increase cannot be determined with precision, but it is significant that, of 98 cases of cataract among survivors of the Hiroshima explosion, 85 occurred in persons who were within 1,000 metres of the centre of the explosion and would thus have been subjected to neutron- as well as gamma-irradiation. Confirmatory evidence of the high dosage which they had received is provided by the fact that most of them had suffered epilation of the scalp and that two subsequently developed leukaemia.

EFFECTS ON THE SKIN OTHER THAN CANCER

91. In the paragraphs dealing with the induction of skin cancers by irradiation, it was noted that cancers develop mainly in skin which has been subjected to such heavy doses of radiation as to be obviously damaged. Most of our knowledge of the less serious delayed effects on the skin has been obtained from observation of the results of therapeutic irradiation with X-rays, during which the skin may be exposed to large doses of radiation directed to underlying tissues. With doses of 1,500 r or more, a certain amount of permanent skin-damage is likely to occur, but it will not be particularly severe unless a large area has been irradiated. Larger doses, however, say of 4,000 r or more, are often followed by obvious skin-damage, the texture becoming thinner, and the surface being usually covered with dilated blood vessels. In such cases, the skin may be very sensitive and prone to infection, and it is in this type of damaged skin that radiation-induced tumours are most likely to develop.

92. The hair follicles and glands of the skin may also be affected by radiation. A dose of the order of 300 to 400 r will cause temporary loss of hair, and with higher doses, perhaps 700 r or more, hair-loss may be permanent. It is a common finding that, owing to the destruction of the sweat glands, heavily irradiated skin permanently loses its ability to sweat. After doses of the order of 1,500 r, the sebaceous glands are destroyed and the skin loses its normal greasy texture.

EFFECTS ON THE KIDNEY AND LUNG

93. It has been reported that therapeutic doses of X-rays to the region of the kidneys may affect their function and lead to the development of high blood pressure which may prove fatal. The damage described has followed the treatment of certain rare tumours with large doses of radiation and it is unlikely that such effects will occur under other conditions of exposure. It has also been reported that pneumonitis, sometimes fatal, has followed radiotherapy directed towards the chest.

SHORTENING OF THE LIFE-SPAN

94. A number of reports based on observations made on animals suggest that exposure to ionizing radiations may lead to a reduction in the expectation of life. No evidence has yet been published that this occurs in man.

The Effects of Exposure to Radiation during Pregnancy

Abortion and stillbirth

95. After heavy doses of radiation, a pregnant woman may miscarry or give birth to a stillborn child. Information from the Atomic Bomb Casualty Commission shows that in Hiroshima and Nagasaki there were higher abortion and stillbirth rates among pregnant women near the explosion than among those at greater distances. Of 98 pregnant women in Nagasaki who were within 2,000 metres of the centre of the explosion, about 23 per cent of those who had severe radiation illness miscarried, in comparison with only about 4 per cent of those who did not develop any severe illness, and with about 3 per cent of women who were between 4,000 and 5,000 metres distant. It is apparent that abortion and stillbirth as a result of irradiation during pregnancy do not constitute a problem unless the dose of radiation is large.

Effects on the children of women irradiated during pregnancy

96. There is considerable evidence, both from the case records of patients treated with radiotherapy and from reports published by the Atomic Bomb Casualty Commission, that heavy irradiation of pregnant women can lead to the birth of children who are either abnormal at birth or who later develop in an abnormal way. The case records of women therapeutically irradiated during pregnancy describe a number of different developmental abnormalities in their children, the most striking of which is the condition known as 'microcephaly'; one such case was found during the course of our investigation of patients treated by X-rays for ankylosing spondylitis. The underlying cause of this condition is a partial failure of the development of the brain, as a result of which the head is smaller than that of a normal baby. All grades of the condition exist, ranging from the most severe, in which the child usually has to be maintained in a mental institution, to others in which there is only slight impairment of development and mental powers.

97. There are published records of eleven mentally-retarded children in Nagasaki and Hiroshima who were exposed before birth at a distance of between 700 and 1,200 metres from the centre of the explosion. Ten of the mothers of these children suffered acutely from the effects of radiation, and the eleventh probably did so. The head circumferences of all eleven children were appreciably less than those of unirradiated Japanese children of the same age-group and, in the cases among Nagasaki children, smaller than those of children exposed before birth at distances of between 4,000 and 5,000 metres from the explosion, where the dose of prompt radiation would have been less than 1 r. The evidence from Hiroshima suggests that children irradiated between the twelfth and eighteenth weeks of intra-uterine life are more likely to develop microcephaly than children irradiated either before or after this period.

The Effect on Fertility of Exposure to Radiation

Permanent sterility

98. It is well-established that irradiation may reduce the fertility of men and women, and even render them permanently sterile. In men, a single dose of 500 r to the testes would probably produce permanent sterility. The dose to the ovaries likely to produce the same result in women would depend to some extent upon the age of the woman concerned; a woman nearing the end of her reproductive life would require a smaller dose, about 300 r, than a woman in her early reproductive years. These levels of dose are so high that, if they were received in the course of whole-body irradiation, the individual would develop the early acute illness already described. It is extremely unlikely, therefore, that permanent sterility would be induced in any one accidentally exposed to a large whole-body dose of radiation, unless the acute illness had been manifest.

99. Under modern conditions of occupational exposure, for example among radiologists and radiographers, there is no evidence of any impairment of fertility. Furthermore, there is no suggestion that female radiographers suffer from radiation-induced menstrual disturbances which might be accompanied by diminished fertility.

CHAPTER IV

THE GENETIC EFFECTS OF RADIATION

Introduction

100. Nowhere in our report have we been more conscious of the difficulties of the task which we have undertaken, and of the limitations of the knowledge at our disposal, than in considering the genetic effects of radiation. The established scientific evidence in this field provides but an insecure basis on which to frame answers to the many important questions that are now being asked. Consequently we have been forced to make many assumptions of questionable validity and our conclusions must be regarded as provisional and treated with a measure of reserve. An essential part of future studies will be the collection of more detailed vital statistics. Moreover, it must be realised that genetic studies inevitably tend to be slow and that sufficient knowledge on which to base firm conclusions will be accumulated only after many years of intensified fundamental research.

The Material Basis of Heredity

101. In man and other sexually reproducing animals, every individual arises from a single living cell, which is formed by the fusion of two germ cells, an egg cell from the mother and a sperm cell from the father. Soon after it is fertilised, the egg cell divides into two; each of these divides again, to give a total of four, and this process is repeated until there are enough cells to give rise to all the organs and tissues of the body, among them the sex glands from which in time new germ cells will be formed.

Chromosomes and genes

102. Each cell contains within it a nucleus whose essential component is a number of microscopic thread-like structures, the chromosomes. These are aggregates of sub-microscopic particles—the genes—which determine the hereditary nature of the individual. The total number of genes in a cell is not known with any accuracy but it is certainly high, perhaps thousands or even tens of thousands in a human cell. Each chromosome carries a large number of them arranged in order along its length, so that each gene has its own special place, or locus, in a particular chromosome.

Cell division

103. The nucleus of each human germ cell carries 24 chromosomes, all of them different from one another. When egg and sperm come together, their nuclei also fuse, so that the fertilised egg contains a nucleus carrying 48 chromosomes constituting 24 pairs. The members of a pair, derived one from the mother via the egg and one from the father via the sperm, normally correspond to each other both in the gene loci which they carry and in the order in which these loci are arranged. When the fertilised egg, or any later cell in the body, is about to divide, a replica is first formed of each of the 48 chromosomes. This makes it possible for the two cells so produced to receive sets of chromosomes exactly like each other and like those of the parent cell. The cell divisions immediately preceding the formation of germ cells, however, follow a somewhat different pattern, which

results in the egg or sperm receiving only one member of each pair of chromosomes; thus the number that the egg or sperm contains is reduced to 24.

Gene mutation

104. In the normal course of events, each cell possesses a set of genes identical with those of the cell from which it is derived. Occasionally, however, a sudden change occurs in a gene, which is converted into a slightly different form. The altered form of the gene is spoken of as a new allele and the process of change is known as mutation. Once such a mutation has occurred, the gene is reproduced and passed on in the new form at all subsequent cell divisions. Thus, each locus can come to be represented in the population by a number of these variants or alleles.

Homozygotes and heterozygotes

105. Having but one chromosome of each kind, the germ cell carries only one allele at each gene locus, whereas the body cells, with a pair of each kind of chromosome, carry two alleles. These two alleles may or may not be exactly the same. An individual bearing two identical alleles is said to be homozygous at that particular locus; one with two different alleles is said to be heterozygous at the locus. A homozygous individual clearly must have received the same allele from each of his parents and he will pass it on to all his offspring. A heterozygous individual must have received different alleles from his two parents, and he will on the average pass on one of the two to half his offspring and the other to the other half. This sorting out of the genes when they are distributed to the offspring of a heterozygote is a direct result of the halving of the number of chromosomes during germ cell formation and is known as segregation.

Gene reassortment

106. If an individual is heterozygous for two or more loci, the process of segregation will result in his genes being reassorted into new combinations in his offspring. Thus, in the process of reproduction, the various alleles at the different loci are continually being reassorted into an immense variety of combinations, with the result that each person has a particular hereditary constitution not exactly like that of anyone else. Each of us is, in fact, genetically unique, with the exception of identical twins, who are produced when the fertilised egg—very early in its development—splits into two parts each of which gives rise to a complete individual.

Dominant and recessive alleles

107. Some alleles produce a noticeable effect only on those individuals who are homozygous for them. Such alleles and the characters which they determine are spoken of as recessive. Other alleles have some effect even when the individual is heterozygous, and the characters which these determine are described as dominant. Among the numerous genes which have been studied in man and other animals, all gradations are known between the extremes of fully recessive alleles, which have no effect at all on the heterozygote, and fully dominant ones which have as strong an effect on the heterozygote as on the homozygote.

108. A dominant allele which is being transmitted in a family will be manifest in every generation unless it dies out. A recessive allele, on the other hand, can be transmitted to later generations by an individual who shows no sign of carrying the allele in question. The character produced by such

a gene will appear from time to time in the population, in families where both parents carry the gene ; this is especially likely to occur where there is a marriage between cousins.

Sex-linked genes

109. There is a special category of genes whose transmission is connected with the determination of sex. The sex of an individual is determined by one particular chromosome pair. In the female, both chromosomes of the pair are similar ; in the male, one is of another type. Thus, the structure of the female sex chromosome pair can be represented as XX, that of the male as XY. Genes carried on these chromosomes are said to be sex-linked, but the Y chromosome, having few geni loci, has little effect on most hereditary characters. If a male contains an abnormal allele on his X chromosome, he will show its effect, even if the same allele situated on one of the two X chromosomes of a female would be recessive. If a recessive sex-linked gene is uncommon, it will occur only very rarely on both the X chromosomes of a female and the characteristics it produces will therefore not often be found in females. They will be commoner among males, who will be affected whenever their single X chromosome contains the allele. The apparently normal females can, however, transmit the chromosome carrying the affected gene to the next generation, and so act as carriers of the abnormality. The classical example of this type of condition is the disease haemophilia, which appears in males but is transmitted by apparently normal females.

Continuous variation

110. Not all gene differences have effects which are sufficiently distinct to be recognised by their segregation among the members of a family. As can be seen in human stature and intelligence, for example, some characteristics vary by continuous gradations over a wide range which is regarded as normal. Such characters are believed to be controlled by the combined action of a large number of genes, the effects of which are supplementary and each so small that they are not individually distinguishable.

Causes of mutation

111. Reference has already been made to the process of mutation by which one allele changes into another. We do not know all the factors which cause mutation. It is believed to be due sometimes to chance disturbance of the complex molecules which constitute the genes, and sometimes to external influences such as certain chemicals or natural background radiation. We shall discuss later the extent to which naturally occurring mutations are likely to be attributable to background radiation, but from observation of organisms other than man it is known that other causes can be important. Present evidence suggests that mutations, whatever their origin, are for the most part random changes not specifically related to the nature of the stimulus or to the needs of the individual.

Mutation rate

112. So far as is known, all genes are subject to mutation, and, over the population as a whole, mutation is continually occurring at a definite but very low rate (Appendix C). Factors influencing mutation increase the rates at which the genes change ; they do not produce changes of a novel type. Most genes, if not all, are susceptible to such factors, though some may be affected more readily than others. No methods are yet known for stimulating the mutation of only one particular gene, or even of a small selected group of genes.

Mutation and natural selection

113. The hereditary variation found in human or other populations is the result of mutations which occurred in past generations. New alleles which cause abnormalities harmful to those who possess them tend to be eliminated from the population. Thus, a new dominant allele causing death before the reproductive age, or for some other reason preventing the individual from leaving descendants, is eliminated in one generation. Those with less drastic effects are eliminated with a speed related to the severity of the handicap which they impose. Recessive alleles, which give rise to harmful conditions only when they are in the homozygous state, are also eliminated, but very slowly, since the allele can continue to exist or to spread in the heterozygous state without contributing any significant handicap to the perpetuation of the line. Thus, in the case of both dominant and recessive harmful alleles, natural selection is constantly operating towards their removal from the population. Equally, it will operate even against genes whose effects are valuable to the individual during his own lifetime, if they reduce his chances of leaving offspring.

114. For alleles which increase the chances of leaving offspring the situation is the reverse. Those individuals in which these effects are manifest are more than ordinarily likely to propagate their kind, so that their descendants tend to become the predominant type. Many, but perhaps not all, of the genes which tend to increase fertility will also have effects which are useful in other ways to the individual in whom they are manifest; they can be considered as generally advantageous.

115. Between these clear-cut examples there are genes with every gradation of effect. The manifestations of genetic abnormality in the individual may vary from the trivial to the disastrous. The action of some genes is delayed until after the end of the reproductive period of life and they are therefore largely immune from the eliminating influence of natural selection. There are genes which are harmful whether the individual carrying them is homozygous or heterozygous; there are others which are advantageous to heterozygotes and harmful to homozygotes. The relative prevalence of any particular allele, in any particular population, can be understood only in the light of the relationship between the environment and the advantages or disadvantages of the condition to which the allele gives rise.

Genetic equilibrium

116. As natural selection is constantly operating towards the elimination of harmful genes from the population, the incidence of the conditions to which they give rise would steadily decrease, were it not that they are being replenished by the occurrence of new mutations. Their frequency will therefore tend to reach the level at which their loss by selection is balanced by new mutation. A state of genetic equilibrium has then been reached. This is the situation with regard to many of the more harmful abnormalities in man, the incidence of which remains relatively steady in the population despite the failure of those affected to leave normal numbers of offspring.

The general effects of increasing mutation rates

117. The above considerations suggest the broad qualitative effects which can be expected to follow an appreciable increase in mutation rates. At this present time, the advantageous alleles that have appeared in the past are already widespread and will have become part of the normal constitution of the population. An increase in the rate at which they are produced can therefore have little effect. The harmful alleles, on the other hand, have been restricted to a low incidence by the operation of natural selection. A

significant increase in the rate at which these are produced will therefore have a more easily detectable effect. The general inference is that increasing the mutation rate in a human population would have a relatively much greater effect upon the incidence of harmful than upon that of harmless or of advantageous hereditary traits.

Mutation and the adaptability of populations

118. Mutation provides the means by which the human race, through hundreds of thousands of years, has successfully adapted itself to its environment. There is no reason to believe that this adaptation has proceeded by sudden and conspicuous changes in human characteristics. The consensus of opinion is that evolution has occurred by a succession of small variations from the average, which conferred a slight but eventually important advantage in relation to the trend of environmental change. It is the existence of this galaxy of hereditary traits, varying only slightly from the accepted normal, that has conferred adaptability upon the human race. The recurrence of a small but steady incidence of harmful mutations is the price that has to be paid for this asset.

The Genetic Effects of Radiation : Basic Principles

119. There is as yet little direct information about the genetic effects of ionizing radiations on man and, for reasons which we examine later, the few observations that have so far been made present many difficulties of interpretation. We have therefore to rely on information obtained from experiments on other organisms. The experimental evidence is itself incomplete and largely derived from observations on forms of life other than mammals, but a general picture is beginning to emerge which appears to be consistent for the organisms which it has been practicable to study. The question arises, however, to what extent it is justifiable to draw inferences concerning man from the reactions of remote organisms observed under the artificial conditions of laboratory experiment. Since the genetic mechanism in man is the same as that in other animal and plant species, and since the animals and plants that have been studied all show the same type of genetic response to ionizing radiations, it would be unreasonable to suppose that the response in man will do other than follow the same general pattern. On the other hand, we do not think that conclusions derived solely from observations on other organisms offer a secure basis for quantitative estimates concerning man and, except where we have explicitly stated the contrary, we have not used them for this purpose.

Effects of radiation on germ cells

120. Ionizing radiation will have genetic consequences only in so far as it affects any of the germ cells or the cells ancestral to them in the reproductive organs. It may then have one of three results: the affected cells may die, their chromosomes may be broken, or the genes may be caused to mutate.

121. Death of a germ cell, or indeed of any cell ancestral to it, can have no genetic consequence, because this very death will terminate the lineage of cells to which it belongs.

Chromosome structural change

122. Chromosome breakage may also lead to death of the cell lineage. Broken chromosomes may fail to reunite or they may join up to give new forms of chromosomes which are incapable of passing through the process of cell division in the normal way ; either of these circumstances leads to

subsequent death of the cell lineage. If the damage occurs in an immature germ cell in the sex gland, the cell lineage will usually die before mature germ cells are formed, and there will be no genetic consequences. If it occurs in a mature germ cell which later participates in fertilisation, the ensuing embryo will usually die early in gestation, and the ultimate genetic effects will be minimal.

123. Chromosome breakage may, however, have a different outcome if the fragments reunite in new patterns which are capable of passing through cell division. The resulting kinds of structurally changed chromosomes may be transmitted to apparently normal offspring, and at least one type of change will manifest itself by the occurrence of repeated abortions or malformations among the descendants of the irradiated individual. Experiments on mammals indicate that this inherited effect will appear only if conception takes place within a few months of irradiation.

124. Such structural changes of the chromosomes are induced especially by large single doses of radiation, for example heavy doses of X-rays or the prompt radiation from atomic bombs. They are induced only rarely by long-continued exposure to low-intensity X- or gamma radiation, although relatively small doses of neutrons or alpha particles are more effective in bringing them about.

125. Although they may cause partial sterility or abortion in their carriers, major structural changes of the chromosomes do not as a rule bring about other kinds of abnormality in individuals bearing them. For this reason, and also because of their low rates of spontaneous occurrence and induction by chronic irradiation, and of the probability of their having an adverse effect on fertility, chromosome structural changes are likely to be of comparatively little importance among the radiation hazards to man.

Induced gene mutation

126. The third and, from the genetical point of view, the most important effect of exposing germ cells to additional radiations is the induction of increased gene mutation. Since all germ cells from time immemorial have been continuously exposed to some radiation from natural sources, it would be surprising if exposure to additional radiation were found to induce any novel types of mutation. The results of experiment support this view; the types already known recur, but at an enhanced rate.

Proportionality of induced gene mutation to additional radiation

127. There is no known threshold for the induction of gene mutations by radiation: that is to say, any additional exposure, no matter how small, must be expected to raise the mutation rate, if only by a minute amount. Furthermore, to judge by our experience up to the present, it is probably true that the rise in the rate of mutations is directly proportional to the amount of additional exposure. This law is known to hold good, for such organisms as have been studied, when the radiation dose is fairly high. It is also known to hold good for the induction of one class of lethal genes in the fruit-fly, *Drosophila melanogaster*, by X-ray doses as low as 25 r. In this chapter of our report we are chiefly concerned with doses well below this level; but, for the present, there does not appear to be sufficient evidence to warrant the assumption that there is any real departure from this law even at the lowest doses, and proportionality has therefore been accepted as the basis of what follows.

Accumulated dose to germ cell lineage

128. Cells arise only from pre-existing cells. Mature germ cells are produced from a line of ancestral immature cells which have been present at every instant during the individual's life, from conception onwards. It will be remembered that mutated genes reproduce themselves as faithfully in a cell lineage as do the normal genes from which they arise. In consequence, the mutated genes arising throughout a germ cell lineage will be accumulated in the mature germ cell, and a given radiation dose will therefore have the same order of effect whether it is given over a short or a long period of time. In other words, long continued exposure to low-intensity radiation induces as much gene mutation as a single exposure to high-intensity radiation, provided that the total dose is the same. Experiments have shown that this probably remains true even when the dose is split into a series of small fractions, and no matter what interval elapses between the separate irradiations. Thus, in contrast to most other types of biological response to radiation, damage to the genetic material cannot be repaired and the effect from repeated exposures is cumulative.

Genetically effective dose to a population

129. This cumulative effect of radiation indicates that the genetic effects of exposure will depend on the ages of the individuals exposed as well as on the dose they receive. If, for example, all are past the reproductive age, the genetic effects will be nil ; if they are younger, the possible number of offspring they may have is of importance. The age distribution of those exposed is therefore an important factor to take into account when estimating the consequences to future generations of additional radiation.

The Effects of Increased Mutation Rates on the Incidence of Disease in Human Populations

130. In approaching the problem of making some quantitative assessment of the genetic effects of radiation upon a human population, we have been very conscious of the inadequacy of the evidence in two essential respects. First, we do not know the dose of radiation required to double the mutation rate of any specified human gene ; secondly, there is reason to suspect that the radiosensitivity of human genes may vary considerably, so that it could be very misleading to treat them as an approximately uniform group which would respond to any particular dose of radiation with a standard increase in mutation.

131. It is, however, possible to give a general idea of the effects of an increase in the mutation rate of particular human genes without raising the question of dose or degree of uniformity in sensitivity to radiation. We have therefore taken selected examples of diseases in which genetical factors are known to play an important part, and have attempted to assess the effects in a human population of doubling the spontaneous mutation rates of the genes concerned, without specifying the agent or agents causing this increased rate of mutation.

132. The role of heredity in the production of disease ranges from that of a predisposing to that of a preponderating cause. Thus, there is some evidence that heredity is a factor influencing susceptibility to tuberculosis ; but this could be of no significance unless the individual were infected with tubercle bacilli. On the other hand, achondroplasia, a form of dwarfism, is probably determined entirely by genetic factors, in the sense that no known modification of the environment can prevent its appearance in those who possess the necessary gene.

133. In making our assessment we have confined our attention to those conditions which impose a significant handicap and which are determined, entirely or to an important extent, by hereditary factors. To put it another way, our object has been to give an assessment of the social load that would be imposed upon a population, like that of this country, by increasing mutation rates. We propose to consider two possible situations: first, when the mutation rate of every gene concerned is supposed to have been doubled in one generation only, thereafter reverting to its former level, and secondly when the rates having been doubled remain at that new level generation after generation.

THE EFFECTS OF DOUBLING MUTATION RATES ON DISEASES DUE TO A SINGLE GENE

134. For abnormal genes with effects which are masked in any way, either by normal alleles—as in recessive traits—or by the influence of environment, mutation has less immediate or less apparent consequences than for genes with effects which are directly manifest, as in dominant traits. Severely disabling dominant diseases reveal the results of recent mutation most readily. Sex-linked traits show a less rapid response. The effects of genes which produce recessive traits are masked until two genes come together in the homozygous state; hence, the results of their recent mutation will be less noticeable, although in the course of time their full effects will appear. Where a gene is common and mainly benign, but can occasionally cause disease, the results of its recent mutation will be scarcely detectable.

135. The effects which might be expected to result from an increase in mutation rates can most easily be calculated for diseases known to be caused by single genes (Appendix D). For this purpose we need to know what proportion of cases in a given generation is due to recurrent fresh mutation. The incidence of the particular disease in the population must be ascertained; so must its mode of inheritance (dominant, partially recessive, recessive or sex-linked), the degree to which it handicaps or favours the affected individuals—as shown by their length of life and reproductive capacity—and the modifying effect, if any, of environmental factors. This information is available for only a relatively few conditions and much more research will be required before we can feel reasonably confident in making estimates for groups of diseases. To illustrate the different types of effect, we have chosen three examples about which we have some fairly accurate information.

(*i*) *A dominant trait*

136. Achondroplasia (chondrodystrophia) is a dominant form of dwarfism. Although there are many clinical types, it will be assumed for the present purpose that the condition is due to a single gene with a manifestation which is independent of environmental factors. The incidence at birth, according to Danish estimates, is one in 9,400. Biological fitness is greatly reduced on account of high stillbirth rates and also, in adult life, on account of low fertility, especially of females. The chance that an affected individual will have offspring is estimated at only 1 in 5. The majority of cases are believed to arise through fresh mutation.

137. Doubling the mutation rate of the causal gene for one generation would produce an 80 per cent increase in the incidence of the condition in the first generation, that is the incidence at birth would rise to nearly one in 5,000. The excess would, however, rapidly disappear and within five or six generations the incidence would return to normal (Fig. 1a). If the

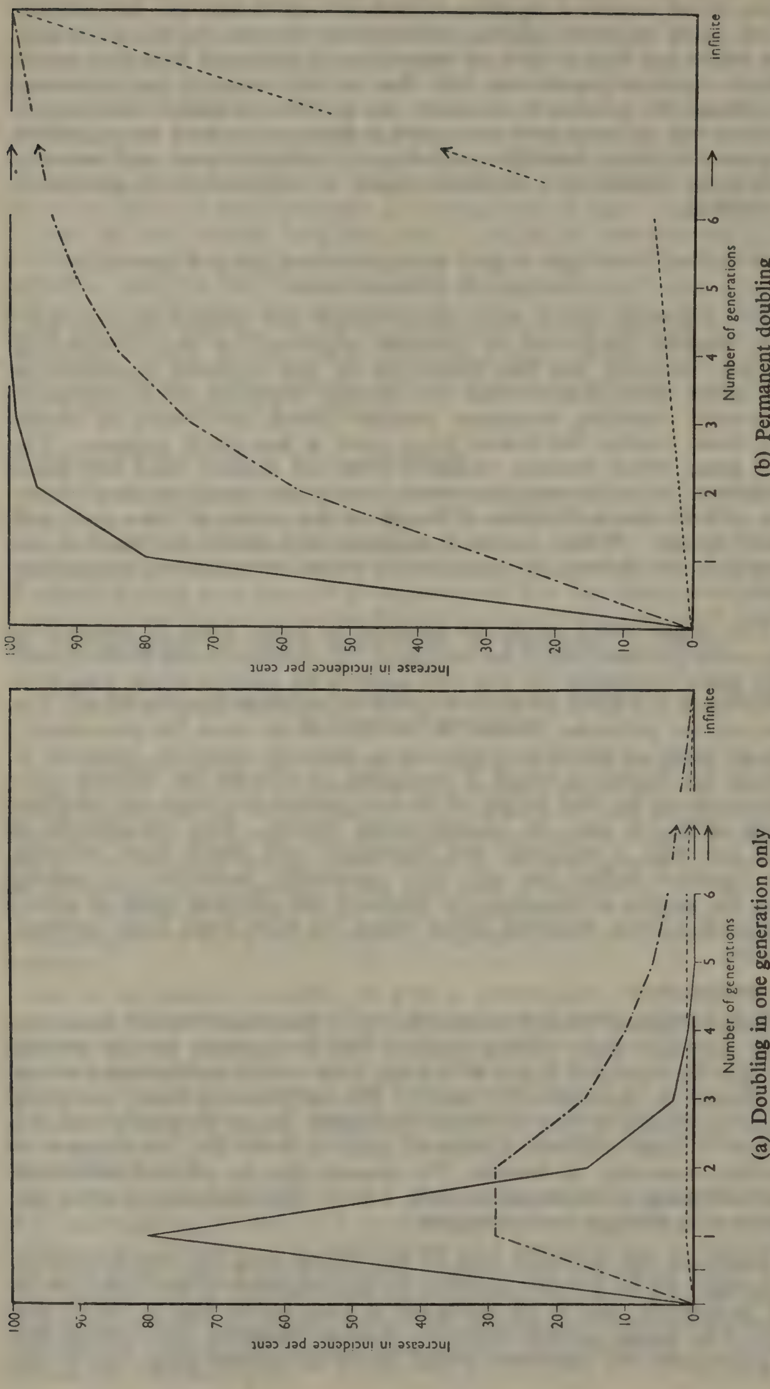


FIG. 1. The effects of doubling the mutation rates for three hereditary conditions, expressed as the percentage increase in incidence (Appendix D, Table 2D).

mutation rate were permanently doubled, the incidence would rise to a level close to double the present figure (i.e. 100 per cent increase) within three or four generations (Fig. 1b).

(ii) A sex-linked trait

138. The term haemophilia covers a group of sex-linked traits in which there is impairment of blood clotting. For the present purpose it will be assumed that all severe cases are caused by abnormal alleles at a single locus. The incidence is at least 1 in 12,000 of the male population at birth and the chances that an affected male will survive and have descendants is about one in eight. Females carrying the abnormal gene are healthy and have normal chances of reproduction. Doubling the mutation rate for one generation would produce a 29 per cent increase in incidence in the following generation, that is, the incidence would rise to about 1 in 9,300. In the next generation this level would be sustained but thereafter it would sink back moderately quickly towards the previous level (Fig. 1a). If the mutation rate were permanently doubled, the incidence would rise to 90 per cent above the previous level in about six generations, and thereafter slowly approach the point where the incidence was doubled (Fig. 1b).

(iii) A recessive trait

139. Phenylketonuria is an example of a deleterious recessive trait associated with severe mental deficiency. Its incidence in the population at birth is about 1 in 40,000 and, although the early mortality is not high, the chance that an affected person will have offspring is practically nil. A doubling of the mutation rate in one generation would cause an increase of one per cent in the incidence of the disease in the first generation. If allowance were made for the effects of inbreeding, a further small increase would be predicted sometime after the second generation and this would be followed by a very slow return to the previous level (Fig. 1a). The response to a permanent doubling of the mutation rate would be a slow rise by almost equal increments. It would take more than 50 generations of 30 years each to increase the incidence by 50 per cent, and many more to approach an increase of 100 per cent (Fig. 1b).

THE EFFECTS OF DOUBLING MUTATION RATES ON BROAD GROUPS OF DISEASES

140. The three examples given above illustrate the kind of result to be expected from doubling the mutation rates of genes representing each of the three classical types of genetical effect. A large number of dominant, sex-linked and recessive diseases are known; many of them are rarities but together they may account for a relatively large proportion of serious hereditary disability in the population. However, to give an idea of the extent of the problem of hereditary disability, and the total results of doubling the mutation rates of the genes which are responsible, common categories of illness must be considered. Our information, from the genetical point of view, is unfortunately not often precise and, in addition, the effects of genes are in many cases modified by environment. Mental diseases and mental deficiency, when taken together, account for nearly half the hospital beds provided in this country and are the most extensive inclusive category in which hereditary causes are known to be important. We shall first consider severe mental defect and then the two main types of mental illness.

(i) Severe mental defect

141. The incidence in the population of cases of severe mental deficiency which survive has been estimated to be about 1 in 500. It is higher than this at birth but subsequently reduced by the heavy mortality in early life. Beyond

this, there are grossly affected individuals, many with malformations of the nervous system, who are stillborn or who do not survive early infancy but who would have been mentally defective had they lived; the incidence of such cases is at least 1 in 200. The number of cases of severe defect at all ages surviving in England, Wales and Scotland may be nearly 100,000. The fact that not far from 30,000 hospital beds are provided for these cases indicates the extent of the medical care required.

142. In this broad category of disease, conditions with dominant inheritance include epiloia (sebaceous adenoma with tuberose sclerosis), several types of acrocephaly, hypertelorism and neurofibromatosis. In such diseases, many of the severely affected cases, say one half, can be attributed to recurrent fresh mutation. Doubling of all the mutation rates, for one generation, would have a large effect in the subsequent generation, and a permanent doubling of the mutation rates would soon permanently double the incidence. We may suppose that diseases such as these, collected together, form four per cent of all surviving cases of severe mental defect, as shown in Table 1. If all mutation rates were doubled, this would add 50 per cent to the numbers of these cases in the first generation, that is to say two new cases in every 100 in the whole category, an increase which would mean a thousand extra cases requiring medical care.

TABLE 1

Effect of doubling mutation rates of genes concerned with severe mental deficiency: cases per generation classified according to probable causation

Type of Diagnosis	Probable causation	Surviving cases of severe mental deficiency under present conditions (i)*	(ii)†	Increase in first generation if mutation rates are doubled (i)*	(ii)†
Acrocephaly ... ... Epiloia ... ... Neurofibromatosis ... *et cetera*	Dominant gene	4·0	2,000	2·00	1,000
Microphthalmos ... Gargoylism ... ... *et cetera*	Sex-linked gene	0·5	250	0·15	75
Amaurotic idiocy ... Cerebral diplegia ... Phenylketonuria ... 'True' microcephaly ... *et cetera*	Recessive gene ...	40·0	20,000	0·40	200
Mongolism	Environmental influence plus common genetical susceptibility	12·5	6,250	?+	?+
Others	Miscellaneous, including birth injury and infection	43·0	21,500	?+	?+
All	—	100·0	50,000	3·0	1,500 (approximate figures)

* (i) percentages based upon all cases in the category.

† (ii) numbers which would occur in a generation of 20 million births.

143. A few rare diseases causing severe mental defect are known to be due to sex-linked genes, for example one type of microphthalmos and one type of gargoylism (Hurler's syndrome). The contribution of this group to the total number of defectives is small and can hardly exceed half of one per cent of severe cases. Nevertheless, for the reason given above, diseases of this kind must be due to genes with significant mutation rates: if these rates were doubled in one generation, the incidence at birth of the diseases would be increased by nearly one-third, as in haemophilia. After a doubling of the mutation rates in one generation, there would be an increase of 0·15 per cent in the total number of cases of severe mental defect, or 75 extra cases requiring medical care.

144. Other important known genetical causes of mental defect are recessive conditions, such as the two kinds of amaurotic idiocy, phenylketonuria, cerebral diplegia, and 'true' microcephaly. Known conditions caused in this manner account for 20 per cent of low grade mental defect and the same type of causation may easily account for twice as much as this. The case frequency of these traits individually is low, that is about one in 40,000 in the population at birth; about 20 conditions of this type are already known and perhaps another 20 may exist undetected. Since these diseases are all very deleterious, so that those affected scarcely ever have offspring, it is generally accepted that the genes causing them arise continually by spontaneous mutation. As previously explained, the reason for this assumption is that the incidence in the population can only be maintained if loss of genes through failure of reproduction is balanced by an equivalent appearance of new mutations. From this consideration the mutation rates can be estimated for these recessive traits but the method is indirect and the results are imprecise; they are likely to be too high. A doubling of mutation rates in one generation would cause a one per cent increase in the incidence of each lethal recessive trait the original incidence of which is one in 40,000. If 40 per cent of severe mental deficency were determined in this manner, doubling mutation rates would cause an increase of 0·4 per cent in the whole category or 200 extra cases requiring medical care.

145. The problem of mongolism, a disease responsible for between 10 and 15 per cent of all cases of severe mental defect living in the population requires separate consideration. There is strong evidence of a genetical element in the causation but maternal age is also a very significant factor. The hereditary predisposition must be very common and only harmful in exceptional circumstances. Although a slight increase in incidence might be expected as a result of doubling mutation rates, the nature of the predisposition is so imperfectly understood at the present time that it does not seem useful to make a numerical estimate for this condition.

146. As shown in Table 1, 43 per cent of the cases are not yet accounted for. Among these, there must be a large group in which injury or infection is the main cause, perhaps 15 per cent of all cases of severe mental defect. A still larger number are of quite unknown origin, although genetical factors may have some influence. Furthermore, there may be a residual proportion of cases due to relatively common genes, acting either singly or in combination with one another, on which the effect of increased mutation could be appreciable. It is impossible to make quantitative predictions about mutation for this group with unknown causation but the number of cases ordinarily caused by fresh mutation must be very small.

147. The conclusion is that, after doubling the mutation rates, an overall increase of three per cent in the category of low grade mental deficiency in one generation is possible; in a generation of 20 million births the known

surviving cases would number 50,000 and an increase of three per cent would mean 1,500 additional cases requiring care. If the mutation rate remained permanently doubled, the incidence in the population would, on the most pessimistic assumptions, eventually, after very many generations, double also and twice as much medical care would accordingly be needed.

(*ii*) *Mental illness*

148. Current theories concerning the genetical factors underlying mental illness imply that a small but substantial proportion of cases must owe their origin to the recurrence of mutations. This is almost certainly true for Huntington's chorea, a rare dominant disease. For the common disease groups, schizophrenia and manic-depressive reaction, the situation is less clear. According to figures for 1954, there were approximately 63,000 cases of schizophrenia and 31,000 cases of manic-depressive reaction under hospital care in England, Scotland and Wales. Since these diseases account for about half of all mental illness, it may be worthwhile to attempt a rough estimate of the effects of changing mutation rates upon their incidence.

149. Theoretically, any genes responsible for conditions, like mental illness, which lower biological fitness to a marked degree would have been eliminated from the population, or would have become very rare, unless they had been continually replaced by fresh mutation. However, there are uncertainties about the relevant facts concerning the genetics of schizophrenia and manic-depressive reaction. First, the incidence of these diseases in the population is not accurately recorded; secondly, the biological fitness of predisposed and even of affected individuals has not been fully investigated; thirdly, the nature of the genetical contribution is known only by surmise; and, fourthly, nothing definite is known about possible compensating mechanisms which might, if they existed, make unnecessary the assumption of gene replacement by spontaneous mutation.

150. Calculation, on the basis of elementary and simplified assumptions about gene action, leads to the conclusion that doubling the mutation rate might have the effect of raising the incidence of schizophrenia by a factor of one per cent and of manic-depressive reaction by a factor of 1·4 per cent in the first generation. It is not possible to estimate the number of extra hospital beds which this proportional increase in frequency of genetical predisposition would imply. We can, however, obtain an idea of the expected number of extra chronically incapacitated patients from the calculations set out in Appendix E. The total number of such cases which would appear among the first generation of 20 million births after doubling mutation rates would be 200 schizophrenics and 200 manic depressives. The number of extra patients needing psychiatric care at one or other time during their lives, on account of these genetical predispositions, would be from 5 to 10 times as great. A permanent doubling of mutation rates would have in each succeeding generation an effect similar to that in the first generation. Thus, over a very long period of time, the number of cases would slowly increase until the limiting value of twice the initial number was approached.

(*iii*) *Blindness*

151. A frequent cause of severe disability is blindness. The extent of the morbidity is shown by the fact that every year, in England and Wales, 12,500 new cases of blindness are registered. These include cases of developmental abnormality, tumours, metabolic diseases and the results of injury and infection. The genetical background is extremely varied but at least half of the hereditary cases can be attributed to single genes, often recessive like those causing retinal degeneration, though a few are dominant like that for

retinoblastoma; some are known to be sex-linked. High myopia is believed to have a complex genetical background. The important cases from the present point of view are those with onset in early life and about three-quarters of all such cases of blindness are thought to be hereditary. About 300 children between the ages of 0 and 15 years are registered as blind annually. Severe cases in the causation of which mutation is likely to play an important part will mostly be in this group which includes aniridia, microphthalmos and retinoblastoma. The mutation rates for these dominant diseases are listed in Appendix C. Assuming that these figures apply to England, Wales and Scotland, we can estimate that, in one generation of 20,000,000 births, there would be 80 cases of aniridia, 80 cases of microphthalmos and (using the mean of three estimates) 560 cases of retinoblastoma due to spontaneous mutation in ordinary circumstances. These numbers would be almost doubled if the mutation rates were doubled for one generation. If the mutation rates were permanently doubled, this increase would be continued until the total incidence of these diseases was doubled, as was calculated for achondroplasia.

152. Numerous cases of blindness due to recessive conditions are known, and some have sex-linked inheritance. Figures for the incidence of these traits are not well enough established for the effects of doubling mutation rates to be estimated.

(*iv*) *Neonatal deaths, stillbirths and congenital malformations*

153. An increase in mutation rates would be expected to have an effect upon the abortion, stillbirth and neonatal death rates and upon the incidence of congenital malformations. These deaths and malformations are known to be caused in large part by the environment of the unborn child, which may be affected by illnesses and other conditions in the mother. Many may be due to single recessive genes, some to chromosome abnormalities and others are known to be caused by immunological incompatibility between mother and foetus. For these reasons we have not found it possible to make detailed calculations of the kind used above for other conditions, but it is certain that the total effects of doubling mutation rates in one generation would be slight. Observations on these foetal conditions in actual circumstances where the mutation rates might have been increased have been made on human populations and are discussed below in paragraphs 162 to 170.

THE OVERALL LOAD OF ILLNESS IMPOSED BY DOUBLING ALL MUTATION RATES

154. We have expressed the opinion that, from the standpoint of the social load imposed, mental diseases constitute the most important single category of disease which is determined to a marked degree by heredity and which is serious, in the sense both of being highly harmful to the individual and of making heavy demands on medical resources. We are aware that this is only an opinion and that others may have different views. We believe, however, that it will be conceded by all that the mental diseases contribute a very substantial proportion of the total number of those suffering from serious hereditary disorders. It seems reasonable, therefore, to suggest that the total increase in the social load due to serious hereditary illness of all kinds, which would follow doubling all mutation rates, would be unlikely to exceed more than a few times the estimates we have given for mental defect and mental illness combined.

155. Hereditary diseases are sometimes thought of as incurable, and it is true that present knowledge provides no grounds for believing that cures will

be found for some of the grosser forms. With the advance of medical science, however, it has become possible to alleviate many hereditary conditions or even to maintain the affected patients in good health. A classical example is diabetes mellitus, into the causation of which a hereditary factor enters in many cases. Before the discovery of insulin, the majority of diabetics were destined for invalidism or premature death. Now with its aid they live essentially normal lives. With further advances of medical knowledge, it may be expected that an increasing number of hereditary conditions will be brought into this category, and thus the load of suffering from illness of this kind be reduced. It should be realised, however, that the preservation of those afflicted by hereditary conditions will increase their chances of having children and so lead to an increased prevalence of the condition in the population and with it an increased need for medical services.

156. From the point of view of the long-term effects on the population, it must be remembered that, even though most of the new recessive alleles produced by an increase of mutation rates will not meet a like partner in the first generation after they are produced, and will therefore remain concealed, they will still exist in the population. They will in fact persist until, at some later time, two carriers of like genes happen to mate. The recessive effects will then become manifest in some of the children of such matings. If such genes are harmful, these affected children will produce fewer offspring than the normal, so that there will be a reduced chance that the genes will be passed on to later generations. In this way mutated genes can eventually die out. The extinction of the gene will have been brought about, however, only by the failure of some affected individual or individuals to reproduce at the normal rate, a circumstance which may sometimes be merely unfortunate but in other cases may be the expression of a hereditary defect which causes great suffering. Thus, if the mutation rate is increased, and a crop of newly mutated recessive genes produced, they will continue to cause harmful effects for many generations.

The Effects of Increased Mutation Rates on Hereditary Traits showing Continuous Variation about the Normal

157. Although in any human population we can find individuals who are physically, biochemically or mentally abnormal in a relatively gross way and whose abnormality can be traced to single gene differences, most of the variation between human beings is not in fact of this kind. Even in that greater part of the population which we should describe as normal, no two individuals are alike: they vary by imperceptible gradations over a wide range in respect of many characters such as physique, general well-being, life-span, intelligence and so on. Some of the variation is hereditary but some is due to differences in environment—in the circumstances under which the individual lives and has grown up. The hereditary portion of this variation is believed to be due to the combined action of many genes which supplement one another in producing their effects. These genes cannot be distinguished one from another, and their effects have therefore to be measured in a way differing from that used where a gene has consequences sufficiently drastic for it to be followed as a separate entity. The importance of heredity in such cases is expressed by estimating the proportion of the variation which is traceable to gene effects. The properties of the system of genes are inferred from observations on the amount of this variation which is shared by relatives and on the change in the proportion from generation to generation. In particular, the effect of mutation will be measured by the increment it adds to the variation in each generation.

158. One difficulty should be observed at the outset. The success of this method of approach depends on the ability to measure separately the proportions of the variation attributable to the combined effects of the genes, and the proportion attributable to environmental factors external to the individual. Even in animals and plants, under the conditions of controlled experiment, this is not always easy. In man, whose parents give him not only his genes but also the home and environment in which his early and most formative years are passed, the separation of hereditary and environmental effects is always extremely difficult. Observations on, for example, children brought up by foster parents are of some assistance; but, even so, the conclusions at which we have been able to arrive must be regarded as rough estimates and treated with due caution.

159. Genetic theory leads us to expect that, since mutation brings new gene differences into the population, the basic effect of an increase in the mutation rate will be to increase the variation shown by these characters, that is to raise the numbers of the more extreme types at the expense of the more central, average individuals. Very little information is available, however, even from experimental animals and plants, about the magnitude of the effect to be expected. Such few observations as we have (Appendix F) suggest that in any generation the variation due to new mutation is but a small fraction of the heritable variation observable, and, of course, a still smaller fraction of the total variation, which includes that due to the environment. Indeed, the available data would lead us to expect that hundreds of generations of mutation would be needed to build up the variation which is seen in a human population. A doubling of the mutation rate for a few generations would therefore be expected to have only the most trivial effect on the variation in such characters, and even a persisting doubling of the mutation rate would take very many generations to approach its full effect, which at most would be to double the variation.

The distribution of intelligence

160. The effect on the distribution of intelligence—or, more accurately, of the score in intelligence tests—of an increase in the hereditary variation, such as would be expected to result from a raised mutation rate, is considered in Appendix G. Extensive studies have been made of the intelligence score and something is known about its distribution in the population and the extent to which it is inherited. Increase in the variation, that is in the spread of the distribution, will lead to an increase in the numbers both with markedly low and markedly high scores. Furthermore, the more extreme the class under consideration, the greater the increase in its numbers relative to the overall change in the variation. Thus, from the table in Appendix G, it will be seen that a doubling of the heritable variation could lead in the long run to nearly a tripling of the numbers falling short of an intelligence score of 70 and conventionally regarded as requiring special schooling. On the assumption that the average score did not fall, a corresponding increase would be expected in those with the high scores of over 130.

161. The increase in the two extremes of the distribution may, however, not be symmetrical. Evidence from experimental organisms shows that, where a character has been subjected in the past to much selection in a particular direction, new variation is likely to produce a disproportionately large increase of the more extreme types in the direction opposite to that towards which selection has been pushing the character. This would still be true when the variation is being increased by irradiation. It seems probable, in the light of man's evolutionary history, that he has been subjected to fairly

intense natural selection for increased intelligence. It might therefore be expected that an increase in the variation, resulting from a raised mutation rate, while leading to some increase in the fraction of those who are highly intelligent, would lead to a greater—perhaps much greater—increase in the other extreme fraction with low intelligence. In addition to the calculation which assumes that the average intelligence score remains constant and so assumes symmetrical increases at the two ends of the distribution, Appendix G includes a calculation which assumes an asymmetrical effect, the overall average of the population falling but the proportion of children of grammar school ability remaining constant. The disproportion in the increase of the low end of the distribution is of course increased, a doubling of the variation raising the proportion with a score of less than 70, perhaps by as much as four or five times. It should be remembered, however, that these calculations apply to the situation when the increase in variation has reached its full extent. We have already seen that such data as are available suggest that a permanent increase in the mutation rate would take hundreds of generations to produce its full effects on hereditary variation.

Observations on Populations Exposed to Radiation

162. An alternative approach to the problem before us, and one which in addition might provide direct evidence of the relation between the dose of radiation and increased incidence of hereditary traits in man, is to observe the effects on human populations which have been exposed to ionizing radiations. Three such studies have been carried out, two on American radiologists and the other on the Japanese populations who were in Hiroshima and Nagasaki at the time of the atomic bomb explosions. For various reasons, the evidence from each is inconclusive, even that from the extensive study by the Atomic Bomb Casualty Commission in Japan.

Possible indicators of change in mutation rates

163. Among the possible indicators of a change in the mutation rate are changes in the sex ratio at birth, the congenital malformation rate, the stillbirth rate, the neonatal death rate, the weight at birth, the weight at nine months, and measurements of the head and body. Changes in the sex ratio may be used as an indicator of genetic damage. The inheritance of abnormal genes in the sex chromosomes has been considered in relation to haemophilia. Experimental observations have shown that abnormal genes which kill the infant long before birth can be carried on the sex chromosomes and there is reason to believe that this may be true in man. Such genes will necessarily disturb the sex ratio at birth ; mothers with such mutations will have too few sons, and, in rare cases, fathers too few daughters.

164. It is known, from both human and experimental evidence, that genes can produce abnormalities which are evident at birth. Estimates of the congenital abnormality rate vary with different observers from just over one per cent to about six per cent, owing to lack of agreement on what shall be reckoned an abnormality as well as to real differences in populations with respect both to their environments and to the frequencies of the causal genes. Further, genes are not the only cause of such abnormal conditions. Both clinical and experimental evidence suggests that maternal ill health, particularly infectious disease and malnutrition during pregnancy, can produce them. There is no definite information as to what proportion of cases of malformation should be attributed to the effects of single genes ; nor are the forms of inheritance understood.

165. The stillbirth and neonatal death rates are also influenced by a variety of factors. The evidence that abnormal genes can play a part is based

partly upon experimental genetics and partly upon investigations of family histories. On the evidence available at the present time, it is difficult to estimate the extent of the part played by genetical causes.

Genetic studies on radiologists

166. The evidence provided by the studies of congenital abnormalities in the offspring of American radiologists is inconclusive for two reasons. First, no measurements were made of the radiation doses which were received by the radiologists in the course of their work and it is virtually impossible to deduce these in retrospect. Secondly, the data were obtained by postal questionnaire, to which only three-quarters of the radiologists replied and little over half the other specialists who were used as a control group. Whether those who chose to answer were representative of the total is open to question. As the magnitude of the effects observed was small (slight rises in the incidence of twinning, foetal death and congenital malformation), one cannot exclude the possibility that the increases were due to statistical bias in the data rather than to the radiation exposure, or alternatively that statistical bias in the other direction may have partly concealed a somewhat larger increase than was observed.

Studies on Japanese populations

167. The Atomic Bomb Casualty Commission's genetic study was much more extensive. An attempt was made to assess the prompt radiation dose received by each individual; and in each city those remote from the burst constituted a control population with which to compare those close to it. More than 80,000 subsequent pregnancies were followed, and a third of the children were re-examined at nine months of age.

168. The final report on the genetic programme of the Atomic Bomb Casualty Commission has not yet been published; but through the courtesy of the United States authorities, and especially of Dr. James V. Neel and Dr. William J. Schull, we received copies of the draft and are permitted to refer to it. The data present many difficulties of interpretation for several reasons. First, the radiation dose was not known with any accuracy. Second, the parents with different degrees of exposure were not entirely comparable in various characteristics, such as maternal age at birth of the child, to which the congenital malformation rate is related; for this reason, even if there were no effect due to exposure, the children of the highly exposed parents would be expected to differ in their congenital malformation rate from those of the slightly exposed. Complex statistical procedures are necessary to allow for this. Even more open to error is the comparison between the children of exposed parents and those of parents who were entirely unexposed. The latter group of parents included immigrants from other cities or from rural areas after the time of the bombing, and some who were away from home at the time, and the effect of these factors on, say, the congenital malformation rate is quite unknown. Thirdly, the number of people who survived high exposures was not large and therefore there were comparatively few births in this group; estimates of the incidence of congenital malformations and other abnormalities are consequently of low statistical precision, being open to relatively large disturbances through the operation of chance. Fourthly, only small effects would be expected in any given generation, even if the mutation rate had been raised many times.

169. Any opinion on a report which is still only in draft must be regarded as provisional. In our view, however, the data suggest an effect of the bomb radiations on genetic factors in prenatal survival, as shown by the sex-ratio at birth. The evidence for this effect is not highly significant statistically

and any change which was induced in the sex-ratio is unlikely to have exceeded 2 per cent per 100 r exposure of one parent. This appears to be the only positive conclusion that might perhaps be drawn but it is possible, for several of the measurements, to set upper limits to the changes that might have occurred without being detected. From the nature of the evidence a possible doubling, but not more than doubling, of the congenital malformation rate, or a 50 per cent rise in the stillbirth rate, following exposure of one parent to 200 r, might have escaped detection.

170. Although it was possible to set an upper limit to the increase in sex-ratio, congenital malformation rate and stillbirth rate, we were unable to do so for the increase in mutation rates of the genes responsible. For this purpose it would first be necessary to know what proportion of prenatal death or malformation is in ordinary circumstances due to newly mutated genes and what proportion to genes already present in the population. We cannot, therefore, derive from the Atomic Bomb Casualty Commission's data any estimate of the mutation-rate-doubling radiation dose for man.

The Radiation 'Doubling Dose' for Human Mutation Rates

171. At this stage it becomes necessary for us to attempt to give a quantitative estimate of the magnitude of the effect of any given dose of radiation on the mutation rate in human populations. This is an extremely difficult task, since not only have we as yet too little precise information on which to base an accurate estimate, but also it is by no means simple to know in what terms the effect should best be measured.

172. We have seen that all genes mutate spontaneously. The spontaneous mutation rate (s) of any particular gene can be considered to be made up of two parts; some of its mutations (x) will be provoked by the naturally occurring radiation, while others (y) will be due to other influences, so that $s = x + y$. It would be easy to find a theoretically adequate measure of the effect of increased radiation on mutation, if all mutations were caused by radiation of some kind, y would then be zero; and, since we have seen (paragraph 127) that radiation-induced mutations increase in simple proportion to the amount of radiation, it follows that, if the amount of radiation were doubled, the mutation rate would be doubled also, and so on. We could express the effect of increased radiation in terms of the 'doubling dose', that is the quantity of radiation required to double the spontaneous mutation rate. It is clear that the doubling dose under these circumstances would be equal to the naturally occurring radiation.

173. However, as we shall see later (paragraph 178), there is good evidence that in the few well-studied animals spontaneous mutations are not due solely to radiation, and therefore we cannot safely assume that y is zero. The situation would remain fairly simple, provided x and y were always in the same proportion, so that we could assume that a certain constant fraction of the spontaneous mutations of each gene is caused by natural radiation; but this also seems unlikely to be the case. Older parents have accumulated higher doses of radiation than younger parents; if all genes were equally sensitive to radiation, the frequency of all new mutations should then show the same increase in the children of older fathers as in the children of older mothers. It is found, however, that for some human genes, but not for others, the increase is more marked in children of old fathers than in those of old mothers. This fact suggests that the genes which exhibit this relationship to paternal age differ from other genes in that their mutation is dependent in an important way on something other than radiation, perhaps on the number

of cell divisions since conception, which is much greater for sperm than for eggs. There is also some evidence from experiments on flies and other organisms, in which high doses of radiation were employed, that certain genes are more radiation-sensitive than others. In view of these facts, it is only safe to assume that the same may be true of human genes, and that for each gene the spontaneous mutation rate is built up of both an x and a y fraction, which do not always bear the same proportionate relation to each other.

174. If this is so, an amount of extra radiation which will double the mutation rate of the most radiation-sensitive genes will have a much smaller effect on the more radiation-tolerant ones. It is then impossible to give any one figure which will measure the effect of radiation on the whole set of genes. However, in practice we still know so little about human mutation rates that we can, provisionally, make some simplification of the theoretical considerations. We can attempt to assess the effects of increased radiation in terms of that dose of radiation which will double the spontaneous mutation rate of an adequate and representative sample of the most sensitive genes. This would be a minimum estimate of the doubling dose. By 'adequate and representative' we mean that we must consider a sufficient number of the more sensitive genes to get examples of all the different kinds of genetic effects. Fortunately there is no reason to doubt that, if one considered a fairly large number of the most radiation-sensitive genes, they would contain examples of genes with all possible kinds of effect. We shall assume that this is indeed so, and, further, that there are sufficient genes with roughly the same degree of radiation-sensitivity for us to employ the concept of a representative doubling dose of radiation of the kind which we have been discussing.

175. The attempt to estimate a figure for the minimum representative doubling dose in man is beset with many difficulties. We have as yet no useful direct evidence. The only data which might provide information about actual increases in human mutation following irradiation are those from the investigations of the results of the atomic bombs in Japan, and those on the offspring of radiologists, and, as we have already seen (paragraphs 162-170), in neither is the material sufficient to lead to any firm conclusions. At the present time, therefore, we are driven to making indirect estimates.

176. Perhaps the most firmly based line of argument towards an indirect estimate is one which leads us to an assessment of a minimum figure above which the doubling dose must almost certainly lie. Let us suppose that all human spontaneous mutations are radiation-induced; then, provided the mutation rate increases in direct proportion to the radiation, the doubling dose would be the same as the quantity of natural radiation received. The only way of escape from this argument would be by the supposition that for human genes the mutation rate is not directly proportional to the radiation, but that they are comparatively insensitive to small doses up to the level of natural radiation, and relatively much more sensitive to doses slightly greater than this. There is no evidence in any other animal for such an effect, but a few experiments in plants, the results of which are not entirely consistent, have suggested an effect of this kind, though only a slight one. It therefore seems safe to argue that, even under the most pessimistic assumption that all human spontaneous mutations are induced by radiation, the doubling dose could not be less than the normal amount of natural radiation. It will be seen, in the chapter on exposure levels, that in this country, over a period of 30 years this amounts to about 3 r to the reproductive organs. We may therefore take this figure as the lower limit of our estimate.

177. The next step is to try to determine whether the actual value of the doubling dose is quite near this limit or considerably above it. There are several ways in which we can proceed. We may first ask whether the Japanese data are compatible with a doubling dose as low as 3 r, or whether, if the value were as low as that, one would have to anticipate rather striking effects in place of the almost complete absence of definitely significant results which was actually observed. Calculations show that, if some more or less plausible assumptions are made, the absence of definitely recognisable effects in the Japanese data does not contradict a doubling dose as low as 3 r, although it is of course more easily accounted for if the real doubling dose is considerably higher.

178. Our lower limit for the doubling dose was based on the supposition that all spontaneous human mutations are caused by radiation. If natural radiation accounts for something less than 100 per cent of spontaneous mutations, then this lower limit would be raised accordingly. In experimental animals, one can determine what fraction of spontaneous mutation is due to radiation by measuring the effects of several different large radiation doses and extrapolating the results to the naturally occurring dose. Even among experimental animals, it is only for fruit flies that we yet have sufficient information to do this with much confidence. It turns out in this case that natural radiation accounts for only about one ten-thousandth (0·0001) of their spontaneous mutations. In trying to extend this result to man, we have to take into account two considerations. The first is that the longer the time elapsing between the conception of an individual and his reproducing, the greater the dose of radiation he will accumulate. The second is that, if the genes are equally radiation-sensitive in two species, the fraction of the spontaneous mutation induced by natural radiation will be smaller if the spontaneous rate is large than it would be were the spontaneous rate small. If one compares the lengths of the pre-reproductive periods of man and flies one finds that man has time to accumulate about 1,000 times as much radiation as the fly. We are much less certain about the comparison of their spontaneous mutation rates, since figures for man are not available for many loci (Appendix C). However, estimates have been made that the human mutation rate is probably about five times as great as that in flies. We should then find that the fraction of the human spontaneous rate due to radiation can be estimated at 1,000/5 times the fraction which holds for flies. i.e 200 × 0·0001, or about 2 per cent.

179. The argument given above is based on the hypothesis that the sensitivity of human genes to radiation, that is the mutation rate induced per roentgen, is the same as it is in flies. As has been repeatedly pointed out, we have no definite evidence about the radiation-sensitivity of human genes, by which this assumption could be checked. The mouse is the only mammal for which we have any evidence on the radiation-sensitivity of genes. The induced mutation rates for a small number of genes have been roughly measured for this species. The experiments suggest that its genes are about ten times as sensitive as fly genes; but it should be noted that this figure depends very largely on only one of the seven genes tested. However, in order to be cautious we may make the hypothesis that mouse genes are ten times as sensitive as fly genes, and that human genes are similar to mouse genes. According to this hypothesis, we must increase our estimate of the fraction of human spontaneous mutation rate due to radiation by ten times, from 2 per cent to 20 per cent.

180. According as we suppose the radiation-induced fraction of the spontaneous mutation rate of man to be 20 per cent or 2 per cent, we arrive

at estimates for the doubling dose that are five or fifty times the naturally occurring radiation, that is 15 r or 150 r. It must be pointed out, however, that the calculations which have just been given have involved a number of quantities which are still only imperfectly known ; for instance, the spontaneous mutation rates of flies, mice and particularly men. One cannot therefore, on this basis, absolutely exclude the possibility that the doubling dose may actually lie somewhat below 15 r.

181. Various other theoretical methods have been suggested for utilising the data about experimental animals to calculate a doubling dose for man. They all lead to values within the same rather wide range as we have just reached. Moreover, they all involve even more conjecture about quantities on which we have little precise information, such as the comparative numbers of gene loci in man and other animals. We shall not attempt to summarise them here.

182. There is another rather different type of approach to the problem ; that is, to compare the values of the minimum representative doubling dose in the animals and plants for which we have the most reliable data (Appendix H). They mostly run from about 25 r upwards, many of them being between 25 and 60 r. Only a few types of organisms have yet been studied in detail, but taken as it stands this evidence would suggest that all doubling doses lie in about the same range, and it is therefore possible that man's may do so too. It is unfortunate, though easily understandable, that none of the fully investigated organisms has a lifetime comparable in length to that of man, and this suggests the necessity of caution in applying the results to man. But one might expect, *a priori*, that evolutionary processes would have acted to reduce the radiation sensitivity of the genes of organisms with long pre-reproductive periods, so that they would have higher doubling doses. Thus, this line of approach would lead us to expect the human doubling dose to lie above 25 r.

183. The discussion in the last few paragraphs has been given at some length in order to bring out the great uncertainty of our present knowledge of the doubling dose for most human genes. Mustering all the arguments at our disposal, we can only come to the conclusion that it almost certainly lies above 3 r, but that it may be as much as 150 r or even more. Any statement which goes beyond this can only be phrased in terms of probabilities, and depends on a judgment made by balancing all the different lines of argument against one another. In this tentative fashion, we should advance the view that there is little likelihood that the representative value lies between 3 r and 15 r ; and that, although we cannot exclude the possibility that for some human genes the doubling dose may be less than 30 r and for others more than 80 r, the best estimate which we can make, in the light of present knowledge, is that the representative value lies between 30 r and 80 r.

184. It remains to consider what dangers would arise if we have, for lack of adequate information, materially over-estimated the value of the doubling dose for human genes. Even if we suppose that it is actually as low as the minimum that we can reasonably entertain, namely 15 r, it is extremely improbable that in times of peace the whole population, or a large fraction of it, will receive an additional dose of this magnitude from industrial or other sources. We need not therefore anticipate any general danger. There may, however, be small groups of people, for instance those employed in certain industrial processes or receiving medical treatment involving X-rays, who may be exposed to doses near the representative doubling dose. Have they grounds for fearing any disastrous effects on their descendants? In the first place, it is obvious that if, for reasons of age or other considerations, they do

not reproduce after the period of exposure, no genetic effects at all will eventuate. If they do reproduce, there are two aspects to be considered: the effect on their immediate offspring and the effect on their later descendants.

185. We shall consider first the immediate descendants. It has been calculated on theoretical grounds that at the present time, without any additional radiation, approximately one human germ cell in ten (10 per cent) carries a new mutation. The great majority of these are recessive, and only very rarely have an effect on immediate offspring. Probably not more than one in a hundred is a dominant, the action of which will be seen in the next generation. Thus, a doubling of the mutation rate might lead to an increase of one in a thousand (0·1 per cent) in the numbers of harmfully affected children in the next generation. This must be compared with the present chance that the children born in a family will be congenitally defective. At present, about four per cent of all babies are stillborn or die shortly after birth, while another two per cent survive but are malformed; and in addition a considerable number in later years develop diseases or abnormalities in which hereditary constitution is a preponderating cause. Thus a doubling of the mutation rate in one parent would only add to the chance of producing a defective child an additional 0·1 per cent. above the present level of about seven to eight per cent.

186. A more realistic estimate of individual genetical risk can be obtained from the figures given in paragraphs 141 to 150. For example, the ordinary risk that any pair of parents will produce an imbecile or an idiot—that is a case of severe mental defect—which survives, is about one in 500. The increased proportional risk for parents in both of whom mutation rates have been doubled is three per cent; this means that the risk of their having a child with severe mental defect which survives is one in 485. If only one parent is affected, the risk would be increased by a factor of 1·5 per cent, so that the chance would then be about one in 493. Similarly, the risk of producing psychotic offspring might be increased by a factor of one per cent if mutation rates were doubled in both parents and by half this amount if only one was affected. The likelihood of miscarriage, stillbirth, or foetal malformation would probably be even less increased; compared with the changes in incidence which occur, for example, at different maternal ages or between the first and later births, these alterations would be inappreciable. The risks of occurrence of specific dominant or sex-linked traits, such as those listed in the table of human mutation rates (Appendix C), would indeed be proportionately much more markedly affected; but, because of their rarity, the risks of these abnormalities are ordinarily considered negligible for the individual and, even after being nearly doubled, they would remain so.

187. In ordinary circumstances, if a parent carries any given allele, the chance that one of his offspring receives it is one in two; a grandchild has one chance in four of receiving it and a great-grandchild has one chance in eight. The same rule applies to an allele which has arisen by fresh mutation in the parent. Thus it follows that the extra risk of disability, which applies to the children of an individual who has been exposed to doses of radiation causing mutation, will be halved in each subsequent generation of his offspring, provided that the level of mutation rate in the rest of the population has not also been raised. Moreover, it must be remembered that under natural conditions every human being already carries a certain number of harmful recessive genes, the results of spontaneous mutation in the past. There is therefore no reason why an individual in whom the mutation rate has been doubled, or increased by some similar figure, need fear that he runs an appreciable risk of founding a 'bad line' of descendants.

188. One may conclude that, if a relatively small group of prospective parents receives a doubling dose of radiation, no noticeable effects will be produced either on their immediate offspring or upon their descendants. For levels of radiation up to the doubling dose, and even some way beyond, the genetic effects of radiation are only appreciable when reckoned over the population as a whole and need cause no alarm to the individual on his own account.

CHAPTER V

EXISTING AND FORESEEABLE LEVELS OF EXPOSURE TO RADIATION

Introduction

189. Throughout the whole of his evolutionary history man, like all living organisms, has been exposed to small but variable amounts of ionizing radiation from his natural surroundings. To these he has now added similar radiations from his own inventions. In their biological action these differ but little from each other, and all must be taken into account when assessing the present hazards from ionizing radiation. We shall first consider those inescapable radiations which come from the natural background and, thereafter, those which are derived from sources controllable by man.

190. It will be clear from an earlier chapter of this report that relatively heavy doses of radiation are required to impair the health of the individual and such doses are rarely associated with the ordinary circumstances of civilian life. The use of radiation for medical purposes or occupational exposure to sources of radiation may be associated with the possibility of high doses, but every precaution is taken to safeguard the patient, and the employee is protected by nationally and internationally recognised recommendations which limit the doses received occupationally to levels considered to be safe.

191. On the other hand, our knowledge of the genetic effects of radiation is less precise and it is believed that doses of radiation which have no known effects on the health of the individual may be of genetic consequence. Doses received from all sources, however small, have therefore to be assessed in the light of their possible genetic implications. It has been seen that ionizing radiation can have genetic consequences only in so far as it affects the reproductive organs—the gonads—and it is thus the dose received by the gonads up to the end of reproductive life which must be estimated in all cases. At the levels of dose with which we are mainly concerned, the genetic effects of radiation can be calculated only in relation to the population as a whole. It is therefore in terms of the total gonad dose* to the population that the following estimates of the exposures from various sources have been made

Radiation from Natural Sources

Cosmic radiation

192. Cosmic radiation reaches the earth from interstellar space. The atmosphere surrounding the earth has substance and acts as a filter, absorbing almost all the dose to which otherwise we should be exposed. In general, the longer their path through the atmosphere, the more the radiations will be attenuated ; thus, the dose at sea level is less than that at high altitudes.

---

* The total gonad dose has been calculated on the basis of the considerations set out in paragraph 129.

193. Cosmic radiation has several components—protons,[1] fast and slow mesons,[2] electrons[3] and neutrons[4]—which differ in their relative contribution to the natural background of radiation according to the altitude; at sea level the most important are mesons, electrons and neutrons. Mesons and electrons are considered to be, for equal physical doses, of about the same biological effectiveness as gamma rays. Fast neutrons and protons of high energy may be several times more damaging to the individual but are less likely to induce gene mutation. Neglecting this possible variation in effectiveness and assuming equal biological efficiency for all the particles, one derives a dose at sea level equivalent to 0·028 r per annum from cosmic radiation. Virtually all of this is highly penetrating radiation and can be assumed to irradiate the whole body, including the gonads, almost uniformly.

Terrestrial radiation

194. A few of the naturally occurring elements, particularly the heavy elements, are radioactive, thorium and uranium being the chief primary sources. These two elements are only feebly radioactive and each has a half-life measured in many millions of years. Each atom, in its radioactive disintegration, is transmuted to a daughter atom which is also radioactive and which in turn disintegrates to another radioactive atom, the process being continued until ultimately a non-radioactive stable atom of lead is formed. All the daughter elements decay much more rapidly than the original parents, thorium and uranium, and many of them emit gamma rays as well as nuclear particles in their disintegration.

195. Thorium and uranium are almost universally distributed in trace quantities in rocks and soils, areas of granitic rock usually having higher concentrations than sedimentary rock. Occasionally, the concentration is considerably greater than normal, sufficient to make the area worth mining, but even in these rich lodes it is usually only of the order of one per cent or less.

196. As a consequence of the presence in many soils of the radioactive daughter products of uranium and thorium, emission of gamma rays occurs widely over the land surfaces of the earth. Brick and stone necessarily contain traces of these radioactive substances and, inside houses built of such materials, radiation is added from this source. On the other hand, substantial structures of brick and stone offer slight shielding from cosmic radiation.

197. The amount of radiation contributed from the earth and from buildings varies from place to place even in the same country and any average figure can be only an approximation, but 0·078 r per year would perhaps be representative of the amount received inside buildings on the surface of the body by the inhabitants of this country. The corresponding figure in the open would perhaps be 0·048 r per year. Measurements show that about 37 per cent of the dose of gamma rays is absorbed superficially and filtered off before reaching the internal tissues and organs: allowing for this and esti-

[1] *Protons* are nuclei of hydrogen atoms and carry a unit positive charge. Fast moving protons are a minor component of cosmic rays at ground level. Neutron radiation also ejects protons from hydrogenous material.

[2] *Mesons* are unstable particles with masses intermediate between those of electrons and protons. Energetic mesons constitute the main component of the more penetrating cosmic rays.

[3] *Electrons* are the smallest constituent particles of atoms. The electron carries the elementary negative charge of electricity. It has a positively-charged counterpart of equal mass called the positron. The soft or less penetrating component of cosmic rays consists essentially of positrons and electrons and about an equal number of gamma rays.

[4] *Neutrons*, see Chapter II, paragraph 19.

mating the amount of time that a person spends in the open, the average dose to the gonads of persons in this country from external gamma radiation is estimated to be 0·043 r per year.

Atmospheric radon

198. One of the decay products of uranium is radon, a gas which diffuses out of the earth and buildings and from minerals such as coal. In general, its concentration in the atmosphere is extremely low, about 0·3 of a micro-microcurie* per litre of air, but in cities such as London, where much coal is burnt, it may sometimes reach ten times this amount. In these circumstances, the dose of gamma rays from the further disintegration-products of radon may almost equal the dose of radiation from cosmic sources. The average dose to the gonads from this source, however, probably does not exceed 0·001 r per year from the atmosphere and an approximately equal or slightly greater amount from the gas absorbed into the body from the lungs.

Radioactive constituents of the body

199. Among the normal constituents of the body are the elements carbon and potassium, each of which has a radioactive isotope occurring naturally as a minute fraction of the total element. The radioactive isotope potassium 40 forms 1/8000 of natural potassium and emits both beta particles and gamma rays. An average value for the potassium content of the body is 0·21 per cent by weight, and measurements suggest that the figure for the gonads does not differ greatly. Calculation on this basis gives an estimated dose to the gonads of about 0·02 r per year. Naturally occurring radioactive carbon, carbon 14, constitutes one part in a million millions (10^{12}) of natural carbon ; it emits only beta particles. Taking body tissue to be 18 per cent carbon, one derives a dose from this source of about 0·001 r per annum to the gonads.

Total gonad dose from natural sources

200. Information on the total dose to the gonads from natural sources of radiation is summarised in Table 2. It will be seen that, from all sources, the total is roughly 0·1 r per annum, or about 3 r per generation of 30 years (Appendix J).

TABLE 2

Estimated dose rates to gonads from natural sources of radiation

Radiation source	Estimated average dose rate to gonads in roentgens per year
External radiation	
Cosmic rays (sea level)	0·028
Gamma rays from the earth	0·043
Radon in air	0·001
Internal radiation	
Potassium 40	0·020
Carbon 14	0·001
Radon and decay products	0·002
Total	0·095

* $\frac{1}{1,000,000,000,000}$ of a curie.

Radioactivity in bone

201. Within the body, radioactivity is probably highest in bone. The naturally occurring radioactive heavy elements are contaminants of food and water as well as of soil and they and their disintegration products, of which radium is the most important biologically, are absorbed to a very limited extent from the gut. The body removes them from the circulation and stores them in bone. In this way the radioactivity of bone builds up gradually throughout life but there is no evidence that the rather higher doses in bone compared with other tissues are deleterious. Table 3 gives the estimated doses to bone from these sources.

TABLE 3

Estimated radium content and radiation dose rates to bone-cells

Geographical conditions	Estimated radium in skeleton at age 35 in micro-microcuries	Dose rate to bone-cells in equivalent roentgens per year		
		radium	external sources	total
Average areas	60	0·037	0·08	0·12
Active areas ... ... (See Appendix J)	1,100	0·37	0·18	0·55

Radiation from the Appurtenances of Civilisation

202. Since the discovery of X-rays and radioactive materials man has adopted them increasingly for certain of his needs. In medicine they now provide invaluable, and often irreplaceable, aids to diagnosis and treatment. They are also used extensively in industry and in the amenities of modern civilised life. It is necessary, therefore, to assess the dose that these developments contribute above that received from the natural surroundings.

Diagnostic X-rays

203. For some time it has been realised that by far the largest contribution is made by diagnostic X-rays. Continuing efforts are therefore being made to assess the dose of radiation given to the whole population in this way. The problem of accurate assessment is beset with difficulties and at present any estimate must be based upon very imperfect data. The two basic requirements and the best means of fulfilling them from available figures are as follows:

(i) *The number of radiological examinations made annually, subdividea according to the sex and age of the persons examined and the part of the body under examination.* It is possible to make from published figures a reasonable estimate of the total number of X-ray examinations made within the National Health Service. A minimum figure for the year 1955 would be 12,200,000. To this must be added an unknown but relatively small number for hospitals outside the National Health Service and for private practice. This number has been estimated at rather less than half a million, giving a total of 12,650,000.

The division of this very large figure into sex, age and type of radiological examination has been made on the basis of information supplied by five hospitals only and covering some

21,000 patients (two London teaching hospitals and three others). While there are certainly differences between these hospitals, they are not so wide as to make an approximate calculation unwarranted. The sample has therefore been accepted as representative.

A very important source of radiation to the gonads is provided by radiological pelvimetry, which measures the size of the pelvic outlet of the woman in relation to the actual or potential size of her infant; a second important source is X-ray examination of the abdomen in pregnancy. Information of the frequency with which such examinations are made has therefore been specially sought from a rather wider group of nine hospitals. On the assumption that these are representative, the results have been applied to all live births occurring in hospitals in England and Wales.

In addition to the 12,650,000 X-ray examinations already mentioned, certain others are undertaken for special reasons and these bring the estimated total during 1955 up to nearly 18,000,000. The additional examinations include mass miniature radiography, dental radiography, and examinations of service personnel and mineworkers, but each of these types of examination represents a relatively unimportant source of radiation to the gonads and the effect of any error in estimating their contribution will be slight in relation to the total.

(ii) *The average dose to the gonads—male, female and foetal separately—produced by the diagnostic irradiation of each separate part of the body.* For the purposes of calculation the figures used, which cover X-ray examinations of 24 different parts of the body, have been derived almost entirely from one London teaching hospital where careful measurements have been made and precautions taken to restrict the irradiation of the gonads to the lowest possible level. They are therefore almost certain to be minimum figures. It should be realised that with the X-ray tube only slightly misaligned the dose may sometimes be multiplied many times.

204. Bringing together these two sets of figures—the estimated numbers and ages of persons irradiated and the estimated average gonad dose delivered to them with specified examinations—leads to two important general conclusions. It shows, first, that almost the whole population dose is accounted for by a relatively few sites of examination, principally the hip, the lumbar spine, the lower abdomen and the pelvis. The far more frequent examinations of chest, head and limbs make relatively unimportant contributions. Secondly, according to the present calculations, the amount of radiation reaching the reproductive organs of the people of this country through diagnostic radiology is as much as 22 per cent of that derived from natural sources. Indeed, in view of the minimum figures adopted in these calculations, the contribution of diagnostic radiology may well be very considerably higher than 22 per cent. It undoubtedly forms the most important source of man-made irradiation and its application has been steadily increasing in recent years (Appendix K).

Radiotherapy

205. At present there is little information about the contribution to the population dose from therapeutic irradiation. Although its main use is for patients with malignant disease, the majority of whom are beyond the child-bearing age, some younger patients are treated for non-malignant conditions such as ankylosing spondylitis. Less penetrating X-rays are widely used in the treatment of a large number of diseases of the skin, and the artificially

produced radioactive element iodine 131 is now being administered for hyperthyroidism.

206. It is as yet not possible to state a figure for the population dose to the gonads from this source of radiation. Rough assessments would suggest that it is considerably less than the dose from diagnostic radiology but probably greater than that from any other source. The problem is one upon which research is required.

X-ray fluoroscopy for shoe-fitting

207. X-rays are used commercially for fitting shoes but, with modern equipment and good practice, it appears that the number of machines in operation would probably deliver not more than 0·1 per cent of the dose to the gonads received from natural radiation.

Luminous watches and clocks

208. Watches and clocks with luminous dials depend for their luminosity upon the rays from radium or other radioactive material used in the paint. Measurements and calculations suggest that the average wrist-watch contains about one-fifth of a microcurie of radium. A calculated dose to the gonads from wearing such a watch is about 0·01 r per year.

209. From the information given by the trade, it can be deduced that there are in use about three million men's watches and about a million women's and children's watches with luminous dials. In addition, there may be about ten million luminous alarm clocks. On this basis it can be estimated that the population dose from this source is about one per cent of the natural background.

Television sets

210. Cathode-ray tubes for television sets are capable of causing the production of X-rays. In general, however, the operating voltages are comparatively low and the X-rays are readily absorbed by the walls of the tubes and by protective screens. It can be estimated that the population dose from this source is at present much less than one per cent of the dose received from natural radiation.

Cosmic radiation in aircraft

211. Since the amount of radiation from cosmic sources is greater at high altitudes, the doses received by persons in aircraft have been investigated and the population dose calculated. The additional dose averaged over the whole population is at present insignificant compared with that received from the natural background.

Occupational Exposure to Radiation

Medical and industrial workers

212. Men and women have been exposed to ionizing radiation in the course of their occupations for over half a century. In the early years after the discovery of X-rays and radioactive materials many suffered injury, but safe practices have gradually been elaborated and standards of safety laid down. In this country since 1921 the British X-ray and Radium Protection Committee, and later the Medical Research Council's Committee on Protection against Ionizing Radiations, have considered the available information and made periodic recommendations on occupational exposure levels. Equivalent levels have been advocated by the International Commission on Radiological Protection.

213. Control of radiation exposure may be effected in two ways. In the first, the intensity of radiation in the vicinity of the source is measured at frequent intervals and, provided that the intensities are always below those levels accepted by international agreement as being without danger, no separate check is required on the exposure of the individual. In the second, the doses actually received by the individual are recorded, usually by means of a photographic film which he carries and which blackens on exposure to radiation, so allowing the dose received, if any, to be estimated. For twenty years the National Physical Laboratory has provided a service whereby such films are issued on demand and subsequently read. The worker and his supervisor can thus keep a check on the doses received to ensure that the accepted weekly levels of dose are not exceeded.

214. The provision of film badges has now been taken over by the National Radiological Protection Service which serves the majority of people known to be occupationally exposed, other than those employed by the Atomic Energy Authority or in the many hospital departments which process their own films. The Service will be available to those employed in the many and increasingly varied industrial applications of ionizing radiation as well as to research laboratories.

215. The available records have been sampled and analysed to assess the total dose from these sources to the population but, as precise records are not available from all branches of industry, the contributions from some sources are estimates only. It has not been possible to make as accurate an assessment as for the employees of the Atomic Energy Authority because the number, sex and ages of those exposed are not known with any precision and the monitoring, when carried out, is not as complete.

216. On the basis of figures from the hospitals which make use of this service, it has been roughly calculated that about 60 per cent of the medical workers at risk are women ; in industry and research, women constitute only 15 per cent. It is estimated that in total about 14,000 people are employed, of whom half are women. After allowance for the fact that in women radiation is more completely absorbed before it reaches the gonads, it is estimated that the average gonad dose for both men and women would be about 2·5 r per year. Because the ages are not known with precision, it is not possible to make an accurate estimate of the genetic dose to the population as a whole, but after making certain assumptions we have reached a figure from this source of 1·6 per cent of natural background radiation (Appendix L).

Atomic Energy Authority employees

217. The Atomic Energy Authority now employs about 7,000 people who are exposed or potentially exposed to radiation in the course of their work. All employees liable to be exposed wear film badges that are examined weekly or monthly, and the sex and age of each individual are known. Thus it has been possible to calculate, with considerable accuracy, the doses received by the employees in relation to their expectation of parenthood. The average dose to the Authority's employees from all occupational sources of radiation is 0·4 r per year. The results of personnel monitoring show that in all recent years no employee has had an average weekly dose exceeding the maximum permissible and that about 90 per cent of the persons exposed to radiation averaged less than one-tenth of the maximum permissible weekly dose. It has been estimated that the gonad dose averaged over the population as a whole is about 0·1 per cent of the natural background.

Contamination of the World by Fall-out from the Explosion of Nuclear Weapons

218. Nuclear weapons differ in their construction and size and the same type of weapon can be detonated in different ways—under water, on land or in the air. These variations lead to differences in the radioactive dust produced, in its distribution and in the rate at which it falls out from the atmosphere on to the earth. Except in the immediate vicinity of a nuclear weapon explosion, the ionizing radiations to be considered arise from radioactive particles.

Radioactive fission products

219. Radioactive fission products, formed when the atoms are split, become mixed with the vapourised material from the bomb and with any earth, water or debris caught up in the explosion. Large particles fall quickly and are deposited close to the site of explosion ; small particles are carried up with the hot gases to heights which vary with the power of the explosion. They subsequently travel in air streams for considerable distances and times, depending on the size of the particles and the height to which they were carried.

220. The particles reaching this country from the distant explosion of a typical nuclear weapon detonated over land are spherical in shape and consist of fused silica and metallic oxides impregnated with fission products. The vast majority are smaller than 0·001 of a centimetre in diameter. Particles from the thermonuclear tests in the Pacific atolls differ, in that they consist of calcium oxides or carbonates from coral and are irregular in shape. Both varieties are radioactive.

221. Different types of weapon produce a similar mixture of fission products in slightly different proportions, but the overall rate of decay of radioactivity is almost the same in all types. The total radioactivity decays to one-tenth of the original level for each seven-fold increase of time in days, as measured from the moment of detonation ; thus, if the radioactivity is one unit on the first day after detonation, it is 1/10 unit at seven days, 1/100 unit at 49 days, and so on.

222. At the time of atomic explosions some normally stable elements may become radioactive by virtue of the capture of neutrons. Such induced activities are for all practical purposes short-lived and therefore of little importance when long-term hazards are being considered.

223. Since January 1951, continual watch has been kept by the Atomic Energy Authority on the radioactive fall-out reaching this country from nuclear devices exploded in other parts of the world. The activity in rain water at selected sites is recorded continuously. In addition the atmosphere is sampled daily by the collection of dust on a cylindrical filter through which about 1,500 kilogrammes of air are passed, and the radioactivity of the filter papers is determined (Appendix M).

Radioactivity in air

224. When the ordinary type of atomic bomb is exploded in Nevada, the dust-cloud rises to a height of about 40,000 feet. It then travels eastwards with the winds which prevail at that height and diffuses both vertically and laterally. It may or may not pass over this country on its first circuit round the world ; if it does so, it will usually appear about 5 days after the explosion. The cloud continues circling the earth, and peaks of activity can be detected over any particular place in its path at about monthly intervals. The total radioactivity per unit volume of air falls progressively with time

owing to decay of the radioactive elements, to increased spread of the cloud and to deposition on the surface of the earth; approximately half the available material is deposited every 22 days.

225. Clouds from thermonuclear explosions behave differently because of the far greater height, approximately 100,000 feet, to which the debris is carried. Diffusion downwards from the stratosphere is a very slow process, and months after a thermonuclear test explosion most of the radioactive debris is still at these great heights.

226. Dust clouds from distant tests passing on the first circuit over this country are usually too high to impart measurable activity to air at ground level. Subsequently, at the peak periods, concentrations are in the region of five radioactive disintegrations per minute per cubic metre (dpm/m^3) of air. From April, 1952, to December, 1955, the mean concentration of activity from all bombs exploded in that period was 0·5 dpm/m^3. The corresponding average activity from naturally occurring radon decay products in the air was measured at one of the sampling stations and found to be 130 dpm/m^3. The debris from the thermonuclear tests in the Pacific in 1954, much of it by now already decayed, has mostly still to come down, but it is not expected to exceed 0·1 dpm/m^3 in the next few years. The dose to a person fully exposed to air with a radioactivity of 0·5 dpm/m^3 has been calculated to be one millionth of a roentgen per year.

Deposited radioactivity

227. The radioactive fall-out is cleared, sooner or later, from the air by deposition. Rain contains the bulk of deposited activity and continuing measurements have been made since 1951 of the radioactivity of rain water collected from specially treated roofs. Any radioactive dust deposited on the roofs in spells of dry weather is washed off and included with the next sample of rainwater. From these measurements the amount of radioactivity deposited per square mile can be determined for each explosion.

228. The dose that a man standing in the open in this country would receive from the deposition of radioactivity from all bombs so far exploded has been estimated. It has been assumed that all the radioactivity remains on the surface of the earth and that none is lost, as we know some will be, by drainage and weathering. Including all ordinary atomic bombs exploded before December, 1955, and calculating all the radioactivity which they have contributed and will contribute over the next 50 years, it is found that the total dose which a man continuously out of doors, night and day, would receive is 0·005 r. To this dose from ordinary atomic bombs must be added the dose from thermonuclear weapons. For these latter the dose from the radioactivity still to be deposited is more important. It can be estimated that the accumulated dose from thermonuclear weapons is 0·002 to 0·003 r with another 0·027 r still to come.

Total radioactivity from weapons already exploded

229. All these doses together add up to about 0·035 r from weapons already exploded. This is a maximum dose. The loss of radioactivity from weathering has not been taken into account, nor has the protection afforded by buildings in and around which most people in this country spend a large part of their lives. It would be realistic to divide the dose by three for weathering and by seven for protection afforded as a result of time spent in houses. The average inhabitant of this country may therefore receive in the next 50 years between 0·001 and 0·002 r from this fall-out, or 0·02 to 0·04 per cent of the radiation that he will receive during the same period from natural surroundings.

230. If the firing of both types of bomb were to continue indefinitely at the same rate as over the past few years, there would be a build-up of activity gradually reaching a plateau in about a hundred years time which, on the same basis of calculation, would give the average individual a dose over a period of 30 years of 0·026 r or about 0·9 per cent of what he would receive in the same period from natural sources.

231. The most impressive feature of these figures for exposure from fall-out is the very great effect of a very few thermonuclear explosions. This is not surprising since their power is measured in equivalents of millions of tons of TNT compared with the thousands of tons for atomic bombs. If the rate of firing this type of weapon increases, the radiation exposure will be altered proportionally.

SPECIAL HAZARDS FROM RADIOACTIVE FISSION PRODUCTS

Particulate contamination

232. The total radioactivity in the air from fall-out is measured daily, and determinations are made of selected individual radioactive substances. The activity will arise from particles which may be inhaled into the lung. It can be calculated, however, that not more than one or two particles of the more highly active substances are breathed by any person in the course of a year (Appendix M). Although the radioactivity of these particles is minute, it is concentrated in a few million-millionths of a cubic centimetre (10^{-12} c.c.) and the possibility of their creating a hazard must therefore be considered. The International Commission on Radiological Protection has concluded that the critical volume of tissue to be taken into account is of the order of one cubic centimetre, which is a larger volume than could be heavily irradiated by a few such radioactive particles. It seems unlikely that particulate contamination of the air from the fall-out from test explosions would constitute a problem in ordinary civilian life.

233. Radioactive material from the air is deposited and accumulates on the ground where it may contaminate drinking water and agricultural crops. After deposition it becomes possible to make routine measurements of the present concentration of the more dangerous radioactive substances. By December, 1955, this concentration amounted to 0·011 curie of strontium 90 and 0·0002 curie of plutonium 239 per square mile. The continuing fall-out from explosions which have already taken place will cause a rise, to a maximum by about 1965, of around 0·045 curie of strontium 90 per square mile. These figures should be viewed against the background of the fact that the top one foot of soil has always contained on the average about one curie per square mile of the equally, if not more, dangerous naturally occurring radium.

Strontium 90

234. From the point of view of general contamination of the world, the hazard from deposited strontium 90 might, according to present ideas, be greater than that from external radiation, since strontium, like radium, is ordinarily retained in the body and deposited in bone. The average concentration of radioactive strontium in rain water over a period of three years ending December, 1955, was 1·7 micro-microcuries per litre and, since most water passes through soil before being drunk, the activity reaching human beings through drinking water is extremely small.

235. Strontium 90 is deposited on herbage and soil and is then absorbed into plants. Man and animals consuming the leaves of plants will therefore receive strontium 90 in food as well as in water. The hazard is greater for

grazing animals than for man, since animals may crop herbage from wide areas of contaminated pasture. Man, moreover, relies for his food more on grains and roots, which are not sites of concentration of strontium, and on animal produce from which the animal has removed most of the strontium to its bones. Cows' milk contains strontium, but fortunately the cow in its metabolic processes secretes calcium into the milk in preference to strontium.

236. The importance of radioactive strontium, compared with other long-lived fission products produced by exploding nuclear weapons, derives from four factors ; its relative abundance among the fission products, its facility for following calcium through the human food chain, the ease with which it is absorbed, and the fact that, once absorbed, it is stored for long periods in the bones of the body. In bones it forms more or less localised deposits which, judging by animal experiments and according to analogy with the action of radium compounds on human subjects, can if present in sufficient amounts give rise to bone tumours or, by irradiating the neighbouring bone marrow, to aplastic anaemia or leukaemia. There is evidence that the young are more susceptible to its action than adults. Such measurements as have been made of strontium 90 in human bone suggest that the highest levels are at present about a thousand times less than is considered permissible for those occupationally exposed.

Plutonium

237. Plutonium 239, another very long-lived radioactive element, is also a potentially dangerous contaminant since, like strontium, it is deposited in bone. The amount of activity from plutonium in fall-out is small relative to that of strontium 90, its solubility is low, and less than one-tenth of one per cent of the amount taken by mouth is absorbed. The hazard from plutonium in fall-out debris is thus very small.

Caesium

238. Very sensitive methods of measuring the radioactivity of the human body have now been developed and, on the records obtained in recent months, there has been some indication of gamma radiation suspected to be due to the long-lived fission product caesium 137. Although it is not possible yet to identify with certainty the source of this radiation, calculations have been made on the assumption that it is due to this isotope. Caesium is not concentrated in any particular organ of the body and, on the basis of present information, is unlikely materially to affect the figure given above for the dose from the fission products deposited on the ground.

Other important isotopes

239. Of the other elements in mixed fission products strontium 89 and barium 140 behaves similarly to strontium 90 ; iodine 131, which is easily absorbed, is highly concentrated in the thyroid gland. These three isotopes, having half-lives which are only a matter of days, are of very little significance in relation to the long-range fall-out from atomic bombs, since most of their radioactivity will have decayed before they are deposited. In relation to the heavily contaminated areas within a few hundred miles of the explosion of a thermonuclear bomb, however, they will be of great importance.

TOTAL GONAD DOSE FROM MAN-MADE SOURCES OF RADIATION

240. Information on the total dose to the gonads from man-made sources of radiation is summarised in Table 4, the dose from each source being expressed as a percentage of the dose received from natural sources. It will be seen that the total dose amounts to approximately 25 per cent of that already received from the natural background.

TABLE 4

Summary of estimated population doses of radiation to the gonads expressed as percentages of natural background

Source of Radiation	Approximate dose to gonads as a percentage of natural background
Natural background	100
Diagnostic radiology	at least 22
Radiotherapy	?
Shoe-fitting	0·1
Luminous watches and clocks	1
Television sets	much less than 1
High altitude flying	insignificant
Occupational exposure:	
Radiology and Industry	at least 1·6
Atomic Energy Authority	0·1
Fall-out from test explosions	less than 1

Nuclear Warfare

241. Atomic bombs were developed for their capacity to create blast, which was the chief cause of casualties at Hiroshima and Nagasaki. The additional effects of the detonation of a nuclear weapon are due to the release of other forms of energy—heat and ionizing radiation. The ionizing radiations produced by the weapons are the new feature of military operations. Of the prompt radiations, produced at the moment of explosion, neutrons are the direct result of the process of nuclear fission. Gamma rays are also a by-product of fission but most of these are produced immediately after the detonation by the enormous quantities of radioactive fission products. The rapid ending of the gamma-flash at ground level after an explosion is due partly to the ephemeral nature of the radioactivities of many of the fission products and partly to the very rapid removal of the debris in the up-draught of hot gases.

242. The effective range of the prompt ionizing radiations from an ordinary atomic bomb explosion is less than that of the thermal radiation, and at Hiroshima and Nagasaki the range within which death and severe injury from ionizing radiations were encountered was about one mile, as compared with up to three miles for severe flash burns and five miles for indirect blast effects.

243. The notable feature of the ionizing radiations is that, in contrast to the heat rays, they are very penetrating. Clothing which may be adequate to shield the body from heat flash is ‘ transparent ’ to these rays, and even four inches of concrete transmit half the radiation at a distance of one mile from the atomic bomb burst. Thus, at ranges relatively close to such a weapon, people in stoutly constructed buildings might survive the effects of the heat and blast waves but suffer from the damaging effects of the penetrating gamma rays and neutrons.

244. From the comparative ranges of the heat flash and ionizing radiation it will be seen that distance is a factor of great importance. With gamma-rays, as with heat, the intensity falls in proportion to the square of the distance and is diminished by an attenuation factor. It is unlikely that the prompt radiations from thermonuclear weapons would be relatively more significant than those from atomic weapons. The hazard from radiation is

therefore only one of the immediate effects of nuclear weapon explosions, and a relatively minor one in a holocaust. Its particular importance lies in the delayed and distant effects, which arise from the radioactive fission products.

245. At Hiroshima and Nagasaki the atomic bombs were exploded high in the air so as to obtain the maximum effects from blast. Virtually all the fission products were therefore carried up with the hot gases and must have been enormously diluted before being deposited gradually round the world. However, some did fall to earth locally and presumably contributed to the delayed effects recorded by the Atomic Bomb Casualty Commission.

246. When bombs are exploded in or near the surface of the ground or near the surface of water, much debris highly contaminated with fission products is flung into the air and the large particles are deposited more or less locally. Some of intermediate particle size is carried by the local winds and gradually diffuses and settles down-wind. The best known phenomenon of this character following a test-explosion arose from the thermonuclear device exploded on 1st March, 1954. According to the Press release in February, 1955, of the United States Atomic Energy Commission, the area over which fission products settled to give a radiation dose which might well have been lethal to a man unable to take shelter was about 7,000 square miles. Over a considerably larger area, conditions in the open would have been hazardous to man and beast. The size and shape of these lethal and hazardous areas will vary with the conditions of the explosion and of the local meteorology. Nevertheless, the inferences are plain: weapons such as these can be devastating, not only locally over areas measuring hundreds of square miles, but in their more distant effects, which may occur over thousands and tens of thousands of square miles.

CHAPTER VI

ASSESSMENT OF THE HAZARDS OF EXPOSURE TO RADIATION

247. We have reviewed the effects of radiation both upon the exposed individual during his own lifetime and upon his descendants. It is now necessary to relate these effects to dose levels, and to attempt to assess the consequences of exposure to those levels of radiation which now occur or might conceivably come about in the future.

248. It will be recalled that human beings have always been exposed to radiation from outer space and from traces of radioactive materials in their surroundings and in their own bodies. The problem is, therefore, to assess the effects of any additional radiation to which they are or may be subjected, rather than to define a new experience. Our ignorance of the results of exposure to radiation is great and much intensive research is required in many fields ; but the naturally occurring background of radiation to which mankind has long been adjusted can be taken with some confidence as a safe standard of reference, whether we are considering irradiation from outside the body or from radioactive materials taken into the body and stored, temporarily or permanently, in its tissues.

Differences between individual and genetic effects

249. Different considerations enter into the assessment of the significance of any particular level of exposure, according to whether we are concerned with effects on the individual or with genetic effects. In respect of the effects on the individual, we are concerned with doses received throughout the whole of life. In respect of genetic effects, however, we are concerned only with doses to the gonads received up to and during the reproductive period of life. In this country the average age of mothers conceiving children is about 28 years ; of fathers, two to four years older. We have therefore taken the period of 30 years to represent the average length of time during which the germ cell lineage of the human population is effectively exposed to radiation. From a genetic point of view, any exposure to radiation after reproduction has ceased is irrelevant, since any mutations produced will not be passed on to future generations.

250. The second important difference relates to the accumulation of the effects of exposure on separate occasions. The genetic effects of radiation are cumulative ; a mutation persists once it has been produced in a germ cell lineage, and to this are added any further mutations that are induced in the same reproductive cells. Since dose and effect are proportional, we are concerned, from the genetic point of view, with the total dose of radiation which has been accumulated up to any particular time in the reproductive period of life.

251. The position with regard to the effects of successive doses on the individual is incompletely known. It is certain that a dose of radiation which would produce acute effects if given as one single exposure may produce no similar effects if spread over a longer period. Moreover, if a sufficient period has elapsed after recovery from the acute effects, the individual may again recover from a further exposure which produces acute symptoms. Uncertainty

arises in relation to some important delayed effects of radiation where, in view of the results on the increased incidence of leukaemia in repeatedly irradiated cases of ankylosing spondylitis, we must now entertain the possibility that repeated exposures to radiation may combine to produce certain irreversible changes in the tissues exposed.

Dosage and Effects on the Individual

Acute effects

252. The effects which appear within the first 48 hours are seen only as a result of accidents and in those within a short distance from an atomic bomb explosion, or occasionally after exposure to the heavy doses of radiation which may be necessary in the treatment of serious illnesses by radiotherapy. They are, therefore, not important in ordinary civilian circumstances.

253. The same considerations apply to the acute effects on the blood occurring up to two months after exposure to a single dose or to a few heavy doses of radiation. Similar effects occurring later can, however, be produced by doses of radiation, given continuously or repeated at short intervals, which would not be sufficient to produce any symptoms in the first few hours. Formerly, it was thought that continuous exposure to 1·0 r per week would produce no effects on the blood but, after some individuals were found to be affected by this dose, the figure recommended by national and international bodies as the maximum permissible level was reduced to 0·3 r per week.

Delayed effects

254. Of the delayed effects, the one about which we have most information is leukaemia. It also appears to be the most easily induced and seems, at present, to be the most important as far as radiation of the whole body is concerned. We have therefore taken the incidence of leukaemia as a measure of the doses of radiation that are capable of producing delayed effects. The statistical evidence indicates that an increased incidence of leukaemia can be demonstrated after exposure to doses of radiation which might, in exceptional circumstances, be met with in civil life. For example, after either a single exposure of 200 r, or a few exposures which in total amount to 200 r, there is a noteworthy increase in the small chance of developing this disease. What we do not know for certain is whether there would be an increase if a total dose of 200 r were spread over many years. Be this as it may, however, any risk that there may be from such a dose appears to be within the range of risks of other kinds commonly incurred in industrial and professional life.

255. We consider, therefore, that an individual could, without feeling undue concern about developing any of the delayed effects, accept a total dose of 200 r in his life-time, in addition to radiation from the natural background, provided that this dose is distributed over tens of years and that the maximum weekly exposure, averaged over any period of 13 consecutive weeks, does not exceed 0·3 r. We recommend, however, that the aim should always be to keep the level of exposure as low as possible.

Internal radiation

256. The problem of irradiation by radioactive materials taken into the body is in some ways a more complex and difficult one. The material is often concentrated in a particular tissue, such as bone, where it may give rise to malignant change. Owing to the short distance that the rays from a particular concentration of radioactive material can penetrate the tissues, the dose of radiation is extremely variable from place to place in the body.

Nevertheless, on the basis of the known levels of external radiation that can be tolerated, and of experience gained from the accidental ingestion of substances such as radium, 'permissible' levels of exposure for a large number of radioactive isotopes have been agreed. No single level that comprehends them all can be given, since each isotope emits its own characteristic amount and type of radiation, dies away at its own rate, and is absorbed and excreted at a rate dependent on its chemical form and its method of entry into the body. However, it has now been possible to estimate for many different isotopes the concentrations that it is safe to accumulate.*

Dosage and Genetic Effects

257. Our conclusion in the chapter on genetics was that 'For levels of radiation up to the doubling dose, and even some way beyond, the genetic effects of radiation are only appreciable when reckoned over the population as a whole and need cause no alarm to the individual on his own account'. In other words they are essentially problems for society as a whole, to be assessed in terms of the load of medical care that may, in different circumstances, be imposed on the population.

258. In ordinary circumstances only a small fraction, perhaps one or two per cent, of the hereditary abnormalities which appear in a generation can be attributed to fresh gene mutations. For the offspring of any given parents the risk from increasing the mutation rate is very slight. Nevertheless, if the whole of a large population is exposed to enough radiation appreciably to affect mutation rates, an increase even in this small fraction may add up to a large number of new cases. However, it is only if members of an irradiated group or their descendants intermarry over several generations, and do not mate with the unirradiated population, that there is likely to be a disproportionately greater manifestation of hereditary defects among the descendants. Such an extreme degree of inbreeding is unlikely to occur. A fraction of the community can, therefore, without significant genetic risk to their progeny or harm to the population as a whole, receive doses of radiation which would be likely to have serious effects if applied to the whole population (see paragraphs 185–187).

259. From the genetic point of view, we have therefore to consider radiation dosage under three headings:

(i) The dose which the individual can accept without undue concern about its possible effects on his own progeny.

(ii) The dose which can be accepted by a fraction of the population whose occupation exposes them to more than the dose of radiation received by the ordinary member of the community.

(iii) The dose which can be accepted by the whole population.

Dose to the individual in relation to genetic effects

260. We have concluded that doses up to, and somewhat beyond, the 'doubling dose' need cause no undue concern to the individual as regards his own offspring. Further, we gave reasons for believing that the values for the doubling dose of radiation for human genes may be, in general, in the range of 30 r to 80 r. We consider, therefore, that an individual could

* Precise data for a large number of isotopes are given, for example, in the Recommendations of the International Commission on Radiological Protection (1955) (*Brit. J. Radiol.*, *N.S.*, Suppl. No. 6).

93299 O—57—pt. 2——39

reasonably accept a total dose to the gonads of not more than 50 r from conception to the age of 30 years, in addition to that received from the natural background. There will be no undue risk to the offspring of parents over this age provided the rate of exposure laid down in paragraph 255 is not exceeded.

Dose to occupationally exposed groups

261. Similar considerations apply to groups of the community whose occupation exposes them to more than the usual dose of radiation. Provided that such groups do not in aggregate total more than one-fiftieth of our population, we consider that all their members could each safely receive a total gonad dose of up to 50 r from conception to the age of 30 years, in addition to that received from the natural background.

Dose to the whole population

262. In the chapter on genetics we tried to give an indication of the load on the community that might be imposed by a doubling of mutation rates. It will be generally agreed that such a load should not voluntarily be accepted. In relation to genetic changes in the whole population, the significant figure is the total gonad dose of radiation which is received by all those capable of reproduction. A relatively high dose to a fraction of the population can only be offset by a correspondingly low dose to the remainder. If, therefore, we are to contemplate the possibility—and the necessity to develop the beneficent uses of atomic energy and ionizing radiations forces us to do so—that a significant fraction of our population may in future be allowed to receive doses of radiation of a similar order to the doubling dose, then it becomes additionally important to ensure that the dose of radiation to the rest of the community shall be held at the lowest possible level.

263. In view of the inadequacy of present knowledge, however, we do not feel justified in naming any specific figure as a limit for the average exposure of the whole population. It is nonetheless highly desirable that such a figure should be named as soon as possible and we understand that the International Commission on Radiological Protection has this matter under consideration. In the meantime, we feel bound to state our opinion that it is unlikely that any authoritative recommendation will name a figure for a permissible radiation dose to the whole population, additional to that received from natural sources, which is more than twice that of the general value for natural background radiation. The recommended figure may indeed be appreciably lower than this; and we consider that those on whom rests the responsibility for authorising the development and use of sources of ionizing radiation would be well advised to keep this possibility in mind.

The Hazards from Radiation

264. In the light of these general criteria, we may now proceed to examine the various hazards, actual and potential, to which the population in a country like our own may be exposed in consequence of the increasing use of ionizing radiation. In doing so, it will be necessary to distinguish between the hazards of peacetime experience and those which may be encountered in war.

PEACETIME HAZARDS

265. The development of modern civilisation has led to the increasing use of processes which produce radiations. Its future progress will come to depend, to an ever increasing extent, upon their further exploitation in power production, in industry, in medicine and in agriculture, as well as in basic

scientific research. It would be impossible to abolish their use without denying ourselves services upon which we have already come to rely ; and, in the future, nuclear energy will constitute a major, if not the most important, physical factor upon which our civilisation will depend. In assessing the hazards consequent upon its use, it is therefore necessary to maintain a sense of perspective and to weigh, as society has done in the case of steam-power, electricity and the internal combustion engine, the risks entailed against the advantages to be gained from the employment of this newer source of power. The risks are twofold: those to the individual and those to the population as a whole. In regard to the former, we may indicate our own assessment by saying that, in view of the small numbers likely to be employed in atomic power production and of experience already gained in the effectiveness of protective methods, it seems probable that a given amount of power might be made available to the community at a smaller cost in accidents, illness and disability than that involved in present methods of mining and power production. The novel aspect of the situation lies in the possible genetic risk to the community as a whole. The considerations which we have put forward suggest that, although this is at present small and seems unlikely ever to be large, it is potentially important. We propose now to indicate the relative hazards of particular uses and to point out where and how these may be expected to increase or should be curtailed.

266. In the chapter on exposure levels, the contributions of the various man-made sources of ionizing radiation to the total exposure were expressed as percentages of the radiation received from natural sources ; and it was shown that today the population of this country receives, from man-made sources, a dose of radiation equivalent to at least one-quarter of that from the natural background. In itself this figure, which amounts to less than 1 r over a period of 30 years, can give rise to no immediate apprehensions ; but its significance should not be disregarded. From the point of view of population genetics, all extra radiation is undesirable, and it is at least a portent that, in the half century following the discovery of ionizing radiation, man has increased his exposure levels by about 25 per cent. It does not therefore seem to be too early to suggest where the use of ionizing radiation might be restricted.

Medical diagnostic radiology

267. The greatest contribution in this country to the increased exposure to radiation comes from medical diagnostic radiology, the application of which has been steadily increasing in amount and scope in recent years. A large proportion of the genetically significant dose derived from diagnostic radiology is contributed by relatively few types of examinations, of which fluoroscopic and radiological examination of the female pelvis, and examinations of the hip joint and lumbar spine in males, are important examples. Clearly, the small genetic risk to the community and to individuals must be weighed against the possible great advantage and even necessity of the radiological examination to the particular patient. The final decision must be made on medical grounds. There can, however, be no doubt that the risk could in many instances be reduced, not only by a reduction of the actual number of examinations carried out on young people, but also by the use of modern methods of X-ray examination and by strict limitation of the X-ray beam to those parts of the body which have to be exposed. From the point of view of the dose distributed over the population as a whole, and from the point of view of the special risks to individuals associated with examinations involving heavy exposure, we are of the opinion that the time has arrived for a review of present practice in diagnostic radiology.

Radiotherapy

268. The dangers associated with the heavy exposures employed in radiotherapy are well recognised, and the illnesses for which it is used are usually so serious that the risks can be fully justified. In addition, many of the patients treated for cancer are of an age when the genetic risk will be small. Nevertheless, treatment by radiotherapy of certain non-malignant conditions, particularly in children, could with advantage be reconsidered from the point of view of reassessing the risks entailed against the benefits conferred.

269. The increasing use of therapeutic amounts of radioactive isotopes must also be carefully assessed and controlled, although at the present time the total used is probably of little practical significance from a genetical point of view. Should, however, their use become widespread in relation to common diseases of early or middle life, they might make a significant contribution. The risks from the use of 'tracer' amounts of radioactive substances in diagnosis and investigation are at present very small.

Occupational exposure: Atomic Energy Authority employees

270. For a number of years the main group of persons exposed to radiations were workers in the X-ray and radium departments of hospitals. Later, this experience was supplemented by careful observations of those employed in atomic energy projects and the various atomic industries. We have already seen that, as a result of all this experience, the maximum permissible level of radiation for workers was fixed at 0·3 r per week, and this figure is still regarded as a satisfactory working level provided that it is not maintained for years on end. There is, however, no large factor of safety in this figure and, as we have seen above, other limitations are needed for those exposed over very long periods of time. The excellent practice of the Atomic Energy Authority, now the main industrial employer of persons occupationally exposed to radiation, is shown by their record that the average exposure of their employees is less than half a roentgen yearly, and that their contribution to the population gonad dose is only about 0·1 per cent of that contributed by the natural background. These, however, are average doses; but, even if one takes the maximum exposure of those employees engaged in special tasks, this has only occasionally and for short periods approached the maximum permissible dose of 0·3 r per week. In view of the expected development of atomic energy as a source of power, this evidence of the awareness of the risk, and of the care with which it is being met, gives justifiable grounds for reassurance.

Radioactive effluents

271. Concern has been expressed about the possible emission from some nuclear reactors of radioactive particulate matter, which on inhalation might lodge in the lungs and give rise ultimately to cancer. This possible risk is well recognised and has been effectively dealt with.

272. Similar concern has been felt about the disposal of radioactive wastes. It is true that the amounts of radioactive material involved are formidable, but by treatment, by long-term storage to allow of decay, by concentration to relatively small bulk and subsequent burial in sealed containers or disposal in the depths of the ocean, there seems no reason to doubt that this problem can continue to be solved. In this country strict control of the hazard is exercised by legislation.

Occupational exposure: hospital employees

273. The handling of large quantities of radioactive isotopes in the treatment and investigation of patients in hospitals raises many difficult problems

but, with adequate care and training of the workers concerned, experience shows that the doses received can be kept down to one-tenth or less of the maximum permissible levels.

274. In this country a code of practice, formulated by the Ministry of Health on the advice of the Medical Research Council, has been drawn up for the National Health Service. This lays down the relevant permissible levels of exposure as well as giving instructions and suggestions for the implementation of these in practice.

Other industrial employees

275. The control of radiological hazards in non-atomic industry will be more difficult to codify owing to the wide variety of circumstances in which ionizing radiations are used. Measurements of the doses received by many workers are made periodically, but it is difficult to avoid the impression that industrial personnel are, in general, less aware of the hazards of radiation than those engaged in the fields of medicine and atomic energy. The record of the Atomic Energy Authority shows the standard that is attainable and the practicability of being satisfied with nothing less.

Definition and review of safety standards

276. All these problems are under close and continuous scrutiny. Expert national committees, notably those of the Medical Research Council and Ministry of Health, work in close conjunction with international bodies such as the International Commission on Radiological Protection (itself associated with the World Health Organization), so that a wealth of experience is brought to bear on all these problems, and the standards of safety are under constant review.

Miscellaneous sources of radiation

277. There are several other sources of radiation which at present contribute only very small amounts but which cannot be disregarded. The practice of routinely examining the feet by X-rays when fitting shoes is of dubious value and, in view of the possibilities of multiple exposures to children, may even be dangerous. On the basis of avoiding any unnecessary exposure, it is in our view hardly justifiable. We hope that the procedure will be abandoned, except when prescribed for orthopaedic reasons.

278. The contribution of radiation from watches and clocks with luminous dials is also small but real. The main hazard is to workers in the luminising industry but the risk from the widespread use of such instruments is not entirely negligible. We recognise that there are circumstances which require the use of instruments with self-luminous dials. For the majority, however, there is no such necessity and their wider use constitutes an avoidable, if small, risk which could be minimised for all concerned if the amount of radioactive material in these instruments was reduced to the lowest possible level.

279. It has been recognised that television sets give rise to very small amounts of X-rays, but at the present time radiation from this source does not constitute either a personal or a significant genetic hazard. This applies to domestic sets working at normal operational voltages but, near special types of high voltage projection equipment used commercially, the radiation may reach significant levels, although it is improbable that in practice any operator would be appreciably exposed. Nevertheless, the possibility of television equipment giving rise to radiations should be borne in mind when

considering the design and operation of such instruments. So far as sets used by the general public are concerned, most of the radiation is normally absorbed in the apparatus itself and is insignificant at the usual viewing distances.

Test explosions of nuclear weapons

280. It is impossible to explode a nuclear weapon without liberating radioactive matter into the atmosphere. As described in the chapter on exposure levels, the radioactive material diffuses all over the world and in the course of time is gradually deposited on the surface of the earth and comes into contact with human beings. Continuation for an indefinite time of testing at the same rate as over the last few years would gradually increase the contamination of the atmosphere, until in about 100 years time the average individual in this country would receive a dose of external radiation to the gonads of 0·026 r in 30 years of his life, an amount which represents only one per cent of that received from the natural background. The individual and genetic effects of such a dose of external radiation would be insignificant.

281. Account must be taken, however, of the particular hazard from radioactive strontium in the fall-out. The maximum permissible level of strontium 90 in the human skeleton, accepted by the International Commission on Radiological Protection, corresponds to 1000 micro-microcuries per gramme of calcium. But this is the maximum permissible level for adults in special occupations and is not suitable for application to the population as a whole or to children with their greater sensitivity to radiations and greater expectation of life. It is known that radiostrontium is more heavily concentrated in the bones of young than of adult animals, and the few measurements on human bones indicate that at the present time those of children contain about ten times the concentration found in those of adults. We consider, therefore, that the maximum allowable concentration of radiostrontium in the bones of the general population, with its proportion of young children, should not be greater than 100 micro-microcuries of strontium 90 per gramme of calcium.

282. In this country, measurements on human bones of the radiostrontium content, derived from the nuclear explosions that have already occurred, show that the irradiation from this source is now reaching about one-thousandth of the maximum permissible occupational level ; and calculation of the fall-out likely to come, if the present rate of firing continues, suggests that this level may be increased ten-fold in the course of several decades. The present level would produce no detectable increase in the incidence of ill-effects. It is evident, however, that we are now accumulating radiostrontium at an appreciable rate and that a close watch will need to be kept on this increase.

283. In the light of knowledge at present available, we should feel that immediate consideration were required if the concentration in human bones showed signs of rising greatly beyond one-hundredth of that corresponding to the maximum permissible occupational level.

284. We are well aware of the inadequacy of our knowledge of the biological effects of radioactive strontium and of the urgent necessity to obtain further information. Nevertheless, recognising all the inadequacy of our present knowledge, we cannot ignore the possibility that, if the rate of firing increases and particularly if greater numbers of thermonuclear weapons are used, we could, within the life-time of some now living, be approaching levels at which ill-effects might be produced in a small number of the population.

285. The general conclusion to be drawn from a consideration of the hazards inseparable from the application of ionizing radiations in peacetime is that at present there is no cause for alarm ; but that, as all such radiations are potentially dangerous, their use should be the subject of constant and close scrutiny, and that adequate justification should be required for their employment on however small a scale. There is a limit to the amount of radiation which any population or any individual can accept and we cannot afford to expend, without careful forethought, the margin which is now available to us.

WARTIME HAZARDS

286. We have given a brief résumé of the effects of nuclear warfare. From this it will be seen that there are three broad categories of effect: those within the range of the actual explosion, those within the contiguous area in which radioactive fission products settle, and world-wide effects due to the contamination of the atmosphere.

287. Within close range of the explosion, nuclear radiations are but one element in the destructive effect. Blast and heat would be of major and probably of more immediate importance in producing casualties but survivors, unless heavily sheltered, would have been exposed to such an intensity of radiation that they would be at risk of developing each and all of the effects that we have described.

288. Explosions of atomic weapons always give rise to radioactive fission products, the heavier particles of which settle in the vicinity. With a ground burst of a thermonuclear weapon, the area of intense fall-out may cover hundreds of square miles. Within this area, those who were not in shelter, and did not remain under cover until the radioactivity of the fall-out had decayed substantially, would be exposed to intensities of radiation sufficient to produce the effects described in all grades of severity. Outside this area, there would be another zone, measured in thousands of square miles, where significant intensities of radiation would occur and where a proportion of those exposed would be at risk of serious consequences.

289. It must be emphasised that these doses, from the point of view both of the individual and of the general population, are several thousand times greater than those we have considered as possible peacetime hazards.

290. The importance of the effects of atomic warfare which would be relayed through contamination of the atmosphere to parts of the world remote from the actual conflict, would depend upon the number and type of bombs exploded. Given a sufficient number of bombs, no part of the world would escape exposure to biologically significant levels of radiation. To a greater or less degree, a legacy of genetic damage would be incurred, and an increased incidence of delayed effects on the individual would probably be induced. Although it is difficult to imagine the general occurrence of radiation intensities which would eliminate the entire human race, atomic warfare on a large scale could not fail to increase for many generations the load of distress and suffering that individuals and all human societies would be called upon to support.

CHAPTER VII

SUMMARY

291. The future development of civilisation is bound up with the exploitation of nuclear energy. Its use, like that of other sources of energy, entails risk, but the risk is controllable and, within limits, can be accepted. It is the scale and not the nature of the hazard that is new, for human populations have always been exposed to natural radiation of low intensity. (Paragraphs 1–14.)

THE NATURE OF RADIATION AND ITS ACTION ON LIVING CELLS

292. Ionizing radiations are so described because they cause the formation of electrically charged particles, ions, in the matter through which they pass. The common types of penetrating radiation are X-rays, gamma rays, alpha and beta particles, and neutrons. Alpha particles cannot penetrate tissue beyond a fraction of a millimetre but gamma rays, and X-rays produced by extremely high voltages, can traverse the whole body. (Paragraphs 15–20.)

293. The biological effects of radiation are related to the intensity of radiation and to the period of exposure. The basic unit of radiation dosage which has been generally used is the roentgen (r). All living tissue can be killed if exposed to sufficiently high doses of radiation. The effects of dosages below those which damage tissues irretrievably may be modified by processes of healing, so that the response to a dose of radiation which is spread over a long time may be much smaller than, or quite different from, the response which would occur if the same dose were given in a very short time. This does not apply to the important type of genetic effect, called gene mutation, produced by the irradiation of reproductive cells, the consequences of which are cumulative and irreversible. (Paragraphs 21–27.)

THE EFFECTS OF RADIATION ON THE HEALTH OF THE INDIVIDUAL

Sources of information

294. Our knowledge of the effects of ionizing radiations on human beings comes from four main sources: from the uses of X-rays and radium in the treatment of disease, mainly of cancer; from a study of the occupational hazards of medical radiologists, workers in the luminising industry, and miners of radioactive ores; from a study of the victims of atom bomb explosions; and from experiments on animals. (Paragraphs 28–34.)

The harmful effects of radiation on man

295. Almost all the effects of ionizing radiation on tissues are essentially deleterious. The benefits to the individual patient of the eradication of a malignant tumour by radiotherapy result from selective damage to the tumour cells. The nature and severity of radiation injury is determined by the type and dosage of radiation received, the part and extent of the body irradiated, the length of the period of exposure, and the age of the persons exposed. The harmful effects may be classified into those which develop within a few weeks of exposure, and delayed effects which may not make their appearance until many years after exposure. (Paragraphs 35–42.)

Effects occurring within a few weeks of exposure

296. The effect of exposing the whole body to a single dose of gamma radiation of the order of 500 r is such that all the persons so exposed would develop acute illness and at least half would die. In civil life, exposure to such a dosage could occur only under the most exceptional circumstances. With smaller single doses, for example of 100 r, not more than 15 per cent of an exposed population would suffer acute illness and very few, if any, of those affected would die. After a single dose of 50 r, acute illness would be very rare. The relationship between the dose of radiation received and the effects that may be produced within a few weeks of exposure is not one of strict proportionality ; with each successive and equal increment of dosage the response increases by a progressively greater amount, at least until very large changes have been produced. (Paragraphs 43–50.)

The delayed effects of radiation

297. Delayed effects of exposure to radiation may occur at any time after the end of the second month. Disorders of the skin and underlying soft tissues and of bone may occur and there may be subsequent development of cancer. Cataracts, severe anaemias and leukaemia have been caused and there is evidence from animal experiments that exposure to radiation may cause death at a prematurely early age. (Paragraphs 51–52.)

Leukaemia

298. Leukaemia is a disease in which there is an uncontrolled over-production of white blood corpuscles. Experiments on animals have shown that the incidence of leukaemia is increased by irradiation. Clear evidence that the same is true of man comes from two main sources: a study by the Atomic Bomb Casualty Commission of the incidence of leukaemia in Hiroshima and Nagasaki, and a survey under our sponsorship of the incidence of leukaemia among patients treated by radiation for ankylosing spondylitis. (Paragraphs 53–55.)

299. Ninety-one proven and fourteen suspected cases of leukaemia have been recorded in Hiroshima and Nagasaki between 1947 and 1954 among those present at the time of the explosion and still resident in the cities ; the expected incidence in an unexposed but otherwise comparable population is twenty-five. The difference is greater than would be attributed to chance. Moreover, there was a much higher frequency of occurrence among those who had developed early acute radiation illness and among those who had been nearer to the centre of the explosion. The latent period, that is the average length of the period between the explosion and the first appearance of symptoms of leukaemia, was about six years. The evidence suggests that with this type of exposure to radiation the likelihood of developing leukaemia, after its initial rise, remains approximately constant up to at least the ninth year. (Paragraphs 56–61.)

300. Ankylosing spondylitis is a disease in which the joints, particularly those of the spine, progressively lose their freedom of movement. In the treatment of this condition very extensive areas of the body are exposed to irradiation. The records of between 13,000 and 14,000 patients, who had been treated with X-rays between 1933 and 1954, have been studied. Up to 1955, thirty-eight of these patients developed leukaemia, an incidence which, although only about one-third of one per cent, is about ten times greater than the normal expectation. No increased incidence of leukaemia was found among 400 patients who had not been treated by irradiation, but the number is too small to exclude completely the possibility that ankylosing spondylitis

may of itself predispose its sufferers to leukaemia ; nor can the possibility be excluded that these patients are more liable than the average person to develop leukaemia after irradiation. Nevertheless, there is clear evidence of a correspondence between the dosage of radiation received and the incidence of leukaemia. The average length of the latent period between the first exposure to X-rays and the diagnosis of leukaemia was about six years. (Paragraphs 62–69.)

301. The conditions of exposure to radiation in Hiroshima and Nagasaki, and in the treatment of ankylosing spondylitis, are not comparable with the irradiation in small doses over long periods which might be received by persons engaged in work with a possible radiation hazard. Some evidence has been presented suggesting an increased death rate due to leukaemia among radiologists but our knowledge of the occurrence of leukaemia under conditions of chronic exposure is too scanty to allow any reliable conclusions to be drawn. (Paragraphs 70–71.)

Cancers

302. Two characteristics of cancers induced by radiation are noteworthy : the tendency of tumours to arise in tissues already severely damaged by radiation, and the long latent period, twenty years or more, before they appear. (Paragraph 72.)

303. A study of the pitchblende miners of Schneeberg and Joachimsthal suggests strongly that inhalation of the radioactive gas radon may lead to cancer of the lung. The latent period has been put at seventeen years and the dosage to the lungs over that period at about 1000 r and in some parts of the lung much higher. In theory, the inhalation of radioactive particles in the fall-out from atomic explosions or in the vicinity of nuclear reactors could also lead to cancer of the lung, but the former hazard is extremely unlikely in peacetime, and steps are always taken to ensure that the latter does not occur. (Paragraphs 73–76.)

304. Radium, mesothorium, plutonium and radioactive forms of strontium are accumulated by and retained in bone. Until the enforcement of stringent controls, cancer of bone occurred among workers in the luminising industry as a result of swallowing radium-containing paint. The latent period was more than fifteen years. (Paragraphs 77–82.)

305. Cancer of the skin was the earliest form of radiation-induced tumour to be described in man. By 1911, before the adoption of modern safeguards, fifty-four cases had been described among the pioneers of radiology. The doses of radiation which have led to the formation of skin cancers must have been several thousand r. (Paragraphs 83–84.)

306. Cancer of the thyroid gland in children has been a sequel to irradiation of the neck for enlargement of the thymus gland. This form of cancer is distinguished by its short latent period (about 7 years) and the comparatively low dosage of radiation required to induce it. However, it is not unlikely that other factors are involved here in addition to the direct effect of irradiation. (Paragraphs 85–86.)

Other delayed effects

307. A fall in the number of red cells and white cells in the blood may follow exposure of the whole body to even moderate doses of gamma radiation. If not detected in time a condition known as aplastic anaemia may occur. (Paragraphs 87–88.)

308. Cataract formation is known to have been caused by neutron irradiation, but for all practical purposes the production of cataract by X-rays is not an occupational hazard. (Paragraphs 89-90.)

309. Delayed effects of radiation on the skin extend from a temporary loss of hair after local dosages of 300r–400r to severe and permanent damage after local exposure to single dosages of 1500r or more, or to repeated doses totalling 4000r or more in a number of weeks. It is in the skin damaged by these higher doses of radiation that tumours, when they occur, are most likely to develop. (Paragraphs 91–92.)

310. Miscarriage and stillbirth may be a consequence of irradiation during pregnancy, but they do not constitute a problem unless the dose of radiation is large. A number of different developmental abnormalities have been described in the children of women treated by irradiation during pregnancy, the most conspicuous defect being microcephaly, a partial failure of the development of the brain. Eleven cases so classified are recorded in children irradiated before birth in Hiroshima and Nagasaki. (Paragraphs 95–97.)

THE GENETIC EFFECTS OF RADIATION

311. The assessment of the genetic effects of ionizing radiations is subject to special difficulties. We believe that we have formed as fair an assessment as is possible in the light of present knowledge, but our conclusions must be regarded as provisional. (Paragraph 100.)

The material basis of heredity

312. The physical determinants of heredity are genes, carried on chromosomes in the nuclei of cells. Chromosomes are present in pairs ; one member of the pair is of maternal origin, the other of paternal origin. There are twenty-four pairs of chromosomes in human beings ; the number of genes is not known, but may well be many thousands. (Paragraphs 101–103.)

313. The two genes which occupy corresponding positions on the two chromosomes of a pair are spoken of as alleles of each other. Alleles of different kinds arise by the process of mutation and are thereafter reproduced faithfully in their altered form. (Paragraph 104.)

314. Some genes produce the same effect whether they are paired with like or with unlike alleles. Such genes, and the characters they determine, are described as dominant. Other genes produce a noticeable effect only when paired with similar alleles ; these, and the characters they determine, are described as recessive. There is every gradation between these two extremes. A recessive gene can be transmitted in a family by an individual who gives no signs of carrying it. (Paragraphs 105–108.)

315. Sex difference is determined by a special pair of chromosomes, and the genes carried on these chromosomes are said to be sex-linked. (Paragraph 109.)

316. So far as is known, all genes are subject to mutation, and mutation occurs spontaneously all the time at a very low rate. Factors influencing mutation appear to affect only the frequency with which it happens. New alleles of harmful effect are eliminated by natural selection until equilibrium is reached with the rate at which they are introduced by fresh mutation. Recessive alleles are eliminated much more slowly than dominant alleles. (Paragraphs 111–118.)

Basic principles of the genetic effects of radiation

317. There is little direct knowledge of the genetic effects of ionizing radiations on man, but with certain reservations it is justifiable to draw upon our knowledge of the effects of radiation on other organisms. (Paragraph 119.)

318. Ionizing radiations have genetic consequences only in so far as they affect the reproductive cells or the cells ancestral to them in the reproductive organs (gonads). Two kinds of effect may have genetic consequences: the chromosomes may be damaged or the genes may be caused to mutate more frequently. Chromosome changes of the kind that can persist are only rarely produced by long continued exposure to X-rays or gamma rays of low intensity. They are likely to be a comparatively unimportant radiation hazard. (Paragraphs 120–125.)

319. It is the frequency of gene mutation that is increased by radiation; there is no evidence and little likelihood that radiation produces entirely new kinds of genes. The rise in mutation rate is probably directly proportional to the amount of additional exposure to radiation, and any additional exposure, however small, must be expected to raise the mutation rate, if only by a minute amount. (Paragraphs 126–127.)

320. Damage to genetic material is cumulative and irreparable. Long continued exposure to radiation of low intensity induces as much gene mutation as a single exposure to an equal dosage of radiation of higher intensity. (Paragraph 128.)

321. The age-distribution of those exposed to radiation has an important bearing on the future consequences of its effects. The genetic consequences of the irradiation of individuals beyond the age of reproduction are of course nil. (Paragraph 129.)

Effects of increased mutation on the incidence of disease in human populations

322. The role of heredity in the production of disease ranges from that of a predisposing to that of a preponderating cause. The effects which might be expected to result from an increase in mutation rates can most easily be calculated for diseases known to be caused by single genes, but for relatively few such diseases have we sufficient evidence of the kind upon which such a calculation must be based. (Paragraphs 130–135.)

323. Achondroplasia, haemophilia, and phenylketonuria have been taken as examples of diseases believed to be caused by single genes. If the mutation rates of these genes were to rise to, and remain at, twice their present values, the incidence of the diseases for which they are responsible would ultimately, though at very different rates, rise to nearly twice their present frequencies. Calculations suggest that the incidence of achondroplasia, a dominant form of dwarfism, would rise 80 per cent above its present value in a single generation; haemophilia, a sex-linked disease, would take about six generations to rise by 90 per cent in frequency; and phenylketonuria, a recessive disease associated with severe mental deficiency, would take more than fifty generations to increase its frequency by one half. (Paragraphs 136–139.)

324. Mental diseases, the most important single category in which hereditary causes are known to be important, account in all for nearly half the hospital beds provided in this country. There are grounds for believing that a doubling of the mutation rates of the genes concerned with their causation would, in one generation, increase the frequency of low-grade mental deficiency by three per cent, and of the two principal types of mental illness,

schizophrenia and manic depressive reaction, by about one per cent. If the mutation rates were to remain at twice their present values, the incidence of mental diseases might on the most pessimistic assumptions double also, but would only attain this value after very many generations.

(Paragraphs 140-150.)

325. When all serious illnesses with a hereditary element in their causation are taken into account, it is unlikely that the burden put upon society by a doubling of mutation rates would exceed by more than a few times the contribution made by the increase of mental disease.

(Paragraphs 151–154.)

326. It must be remembered that a harmful recessive gene gives no outward evidence of its presence until chance brings it together with another of its kind. The crop of newly mutated recessive genes caused by an increase of mutation rates could cause suffering over many generations.

(Paragraphs 155–156.)

Hereditary traits showing continuous variation about the normal

327. Most of the variation between human beings is not of the sharp kind that can be traced to the action of single genes. Characters such as physique, intelligence and length of life vary over a wide range by imperceptible gradations, and the hereditary portion of this variation is believed to be due to the combined action of many genes. (Paragraphs 157–159.)

328. The basic effect of an increase in mutation rates upon such characters, here exemplified by scores in intelligence tests, will be to increase the numbers of the more extreme types at the expense of the more average individuals. A doubling of the mutation rates for a few generations would be expected to have only the most trivial effect upon their variation. The effect of a permanent doubling of the mutation rate would be, at most, to double the variation, and this would take hundreds of generations to achieve.

(Paragraphs 160–161.)

Observations on populations exposed to radiation

329. Three direct studies have been made on the children of human beings who have been exposed to ionizing radiations. Two, on the children of American radiologists, were for a variety of reasons inconclusive ; the third is the extensive study made by the Atomic Bomb Casualty Commission on the children of those who were in Hiroshima and Nagasaki when the atomic bombs exploded. All three studies are limited to observations on the first generation, so that little genetic effect would yet have become manifest even if the mutation rate had increased. (Paragraphs 162–166)

330. The evidence assembled in the report of the Atomic Bomb Casualty Commission is beset by many difficulties of interpretation, but we believe that it reveals, in the children of those who were the more heavily exposed, a slight but significant change in the sex ratio at birth which might be due to genetic damage. From the nature of the evidence a doubling of the rate of incidence of congenital malformations, or a 50 per cent rise in the stillbirth rates, might have escaped detection if either had occurred. The evidence does not allow us to make any useful estimate of the radiation dose which doubles the mutation rate in man. (Paragraphs 167–170.)

The 'doubling dose' in man

331. An assessment of the sensitivity of human genes to radiation is particularly difficult. Any such estimate should be based upon a sample of genes large enough to be representative of all the effects they exercise, for it cannot be assumed that all genes are equally radiosensitive, nor that the

proportion of the spontaneous mutation rate which can be attributed to natural radiations is the same for different genes. (Paragraphs 171–175.)

332. If all mutations were indeed due to radiation, then the dosage which doubled their frequency would be expected to be equal to that received from natural sources, namely, a dosage to the gonads of about 3 r in thirty years. The available evidence suggests, however, that the percentage of human mutations that are caused by natural radiation might lie between 2 per cent and 20 per cent, and if this is so the doubling dose will lie between 15 r and 150 r. (Paragraphs 176–181.)

333. The direct estimates which have been made of the doubling doses for a variety of plants and animals mostly run from 25 r upwards. It is true that none of the more fully investigated organisms has a lifetime comparable with man's, but there are theoretical grounds for believing that the organisms with the longer pre-reproductive periods might be expected to have the less radiosensitive genes. (Paragraph 182.)

334. The evidence at our disposal, though far from adequate, leads us to conclude that there is rather little likelihood that the real value for the doubling dose for human genes lies between 3 r and 15 r; and that, although we cannot exclude the possibility that for some human genes the doubling dose may be less than 30 r and for others more than 80 r, the best estimate that we can make in the light of present knowledge, is that the value in general lies somewhere between 30 r and 80 r. (Paragraph 183.)

335. Even if the doubling dose were as low as the minimum we can reasonably entertain, namely 15 r, it is extremely improbable that in times of peace more than a small fraction of the population could receive an extra dose of this size. The prevalence of naturally-occurring hereditary abnormalities is such that, if comparatively few individuals received such a dose, there would be no noticeable effect on their immediate offspring or on their descendants even over several centuries. For levels of radiation up to the doubling dose, and even some way beyond, the genetic effects of radiation are only appreciable when reckoned over the population as a whole, and need not cause alarm to the individual on his own account. (Paragraphs 184–188.)

EXISTING AND FORESEEABLE LEVELS OF EXPOSURE TO RADIATION

336. Doses of radiation which are of no known significance to the individual may have genetic consequences. Exposure levels must therefore be expressed in terms of the total dosage to the gonads received by the population as a whole during the period of reproductive life. (Paragraphs 189–191.)

Radiation from natural sources

337. The natural sources of radiation are cosmic rays and the naturally-occurring radioactive elements. From all such sources an individual in this country receives, on the average, a total gonad dose of about 3 r over a period of thirty years. (Paragraphs 192–201.)

Radiation from the appurtenances of civilisation

338. Over the past sixty years man has made increasing use of X-rays and radioactive materials in medicine, industry, and ordinary civil life. The additional gonad doses received from these sources by people of this country are expressed as percentages of the gonad dose which they already receive from natural sources. (Paragraph 202.)

339. We have conducted a limited survey which suggests that the additional dose received from the various forms of diagnostic radiology may well

be higher than 22 per cent, the major amount of which is accounted for by examination of a relatively few sites of the body. The contribution made by the use of radiation in medical treatment cannot be accurately estimated ; it is probably much less than that made by diagnostic radiology but greater than that received from any other artificial source.

(Paragraphs 203–206.)

340. Watches and clocks with radioactively luminous dials contribute about one per cent of additional radiation. X-rays from television sets account for much less than one per cent. The contribution from X-ray apparatus used in shoe-fitting is not likely to exceed 0·1 per cent. (Paragraphs 207–210.)

341. The contribution arising from the work of the Atomic Energy Authority is the most accurately known, and is about 0·1 per cent. A study of the records of the National Radiological Protection Service has put the contribution from other occupational sources at about 1·6 per cent.

(Paragraphs 212–217.)

Contamination of the world by fall-out from the explosion of nuclear weapons

342. Continual watch is kept by the Atomic Energy Authority on the radioactive fall-out reaching this country from nuclear devices exploded in other parts of the world. From the bombs exploded up to the present time, the population of this country may expect to receive, over the next fifty years, additional radiation amounting to between 0·02 per cent and 0·04 per cent of the radiation which will be received over the same period from natural sources. (Paragraphs 218–229.)

343. If the firing of bombs were to continue indefinitely at the same rate as over the past few years, radioactivity would gradually accumulate to a level at which an inhabitant of this country would receive an average dose of 0·026 r over a period of thirty years, or about one per cent of that which he would receive in the same period from natural sources.

(Paragraph 230.)

344. The contribution to this figure from thermonuclear explosions, relative to their numbers, is very great. If the rate of firing of weapons of this type increases, exposure to radiation will be significantly raised. (Paragraph 231.)

Special hazards of radioactive fission products

345. It is unlikely that the inhalation of radioactive particles present in the air as a result of fall-out would constitute a problem in ordinary civil life. (Paragraphs 232.)

346. The deposition of radioactive strontium is probably a greater hazard, because it is soluble and, if ingested, is deposited and retained in bone. Measurements which have been made of radioactive strontium in bone show that the highest levels are at present about a thousand times less than is considered permissible for those who are occupationally exposed.

(Paragraphs 234–239.)

Atomic war

347. Atomic bombs were developed for their capacity to create blast, but for persons exposed in the open the heat flash is equally to be feared. The ionizing radiations produced immediately after explosions have a much greater penetrating power than the heat rays, but the range at which they cause death or immediate injury is somewhat less. The hazard from radiations is therefore only one of the immediate effects of atomic explosions. Their peculiar danger lies in their distant and delayed effects.

(Paragraphs 241–246.)

ASSESSMENT OF THE HAZARDS OF EXPOSURE TO RADIATION

348. An attempt is made to assess the medical and genetic consequences of exposure to radiation at the levels of dosage which occur now or which might conceivably come about. The naturally occurring level of radiation can be accepted as a standard of reference, because it is a level to which mankind has long been adjusted. (Paragraphs 247–248.)

349. In considering the genetic effects of radiation, we are concerned with the sum, over the whole population, of the total gonad dose received by its members from conception until the end of reproductive life. (Paragraphs 249–250.)

350. In considering the effects of radiation upon the individual, we are concerned with his whole span of life, and with the rate at which the radiation is received as well as with its total dosage; and we must have regard to the possibility that the severity of the effects produced by radiation may increase in more than equal proportion to the dosage that is received. (Paragraph 251.)

Dosage and effects on the individual

351. The acute effects of radiation which appear within two months of exposure to a single dose or a few heavy doses do not enter into ordinary civil calculations; nor is it feared that they may be produced by repeated exposures to doses that do not exceed 0·3 r per week. (Paragraphs 252–253.)

352. Of the delayed effects of irradiation of the whole body, leukaemia is probably the most easily induced. We consider that an individual could, without feeling undue concern about developing any of the delayed effects, accept a total dose of 200 r in his life-time, additional to that received from the natural background, provided that this dose is distributed over tens of years and that the maximum weekly exposure, averaged over any period of 13 consecutive weeks, does not exceed 0·3 r. We recommend, however, that the aim should always be to keep the level of exposure as low as possible. (Paragraphs 254–255.)

Dosage and genetic effects

353. The genetic effects of radiation are essentially problems concerning the future welfare of the population as a whole. (Paragraph 257.)

354. It follows from the nature of the genetic effects of radiation that a small fraction of a population can, without harm to its members, receive dosages of radiation which would be likely to have serious genetic effects if applied to the population as a whole. We feel that an individual, considered as such, can accept a total gonad dose of not more than 50 r, from conception until the age of thirty, additional to that received from the natural background, without undue concern for himself or his offspring, but that the number of such individuals should not exceed one-fiftieth of the population as a whole. (Paragraphs 258–262.)

355. Our present knowledge does not justify us in naming any specific figure as a limit for the average dose of radiation which might be received by the population as a whole. It is highly desirable that such a figure should be named as soon as possible; and we understand that the International Commission on Radiological Protection has this matter under consideration. In the meantime, we feel bound to state our opinion that it is unlikely that any authoritative recommendation will name a figure for permissible radiation dose to the whole population, additional to that received from the natural background, which is more than twice that of the general value for natural background radiation. The recommended value may, indeed, be appreciably lower than this. (Paragraph 263.)

The peacetime hazards from nuclear radiation

356. Nuclear energy may become the principal source of power. So far as its use affects the small numbers likely to be employed in its production, we believe that nuclear energy might make power available at a lower cost in accidents, illnesses and disability than that incurred in connexion with other sources of power. What is novel in the use of nuclear energy and the other, increasing, uses of processes producing radiations is the genetic risk to the community as a whole. The risk from civil usage is at present small, and seems unlikely ever to be large ; but from the point of view of population genetics all possible extra radiation should be avoided, and it is not now too early to suggest where we might restrain its use. (Paragraphs 265–266.)

357. With regard to occupational exposure we consider that the record of the Atomic Energy Authority shows the standard that is attainable and the practicability of being satisfied with nothing less. (Paragraphs 270–276.)

358. We consider that the time has come for a review of present practice in diagnostic radiology, and of certain uses of radiation in the treatment of non-malignant conditions, particularly in children. Among the less important sources of radiation, we hope that the use of X-rays in shoe-fitting will be abandoned except when prescribed for orthopaedic reasons ; that watches and clocks with radioactively luminous dials will be confined to necessary uses ; and that the X-ray hazard from television tubes, at present negligible, will be borne in mind if special types of high voltage equipment come to be widely used. (Paragraph 267–269 and 277–279.)

Test explosions of nuclear weapons

359. The genetic effects to be expected from present or future radioactive fall-out from bombs fired at the present rate and in the present proportion of the different kinds are insignificant. They might not be so, if present rates of firing were increased and particularly if a greater number of thermonuclear weapons were tested. (Paragraph 280.)

360. So far as radioactive fall-out may affect the individual, we believe that immediate consideration would be required if the concentration of radioactive strontium in bone showed signs of rising greatly beyond that corresponding to one-hundredth of the maximum permissible occupational level. (Paragraphs 281–284.)

Wartime hazards

361. The area in which a greater or lesser proportion of those exposed would be at serious risk from the radioactivity released by the ground burst of a thermonuclear weapon is measured in thousands of square miles. If a sufficient number of nuclear weapons were exploded, no part of the world would escape biologically significant degrees of exposure or the load of distress and suffering to individuals and society which such exposure would entail. (Paragraphs 286–290.)

93299 O—57—pt. 2——40

CHAPTER VIII

CONCLUSIONS

362. On the basis of the considerations in this report we feel justified in drawing the following conclusions in relation to the use of ionizing radiations in peacetime :

1. *Limitation of the use of all sources of radiation*

Adequate justification should be required for the employment of any source of ionizing radiation on however small a scale.

2. *Dose levels to the individual*

(*a*) In conditions involving persistent exposure to ionizing radiations, the present standard, recommended by the International Commission on Radiological Protection, that the dose received shall not exceed 0·3 r weekly, averaged over any period of 13 consecutive weeks, should, for the present, continue to be accepted.

(*b*) During his whole lifetime, an individual should not be allowed to accumulate more than 200 r of "*whole-body*" radiation, in addition to that received from the natural background, and this allowance should be spread over tens of years ; but every endeavour should be made to keep the level of exposure as low as possible.

(*c*) An individual should not be allowed to accumulate more than 50 r of radiation *to the gonads,* in addition to that received from the natural background, from conception to the age of 30 years ; and this allowance should not apply to more than one-fiftieth of the total population of this country.

3. *Dose level to the population*

Those responsible for authorising the development and use of sources of ionizing radiation should be advised that the upper limit, which future knowledge may set to the total dose of extra radiation which may be received by the population as a whole, is not likely to be more than twice the dose which is already received from the natural background ; the recommended figure may indeed be appreciably lower than this.

4. *Fall-out from test explosions of nuclear weapons*

(*a*) The present and foreseeable hazards from *external* radiation due to fall-out from the test explosions of nuclear weapons, fired at the present rate and in the present proportion of the different kinds, are negligible.

(*b*) Account must be taken, however, of the *internal* radiation from the radioactive strontium which is beginning to accumulate in bone. At its present level, no detectable increase in the incidence of ill-effects is to be expected. Nevertheless, recognising all the inadequacy of our present knowledge, we cannot ignore the possibility that, if the rate of firing increases and particularly if greater numbers of thermonuclear weapons are used, we could within the life-time of some now living, be approaching levels at which ill-effects might be produced in a small number of the population.

5. *Recommendations regarding specific uses of radiation*

(*a*) All sources of radiation, both medical and industrial, should be under close inspection, in order to ensure that the high standards of protection now

attainable against the absorption of ionizing radiations, and against radioactive materials, are generally observed. Those using radiations should be instructed in the precautions to be taken, and no unnecessary or unauthorised person should be allowed to engage in such occupations. A personal record, not only of doses of radiation received during occupation but also of exposures from all other sources, such as medical diagnostic radiology, should be kept for all persons whose occupation exposes them to additional sources of radiation.

(*b*) Present practice in medical diagnostic radiology should be reviewed, with the object of clarifying the indications for the different special types of examination now being carried out and defining more closely, both in relation to the patient and to the operators, the conditions which should be observed in their performance.

(*c*) The uses of radiotherapy in non-malignant conditions should be critically examined.

(*d*) The small amounts of irradiation from miscellaneous sources, such as X-ray machines used for shoe-fitting, luminous watches and clocks, and television apparatus, should be reduced as far as possible.

6. *Collection of vital statistics*

As an essential basis for future studies of the genetic effects of radiation, further data are required on the genetic structure of human populations; to this end, there is an urgent need for the collection of more detailed information, when births, marriages and deaths are registered.

H. P. Himsworth
E. Rock Carling
J. D. Cockcroft
A. Haddow
A. Bradford Hill
J. F. Loutit
K. Mather
W. V. Mayneord
P. B. Medawar
J. S. Mitchell
L. S. Penrose
E. J. Salisbury
F. G. Spear
J. R. Squire
C. H. Waddington
Lionel Whitby
B. W. Windeyer

ACKNOWLEDGEMENTS

We have expressed in our introductory chapter our sense of indebtedness to those who have been intimately concerned with the preparation of this report. We wish also to thank those others who have undertaken the preparation of special papers for the information of the committee, namely: Dr. J. D. Abbatt, Dr. W. Anderson, Dr. P. Armitage, Dr. Charlotte Auerbach, Mr. W. Binks, Mr. D. V. Booker, Dr. F. J. Bryant, Dr. R. A. M. Case, Mr. R. C. Chadwick, Mr. A. C. Chamberlain, Mr. R. N. Crooks, Dr. R. Doll, Miss E. M. R. Fisher, Dr. A. Glücksmann, Dr. A. W. G. Goolden, Professor J. B. S. Haldane, Dr. G. E. Harrison, Dr. W. G. Marley, Dr. R. H. Mole, Mr. A. Morgan, Mr. S. B. Osborn, Dr. Alan Robertson, Mr. E. E. Smith, Professor D. W. Smithers, Professor F. W. Spiers, Mr. R. W. Stanford, Mr. N. G. Stewart and Dr. R. C. Turner.

We have had the invaluable assistance of the following who have prepared appendices to the report: Dr. W. G. Marley, Mr. S. B. Osborn, Mr. E. E. Smith and Professor F. W. Spiers. We wish also to thank Miss Annette Todd, of the Council's Headquarters Staff, for her work in editing the appendices.

We feel a special debt of gratitude to the United States Atomic Bomb Casualty Commission and to Professor J. V. Neel and Dr. W. J. Schull, who have so kindly made available to us the data on the effects of exposure to the atomic bombs at Hiroshima and Nagasaki.

We wish to acknowledge the great help given to us by the staffs of a number of research departments in different hospitals and elsewhere, especially by Dr. W. G. Marley and the staff of the Health Physics Division of the Atomic Energy Research Establishment, Harwell, by members of the Department of Physics of the Institute of Cancer Research, Royal Cancer Hospital, London, of the Galton Laboratory, University College, London, of the Medical Research Council's Statistical Research Unit, London School of Hygiene and Tropical Medicine, London, and of the National Institute for Medical Research, London.

In connexion with certain statistical aspects of our work, our thanks are due to the Registrar General for England and Wales and to the Department of Health for Scotland.

We are grateful to those who assisted in the special investigation on the incidence of leukaemia among patients treated with X-rays for ankylosing spondylitis, especially to the following: Dr. J. D. Abbatt, Dr. F. Ellis, Dr. Joan Faulkner, Mr. D. Hewitt, Dr. A. J. Lea, Dr. J. H. Mulvey, Dr. W. D. Rider, Dr. A. Stewart, Dr. J. Stubbe and Dr. J. W. Webb, who were members of the teams which visited the radiotherapy centres; to the staffs of the 70 centres in England, 3 in Wales and 9 in Scotland, who freely gave their help in the compilation of the case records for this study; to the Registrars General for England and Wales, Scotland and Northern Ireland, the Deputy Registrar General for the Isle of Man, the Superintendent Registrar, Jersey, Channel Islands, and Her Majesty's Greffier for Guernsey and Dependencies, who provided valuable information; to the Ministry of Pensions and National Insurance, who made their records available and gave much help, and to Dr. J. D. Abbatt and Dr. A. J. Lea who made a special study of these records; to Dr. J. V. Dacie and Dr. I. Doniach who undertook to review

pathological material; to Dr. F. Campbell Golding and Mr. H. J. Seddon who gave expert help and advice; to Mr. R. Ellis, Dr. D. G. Jones, Mr. D. E. A. Jones, Mr. W. C. Lister, Dr. R. J. Munson, Miss J. Perrett, and Dr. B. M. Wheatley, who carried out much arduous work in connexion with dose measurements; and more especially to Dr. W. M. Court Brown and Dr. R. Doll, who were responsible not only for planning the investigation and the compilation of the results, but who also carried out many of the visits and undertook much of the detailed preliminary work themselves.

We wish to record our appreciation of the work of Mr. S. B. Osborn in connexion with his study on the contribution made by diagnostic radiology to the present level of ionizing radiation in this country. We are grateful to the following bodies who co-operated in the study: the Ministry of Health, the Royal Navy, the Royal Army Medical Corps, the Royal Air Force, the Medical Research Council's Pneumoconiosis Research Unit, the National Coal Board, the Ministry of Pensions and National Insurance, the Ministry of Supply, the Ministry of Labour and National Service, the Atomic Energy Research Establishment and the Red Cross and St. John Joint Committee. Our thanks are also due to those hospitals which supplied detailed data on diagnostic X-ray examinations carried out by them.

A number of commercial firms provided information for the study on radiation dose levels in this country. In this connexion we are particularly grateful to the following: members of the British Radio Valve Manufacturers' Association, Brandhurst Co. Ltd., L. Newmark Ltd., Reliance (Nameplates) Ltd., Smiths English Clocks Ltd., Timex Watches, and the Pedoscope Co. Ltd. Other bodies also provided information and especially the British Dental Association, the London Chamber of Commerce, the Horological Institute of Great Britain, the National Physical Laboratory, the Ministry of Transport and Civil Aviation and the Ministry of Labour and National Service.

Finally, we wish to thank all those who have advised us on specialist problems from time to time during our work, especially Sir Stewart Duke-Elder, Professor Aubrey Lewis and Professor Arnold Sorsby, for the help that they have given.

APPENDIX A

The Incidence of Leukaemia among the Survivors of the Atomic Bomb Explosions at Hiroshima and Nagasaki

Information concerning the cases of leukaemia which are known to have occurred among the survivors of the atomic bomb explosions at Hiroshima and Nagasaki has been supplied by the Atomic Bomb Casualty Commission of the National Research Council of the U.S.A. By the end of August 1955, the diagnosis of leukaemia had been confirmed in 125 patients; in all these cases blood smears and, when necessary, bone-marrow and autopsy material, had been examined by a member of the Commission's staff. In 18 other cases, the diagnosis was suspected but the evidence was less conclusive; and in a further 5 the diagnosis was still under review.

Sixty-one of the confirmed and four of the suspected cases occurred among persons resident in Hiroshima at the time the diagnosis was made, and it is possible to relate these cases to the numbers of persons who survived the explosion and who were recorded as residing in the city subsequently. The incidence of leukaemia among survivors at various distances from the hypocentre of the explosion is shown in Table 1A. The incidence was substantially higher among those who were close to the hypocentre than among those who were more distant from it—128·0 per 10,000 among those less than 1,000 metres away, against 1·6 per 10,000 among those more than 3,000 metres away. Separate incidence rates for persons at each distance who showed major radiation symptoms shortly after the explosion and for persons who did not show such symptoms have been published by Moloney and Kastenbaum (1955).

TABLE 1A

The incidence of leukaemia among survivors of the Hiroshima atomic bomb explosion exposed at various distances from the hypocentre; persons subsequently resident in Hiroshima City only

Distance from hypocentre at time of explosion (m.)	No. of survivors on 1.10.50*	No. of cases of leukaemia		Incidence per 10,000 persons (total cases)
		Confirmed	Suspected	
Less than 1,000 ...	1,250	16	0	128·0
1,000–1,499	10,350	28	1	28·0
1,500–1,999	18,450	6	1	3·8
2,000–2,999	30,350	7	0	2·3
3,000 or more	37,700	4	2	1·6
All distances	98,100	61	4	6·6

* The numbers of survivors have been rounded off to the nearest 50. The estimates differ slightly from those published by Moloney and Kastenbaum (1955) in accordance with data provided by the Atomic Bomb Casualty Commission.

Comparable figures are not available for the incidence of leukaemia in the unexposed population of the rest of Japan, but since leukaemia is invariably fatal it is reasonable to use mortality figures to provide an estimate of the incidence of the disease which might have been expected if no explosion had

taken place. National mortality figures are, however, based on the causes of death given on death certificates, and they are not necessarily suitable for comparison with figures obtained after an intensive search for cases and after the submission of each case to expert clinical scrutiny. In fact, the use of national mortality data is justified only by the fact that the number of deaths attributed to leukaemia among survivors who were 2,000 metres or more from the hypocentre and who cannot have received more than very small amounts of radiation, is close to the expected number calculated on the basis of these data. Table 2A therefore presents for comparison the numbers of cases of leukaemia known to have occurred during the eight years from 1947 to 1954 among residents of Hiroshima who survived at different distances from the hypocentre, and the numbers of deaths from leukaemia which would have been expected in a similar period among populations of the same size and the same sex- and age-distribution, subjected to the age- and sex-specific mortality rates from leukaemia observed in the whole of Japan in 1952. It can be seen that the observed incidence among survivors who were less than 1,000 metres from the hypocentre is 100 times greater than the mortality which would have been expected.

TABLE 2A

A comparison between the observed and the expected incidence of leukaemia among survivors of the Hiroshima atomic bomb explosion exposed at various distances from the hypocentre ; persons subsequently resident in Hiroshima City only

Distance from hypocentre at time of explosion (m.)	No. of cases with onset in the 8-year period 1947–54		No. of deaths expected among the survivors in an 8-year period*	Ratio of total cases observed to expected
	Confirmed	Suspected		
Less than 1,000 ...	15	0	0·15	100·0 : 1
1,000–1,499	28	1	1·32	22·0 : 1
1,500–1.999	6	1	2·33	2·6 : 1
2,000–2,999	6	0	3·96	1·5 : 1
3,000 or more	4	2	4·83	1·2 : 1
All distances	59†	4	12·59	4·7 : 1

* Calculated from the Japanese mortality data for 1952. In calculating the numbers of expected deaths, certain assumptions had to be made about the rate of change of the numbers of survivors in the different age groups, and the figures must be regarded as approximate estimates.

† Two cases referred to in Table 1A are omitted, since the onset of symptoms in one patient was in 1955 and in another patient, who died in April 1955, the date of onset is unknown; the latter patient was exposed at a distance of 2,400 metres from the hypocentre.

The data available for survivors of the Nagasaki explosion are less detailed than those for survivors of the Hiroshima explosion. Altogether 32 confirmed cases and 11 suspected cases are known to have occurred among survivors who subsequently resided in Nagasaki. Of these, 32 and 10 respectively occurred during the eight years 1947-54, and the corresponding total number of deaths which might have been expected in that period on the basis of the national mortality data is approximately 12·3.

Table 3A shows the year of onset of the disease for each of the 108 cases which have occurred among the survivors resident post-war in Hiroshima or Nagasaki, together with estimates of the annual incidence rates. The rates in

the first two years may be under-estimated, since medical organisation was incomplete at that period; in 1953 and 1954 they are almost certainly under-estimated since new cases continue to be discovered and some of the patients give histories of one or more years' duration. The data provide no evidence of a sharp peak in incidence at any particular period after the explosion, nor any clear indication that the incidence has yet begun to diminish by the end of the ninth year.

TABLE 3A

The incidence of leukaemia in different years among survivors of the Hiroshima and Nagasaki atomic bomb explosions; persons subsequently resident in Hiroshima City and Nagasaki only

Year of onset	No. of cases at Hiroshima		No. of cases at Nagasaki		Incidence per 10,000 persons (total cases)		
	Confirmed	Suspected	Confirmed	Suspected	Hiroshima	Nagasaki	Hiroshima and Nagasaki
1946 ...	0	0	0	1	0·0	0·1	0·05
1947 ...	3	0	2	0	0·3	0·2	0·3
1948 ...	7	3	2	1	1·0	0·3	0·7
1949 ...	5	0	1	2	0·5	0·3	0·4
1950 ...	6	1	5	1	0·7	0·6	0·7
1951 ...	11	0	7	3	1·1	1·0	1·1
1952 ...	11	0	6	2	1·1	0·8	1·0
1953 ...	10	0	2	1	1·0	0·3	0·7
1954 ...	6	0	7	0	0·6	0·7	0·7
Part 1955 or date unknown	2	0	0	0	—	—	—
All years	61	4	32	11	6·6	4·5	5·6

Reference

Moloney, W. C. and Kastenbaum, M. A. (1955). Leukemogenic effects of ionizing radiation on atomic bomb survivors in Hiroshima City. *Science*, **121**, 308.

W. M. Court Brown
R. Doll

APPENDIX B

Leukaemia and Aplastic Anaemia in Patients Treated with X-rays for Ankylosing Spondylitis

(*A summary of the findings of the investigation sponsored by the Medical Research Council Committee reporting on the hazards to man of nuclear and allied radiations*)

The investigation had two main objectives: first, to see whether the incidence of leukaemia and of aplastic anaemia was abnormally high in patients treated with X-rays for ankylosing spondylitis ; and second, if the incidence of those conditions was found to be raised, to determine the quantitative relationship between incidence and dose of X-rays.

RESULTS

The incidence of leukaemia and aplastic anaemia

The case records of 13,352 patients (11,287 men and 2,065 women) were studied ; these patients had been treated at 81 radiotherapy centres and sub-centres during the period 1935–54 inclusive. Slightly fewer than half of them were known to have been alive in 1955 or to have died earlier ; the remainder were lost to follow-up at various dates between 1935 and 1954. They had therefore been under observation for periods of between one and twenty years, with an average for the whole group of just under five years.

49 of the patients studied were found to have developed leukaemia, aplastic anaemia, or myelofibrosis (a condition considered to be possibly a variant of leukaemia), and, of these, 46 had died by the end of 1955 ; 28 of them were certified as having died from leukaemia, 13 from aplastic anaemia and 1 from myelofibrosis. Three other patients with leukaemia and 1 with aplastic anaemia were found, who had been certified as having died from other causes.

With the co-operation of the Registrars-General, special efforts were made to recognise all patients in the series who had died of leukaemia or aplastic anaemia. The great majority of such cases have probably been traced despite the incomplete follow-up, but there is reason to believe that a few more might be revealed if the follow-up could be made complete.

The numbers of deaths from leukaemia and aplastic anaemia which could be expected under normal conditions were calculated from the national death rates for those diseases in the general population : they have been estimated as 2·9 and 0·3 respectively up to the end of 1955, but these estimates are certainly too high, as they are based on the assumption that all the patients untraced in 1955 were, in fact, alive at the end of 1955. In the compilation of the numbers of observed deaths from leukaemia and aplastic anaemia for comparison with these figures, only deaths *certified* as due to these conditions could be used, since the figures for expected deaths are based on death-certificate data. Thus enumerated, the numbers of observed deaths from leukaemia and aplastic anaemia are respectively 28 and 13. The differences between the observed and expected deaths are highly significant for both diseases.

Three of the observed cases of leukaemia died within the first year, and in these cases it was assumed that leukaemia was already present at the time of first treatment. If these 'co-existent' cases, and the corresponding expected mortality in the first year after treatment, are omitted, the observed and expected numbers of deaths from leukaemia for 1935–55 are 25 and 2·4 respectively.

Clinical review of cases

All the relevant data for each case were reviewed, and it was concluded that many of the patients certified as having died of aplastic anaemia had, in fact, been suffering from aleukaemic leukaemia. The diagnosis of aplastic anaemia was substantiated in only 4 cases and it was, therefore, not possible to examine the relationship between the incidence of this condition and the dose of X-rays received.

The relationship between the incidence of leukaemia and the X-ray dose

This relationship has been determined for male patients only, in order to avoid difficulties introduced by the possibility of there being a sex-difference in susceptibility to leukaemia; the 3 'co-existent' cases were excluded, and there then remained 37 cases of leukaemia for study.

Details of X-ray treatment were obtained from the case records for 1,878 men, a sample of approximately 1 in 6 of the whole group of 11,287 male patients. The X-ray dose was expressed in two ways. By the first method, the total energy absorbed in the whole body was calculated, and expressed in megagramme-roentgens (Mgm.r.); by the second, the maximum dose in the spinal marrow was determined and expressed in roentgens (r)*. The number of man-years at risk following each level of dose, calculated by each of the two methods, was estimated for the whole group of male patients and was related to the number of cases of leukaemia. The results of these calculations are given in Tables 1B and 2B, which show the crude incidence rates per 10,000 men per year at all levels of dose.

TABLE 1B

The numbers of male patients developing leukaemia and the crude incidence rates after different doses of radiation (measured by the total amount of energy absorbed by the whole body)

	Amount of treatment: whole-body integral dose (Mgm.r.)						
	0	Less than 7·5	7·5–14·9	15–22·4	22·5–37·4	37·5–52·4	52·5 or more
Number of men developing leukaemia ...	—	1	9	9	10	4	4
Crude incidence per 10,000 men per year ...	0·5†	0·7	4·7	5·1	11·3	22·6	60·2

* For the second method extensive measurements were made on a 'phantom' man.

† The rate given for 'no treatment' has been estimated from the national vital statistics for all forms of leukaemia, and weighted to allow for the fact that not all the patients in the series were certified as dying from leukaemia. If lymphatic leukaemia is excluded (as may be more appropriate) the rate is 0·3.

TABLE 2B

The numbers of male patients developing leukaemia and the crude incidence rates after different doses of radiation (measured by the maximum amount received at a point in the spinal marrow)

	Amount of treatment: maximum dose to the spinal marrow (r)						
	0	Less than 500	500 to 999	1,000 to 1,499	1,500 to 1,999	2,000 to 2,749	2,750 or more
Number of men developing leukaemia ...	—	2	8	8	8	6	5
Crude incidence per 10,000 men per year ...	0·5*	2·2	4·1	4·2	11·3	13·0	17·6

* The rate given for 'no treatment' has been estimated from the national vital statistics for all forms of leukaemia, and weighted to allow for the fact that not all the patients in the series were certified as dying from leukaemia. If lymphatic leukaemia is excluded (as may be more appropriate) the rate is 0·3.

CONCLUSIONS

Deaths from leukaemia have been found to be greatly increased among the patients studied, and it is believed that this increase is the result of exposure to X-rays ; the possibility that sufferers from ankylosing spondylitis are unusually sensitive to the action of X-rays cannot, however, be excluded.

Both methods of estimating the dose show a relationship between the crude incidence of leukaemia and the dose of X-rays, with an increase in the incidence over the whole range of dose studied. With neither method is there any evidence of a threshold below which no increase in incidence is produced. Both sets of results, therefore, suggest that even very small amounts of radiation will have an appreciable effect if given to a large enough population. The method based on calculation of the whole-body energy absorption, however, shows a disproportionately greater increase at high levels of dose, whereas that based on calculation of the spinal-marrow dose shows a simple proportional increase at all levels of dose. It is of considerable importance to determine the reason for this discrepancy. If the true relationship is of the curvilinear type suggested by the first method, it remains a theoretical possibility that very small doses (which could not be tested in the investigation) will have no leukaemogenic effect at all ; but such a possibility can almost be ruled out if the relationship is linear.

A full account of the investigation will be published by H.M. Stationery Office as a report in the Medical Research Council's Special Report Series.

W. M. Court Brown
R. Doll

APPENDIX C

The Spontaneous Mutation Rate in Man

In certain circumstances the spontaneous mutation rate of human genes, expressed as the number of mutations per locus per generation, can be estimated with a fair degree of reliability. A direct count of cases due to fresh mutation can be made for a gene which has a dominant effect. This number can also be directly inferred for sex-linked traits shown only in the male. If the incidence of a trait in the general population is known, the mutation rate can be determined from the proportion of cases due to fresh mutation.

When the effects of a gene are very disadvantageous a different line of argument can be used, even though the gene may not be fully manifest in the heterozygous state. The principles on which the indirect estimation of mutation rates can be made were laid down by Haldane (1932), and they are used in the formulae given in Appendix D. The assumption is made that the human population is in a state approaching genetical equilibrium ; it is supposed that disadvantageous genes could not have persisted in the population unless their extinction by selective mortality were balanced by the recurrence of mutation.

In the case of dominant or sex-linked characters associated with very high mortality, the direct measurement of mutation rate can be supplemented by the indirect argument. Estimates which are entirely indirect are untrustworthy, but they have been made for a variety of genes recognised only by their recessive effects. One difficulty with recessive traits is that allowance has to be made for the effects of inbreeding. Another likely source of error is that genetical equilibrium can be maintained not only by mutation but also by a slightly advantageous effect in the heterozygote. Hence, indirect estimates are likely to be too high.

There is a further general difficulty ; this arises from uncertainty as to whether or not a single locus is involved in determining the trait under consideration. Mutations at two or three loci might produce similar characters ; this apparently occurs in haemophilia and also in achondroplasia. In these circumstances any estimate of mutation frequency, direct or indirect, based upon accepting one locus as the hereditary cause of the trait, would be too high by a factor depending upon the frequencies of the component causal genes.

Estimates of mutation rate in man are given in Table 1C. Those for dominant traits are based upon the direct method, though they can all be indirectly supported ; the estimates for sex-linked genes are calculated indirectly, but are supported by direct observation of pedigrees almost certainly containing freshly mutated genes ; those for recessive traits are all indirectly estimated.

I should like to acknowledge here the assistance given me in connection with Appendices C, D, and E by Dr. H. Harris and Dr. D. A. Sprott.

TABLE 1C

Estimates of the spontaneous mutation rates of some human genes

Trait: Name	Trait: Mode of inheritance	Mutation frequency of the causal gene (per million per generation)	Region	Source	Date
Epiloia (tuberose sclerosis)	Dominant	8	England	Gunther and Penrose	1935
Achondroplasia ...	Dominant	45	Denmark	Mørch	1941
Aniridia	Dominant	5*	Denmark	Møllenbach	1947
Microphthalmos (without mental defect)	Dominant	5	Sweden	Sjøgren and Larsson	1949
Retinoblastoma ...	Dominant	15	England	Philip and Sorsby	1947
		23	U.S.A.	Neel and Falls	1951
		4	Germany	Vogel	1954
Partial albinism (with deafness)	Dominant	4	Holland	Waardenburg	1951
Haemophilia (severe type)	Sex-linked	20	England	Haldane	1935
		32	Denmark	Andreassen	1943
		27	Switzerland and Denmark	Vogel	1955
Muscular dystrophy (Duchenne type)	Sex-linked	95	U.S.A.	Stephens and Tyler	1951
		45	N. Ireland	Stevenson	1953
		43	England	Walton	1955
Albinism	Recessive	28	Japan	Neel *et al.*	1949
Ichthyosis congenita ...	Recessive	11	Japan	Neel *et al.*	1949
Total colour blindness...	Recessive	28	Japan	Neel *et al.*	1949
Infantile amaurotic idiocy	Recessive	11	Japan	Neel *et al.*	1949
Amyotonia congenita ...	Recessive	20	Sweden	Böök	1952
True microcephaly ...	Recessive	49	Japan	Komai *et al.*	1955
Phenylketonuria ...	Recessive	25	—	(Appendix D)	—

* This estimate differs by a factor of 2 from that given by the author, but it is based on the author's material.

References

Andreassen, M. (1943). *Haemofili i Danmark.* Munksgaard, Copenhagen.

Böök, J. A. (1952). Fréquence de mutation de la chondrodystrophie et de l'épidermolyse bulleuse dans une population du sud de la Suède. *J. génét. Hum.*, **1**, 24.

Gunther, M. and Penrose, L. S. (1935). The genetics of epiloia. *J. Genet.*, **31**, 413.

Haldane, J. B. S. (1932). *The causes of evolution.* Longmans Green and Co., London.

Haldane, J. B. S. (1935). The rate of spontaneous mutation of a human gene. *J. Genet.*, **31**, 317.

Komai, T., Kishimoto, K. and Ozaki, Y. (1955). Genetic study of microcephaly based on Japanese material. *Amer. J. hum. Genet.*, **7**, 51.

Møllenbach, C. J. (1947). *Medfødte defekter i øjets indre hinder, Klinik og Arvelighedsforhold.* Munksgaard, Copenhagen.

Mørch, E. T. (1941). *Chondrodystrophic dwarfs in Denmark.* Munksgaard, Copenhagen.

Neel, J. V. and Falls, H. F. (1951). The rate of mutation of the gene responsible for retinoblastoma in man. *Science*, **114**, 419.

Neel, J. V., Kodani, M., Brewer, R. and Anderson, R. C. (1949). The incidence of consanguineous matings in Japan, with remarks on the estimation of comparative gene frequencies and the expected rate of appearance of induced recessive mutations. *Amer. J. hum. Genet.*, **1**, 156.

Philip, U. and Sorsby, A. (1947). Unpublished communication.

Sjøgren, T. and Larsson, T. (1949). Microphthalmos and anophthalmos with or without coincident oligophrenia. *Acta psychiat. neurol., Kbh.*, Suppl. No. 56.

Stephens, F. E. and Tyler, F. H. (1951). The inheritance of childhood progressive muscular dystrophy in 33 kindreds. *Amer. J. hum. Genet.*, **3**, 111.

Stevenson, A. C. (1953). Muscular dystrophy in Northern Ireland. I. An account of the condition in fifty-one families. *Ann. Eugen., Lond.*, **18**, 50.

Vogel, F. (1954). Uber Genetik und Mutationsrate des Retinoblastoms (Glioma retinae). *Z. KonstLehre*, **32**, 308.

Vogel, F. (1955). Vergleichende Betrachtungen über die Mutationsrate der geschlechtsgebunden-rezessiven Hämophilieformen in der Schweiz und in Dänemark. *Blut*, **1**, 91.

Walton, J. N. (1955). On the inheritance of muscular dystrophy. *Ann. hum. Genet., Lond.*, **20**, 1.

Waardenburg, P. J. (1951). A new syndrome combining developmental anomalies of the eyelids, eyebrows and nose root with pigmentary defects of the iris and head hair and with congenital deafness. *Amer. J. hum. Genet.*, **3**, 195.

L. S. PENROSE

APPENDIX D

Calculation of the Quantitative Effects of Spontaneous and Induced Mutation Rates in Diseases Caused by Single Genes

In order to estimate the quantitative effect of doubling the mutation rate or of raising it by any given factor, it is necessary to calculate the proportion of cases of the disease in question in each generation which can be attributed to spontaneous mutation. This can be done by using the indirect method of calculating mutation rate ; the steps in the argument are set out in Table 1D. The abnormal allele is represented by **a**, and the normal by **A**.

TABLE 1D

Calculation of the number of cases due to spontaneous mutation in diseases caused by single genes

Steps in the calculation	Dominant trait		Sex-linked trait		Recessive trait	
	Formula	Example: achondroplasia	Formula	Example: haemophilia	Formula	Example: phenylketonuria
1. Sex affected ♂ or ♀	♂ or ♀	—	♂	—	♂ or ♀	—
2. Genotype responsible for the disease	**Aa**	—	**a**	—	**aa**	—
3. Frequency of genotype in population (where q is the frequency of a)...x	2q	1/10,000	q	1/12,000	q^2	1/40,000
4. Comparative loss of fitness associated with the genotype	(1–F)	4/5	(1–F)	7/8	(1–F)	1
5. Mutation rate of **A** to **a** per gene per generation ......m	q (1–F)	40×10^{-6}	q (1–F)/3	24×10^{-6}	q^2 (1–F)	25×10^{-6}
6. Proportion of genotypes (or cases of the disease) due to fresh mutation in each generation .... d	(1–F)	4/5	(1–F)/3	7/24	2q (1–F)	1/100
7. Frequency of abnormal genotypes due to fresh mutation:— (i) in each generation of births .... x × d	2m	80×10^{-6}	m	24×10^{-6}	2mq	$0{\cdot}25 \times 10^{-6}$
(ii) among 20×10^6 births (♂ and ♀) over a period of 30 years ...	$40m \times 10^6$	1,600	$10m \times 10^6$	240	$40mq \times 10^6$	5

In this table three hereditary diseases, achondroplasia, haemophilia and phenylketonuria are used to show the methods applicable respectively to dominant, sex-linked and recessive traits determined by single genes. For the present purpose the population is assumed to be in genetical equilibrium; the loss due to unfitness of genotypes is balanced by recurrent natural mutation. The figures for achondroplasia are derived from Mørch (1941); in Step 3, 1/9,400 has been rounded off to 1/10,000. For haemophilia the figure (7/8) in Step 4 has been taken from Andreassen (1943) and that in Step 3, (1/12,000), agrees with estimates by Haldane (1935). The figures in Steps 3 and 4 for phenylketonuria are derived from Jervis (1939) and Munro (1947). Doubling the spontaneous mutation rate on one occasion would increase the frequency of each disease by a proportion, shown in Step 6, in the next generation.

The results given in Step 7 (ii) in Table 1D apply only to the first generation after doubling. The quantitative effects in subsequent generations can be ascertained by substituting appropriate new incidence figures and repeating the steps of the calculation. Calculations made by this method and extended to cover six generations yield the results shown in Table 2D; the theoretical limiting values obtained after an infinite number of generations are also given. The results are shown graphically in Fig. 1 (p. 32). Two situations are considered: (*a*) the effect of a single doubling of mutation rates in one generation, and (*b*) the effect of permanently doubling the mutation rate.

TABLE 2D

The effects, expressed as the increase in incidence per hundred cases, of doubling the mutation rates, (a) *in one generation only, and* (b) *permanently, for three hereditary traits*

(*a*) *Doubling in one generation only*

Number of generations	Increase in incidence per hundred cases		
	Achondroplasia (Dominant)	Haemophilia (Sex-linked)	Phenylketonuria (Recessive)
0	0	0	0
1	80	29	1
2	16	29	1
3	3	16	1
4	1	10	1
5	0	6	1
6	0	4	1
Infinite	0	0	0

(*b*) *Permanent doubling*

Number of generations	Increase in incidence per hundred cases		
	Achondroplasia (Dominant)	Haemophilia (Sex-linked)	Phenylketonuria (Recessive)
0	0	0	0
1	80	29	1
2	96	58	2
3	99	74	3
4	100	84	4
5	100	90	5
6	100	94	6
Infinite	100	100	100

References

Andreassen, M. (1943). *Haemofili i Danmark*. Munksgaard, Copenhagen.
Haldane, J. B. S. (1935). The rate of spontaneous mutation of a human gene. *J. Genet.*, **31**, 317.
Jervis, G. A. (1939). The genetics of phenylpyruvic oligophrenia. *J. ment. Sci.*, **85**, 719.
Mørch, E. T. (1941). *Chondrodystrophic dwarfs in Denmark*. Munksgaard, Copenhagen.
Munro, T. A. (1947). Phenylketonuria: data on 47 British families. *Ann. Eugen., Lond.*, **14**, 60.

L. S. PENROSE

APPENDIX E

Estimate of the Incidence of Cases of Schizophrenia and Manic Depressive Reaction due to Spontaneous Mutation

The calculation of (d), the proportion of cases which can be attributed to fresh mutation in each generation, for the conditions discussed in paragraphs 148–150, can be set out as shown in Table 1E.

TABLE 1E

Calculation of (d), *the proportion of cases due to fresh mutation in each generation, in schizophrenia and manic depressive reaction*

Steps in the calculation	(i) Schizophrenia		(ii) Manic depressive reaction	
	Formula	Example	Formula	Example
1. Sex affected ♂ or ♀	♂ or ♀	—	♂ or ♀	—
2. Genotype responsible for predisposition to the disease ...	aa	—	Aa	—
3. Frequency of genotype in population (where q is the frequency of a) x	q^2	1/100	2q	1/200
4. Comparative loss of fitness associated with the genotype ...	(1–F)	1/20	(1–F)	1/70
5. Mutation rate per gene per generation ... ... m	q^2 (1–F)	1/2,000	q (1–F)	1/28,000
6. Proportion of predisposed cases due to fresh mutation in each generation ... ... d	2q (1–F)	1/100	(1–F)	1/70

(*i*) *Schizophrenia*

This is a type of mental disease which has its onset at about the age of 30 years on the average. There are many degrees of severity, and males are more often affected than females. The genetical predisposition occurs in subjects who are, according to Kallmann (1938), homozygous for a specific recessive gene; it has an incidence of about 1/100 in European populations (Fremming, 1947). On the basis of a rough survey of hospital data, it is assumed here that only one-tenth of predisposed subjects become chronically incapacitated. Neglecting sex differences, the fertility of these incapacitated patients is reduced by 1/2 (Essen-Möller, 1935). The total loss of fitness of predisposed genotypes is thus $1/10 \times 1/2 = 1/20$. It follows that $d=2q(1-F)=1/100$, as shown in Table 1E. In a generation of 20×10^6 births the number of incapacitated people would be 20,000, of whom 200 would be cases due to fresh mutation.

(*ii*) *Manic depressive reaction*

This is a type of mental disease with mean age of onset at about 40 years. There are many degrees of severity, and females are more often affected than males. The genetical predisposition, which is commonly believed to depend upon a dominant gene (Marrell, 1951), has a frequency in the population of about 1/200; Mayer-Gross, Slater and Roth (1954) quote 0·35 per cent for Scotland and 1 per cent for Sweden and Denmark as the total morbidity risk of affective psychosis. The incidence of chronic breakdown among those who are predisposed can be estimated at one-seventh and, for such incapacitated patients, fertility is reduced, according to observations by Essen-Möller, by a factor of 1/10. Hence the total loss of fitness for predisposed genotypes is $1/7 \times 1/10 = 1/70$. It follows that $d = (1-F) = 1/70$. In a generation of 20×10^6 births there would be 14,000 people incapacitated, of whom 200 would be cases due to fresh mutation.

References

Essen-Möller, E. (1935). Die Fruchtbarkeit gewisser Gruppen von Geisteskranken. *Acta psychiat. neurol.*, Suppl. 8.

Fremming, K. (1947). *Morbid risk of mental diseases and other mental abnormalities in an average Danish population.* Munksgaard, Copenhagen.

Kallmann, F. J. (1938). *The genetics of schizophrenia.* J. J. Augustin, New York.

Marrell, D. J. (1951). Inheritance of manic depressive psychosis. *Arch. Neurol. Psychiat., Chicago.* **66**, 272.

Mayer-Gross, W., Slater, E. O. and Roth, M. (1954). *Textbook of psychiatry.* Cassell and Co., London.

L. S. PENROSE

APPENDIX F

The Effect of Changing the Mutation Rate on Characters Showing Continuous Variation about the Normal

Member genes of a polygenic system cannot be followed as individuals in our observations. We must therefore measure mutation by the increment it adds to heritable variation, instead of measuring it by the frequency of change, as is done in the case of individually traceable genes.

The increment added to the variance per generation has been estimated for spontaneous mutation in two hair characters in *Drosophila* (Clayton and Robertson, 1955; Durrant and Mather, 1954; Paxman, 1955). Technically this is a difficult operation, but the estimates agree surprisingly well for each character when allowance is made for various possible sources of bias. The two characters agree, also, in showing an increment of about 1×10^{-3} of the amount of heritable variation estimated by Clayton and Robertson to be present in a normal population of *Drosophila*. The estimate may easily be too small by a factor of 2 or 3, but seems unlikely to be out by a factor of 10. This finding suggests that the relation between the amount of heritable variation in a population and the mutation rate is roughly linear, and certain theoretical considerations point the same way. It also shows that a marked increase in the mutation rate for a few generations would have only a trivial effect on the heritable variation of the population, and that, even with a persistent increase, the new equilibrium showing the full effect of the raised mutation rate in raising heritable variation would take very many generations to achieve.

Clayton and Robertson also record the result of irradiating the adults of each generation with an X-ray dose of 1,800 r. The increment added in each generation to the heritable variation available to selection was about ten times the spontaneous increment. Thus the dose which doubles the effect of spontaneous mutation would be some 200 r as measured by this criterion. But the new heritable variation available to selection in these experiments seems to have represented only about one-sixth of all the new heritable variation as measured directly by the increase in phenotypic variation. If we take the overall total therefore, 200 r must have produced about six times the spontaneous increment, so that the doubling dose becomes just over 30 r. This is more in keeping with the figure obtained from lethal mutation, though it might have been expected that the polygenic figure would be higher, not lower, than the monogenic, because of the way in which mutations can balance one another's effects in a polygenic system. It would, in any case, be unwise to place great confidence in these calculations, both because other experiments of the same kind have given results even more difficult to assess, and because doses as heavy as 1,800 r produce so many lethal mutations and so much structural change in the chromosomes that the polygenic effects may well be quantitatively distorted.

In attempting to extrapolate from these findings to the effects of irradiation in natural, including human, populations, two points must be borne in mind. Firstly, the flies which yielded these observations were from inbred lines, so that any mutation in the polygenic system would add its quota to the increase in variation. In a natural population, however, variation is already present, and mutation from one allele to another, where both already exist

and are not uncommon in the population, could add little if anything to the variation. Only mutation in genes whose alleles are rare or absent in the population will contribute materially to an increase in variation, and the contribution will fall off as the alternative alleles become more common. So if most of the member genes of a polygenic system are already varying, mutation will add correspondingly little to the total variation. On the other hand, if, as seems likely, many of the genes which can prospectively contribute to the variation of a polygenic system are not doing so because only one allele is present or at least common, new mutation increases the number of loci contributing to the variation which will then increase correspondingly. The roughly linear relation between mutation and variation suggested by the experiments is thus to be regarded as a maximum effect, the closeness of approach to it depending on the initial conditions of variation prevailing in the population in question.

The second point concerns the properties of heterozygotes. The linear relation would cease to hold if there were any innate advantage of individuals heterozygous for the genes over others homozygous for them. A situation analogous to balanced polymorphism would then arise, and at equilibrium the heritable variation would become independent of mutation rate. No such heterozygotic advantage was detected in the variation arising by mutation, even though Paxman made a special search for it ; nor is it likely on general grounds. Such an advantage must, however, remain as a possible, even if unlikely, additional reason for regarding a linear relation as representing the maximum, rather than the regularly realisable, effect of mutation on variation in populations such as those of man. In other words a permanent doubling of the mutation rate would not be expected to do more, and under some circumstances might do less, than double the heritable variation in the population.

References

Clayton, G. and Robertson, A. (1955). Mutation and quantitative variation. *Amer. Nat.*, **89**, 151.

Durrant, A. and Mather, K. (1954). Heritable variation in a long inbred line of *Drosophila*. *Genetics*, **27**, 97.

Paxman, G. J. (1955). *Mutation and balance in polygenic systems*. Thesis, Birmingham.

K. MATHER

APPENDIX G

The Effect on the Distribution of Intelligence of Increasing the Heritable Variation

Under conditions of natural selection the effect of raising the heritable, and hence the total, variation of expression of a character, some particular expression of which is the fittest, must in general be to lower the average fitness in the population; though where the mean departs widely from the most advantageous expression of the character the fitness of selected groups might be raised. Furthermore, where all expressions of the character may be displayed, the overall fall in fitness must be directly related to the increase in variation. A fall in fitness is not, however, to be translated directly into social load when we are considering the consequences of raising the heritable variation in such a character as mental capacity in man.

The intelligence score of an individual is derived from his performance in a series of tests, and the frequency distribution obtained from a population must therefore reflect the structure of the tests. The distribution obtained is generally treated as being normal, but in fact certain disturbances occasionally appear, and they seem to be of a type which no simple transformation or statistical adjustment can remove. It is very likely that such discrepancies spring from innate features of the test, and they should not, in any case, be allowed to obscure the essentially normal nature of the distribution. Normality will in fact be assumed in the following discussion. It is considered that by avoiding the use of very narrow ranges and, more particularly, by discusssing relative rather than absolute effects, broadly valid conclusions should be attained.

I am informed by Professor F. A. Peel of the Department of Education in the University of Birmingham, that in educational discussions the distribution of intelligence scores is taken as normal and is standardised to a mean of 100 with a standard deviation of 15. The scores have been found to approximate to I.Q.'s as measured by the original tests, and are commonly referred to as 'I.Q. scores'. In these terms, Professor Peel further informs me, children with an I.Q. of less than 70 are generally regarded as educationally sub-normal and as requiring education in special schools; those with an I.Q. of betwen 70 and 80 are regarded as needing special teaching in ordinary schools; and those with an I.Q. of over 115 as being of grammar school quality. These figures are to be taken only as general guides since they are applied neither rigorously nor uniformly throughout the country.

For information and advice on the estimation of the heritable variation in respect of I.Q., I am indebted to Dr. J. A. Fraser Roberts. Sibs show a correlation close to 0·5 in this character. Data on parent-offspring relations are less full, but suggest a similar figure, so that there is no good reason to postulate any over-dominance or heterozygous advantage. Taken on their face value, such figures would indicate virtually complete genetic determination, but there is strong assortative mating in respect of this character and there is also the effect of a common home environment to be taken into account in assessing the genetic meaning of these familial correlations. Observations on twins and foster children would seem to indicate $\frac{3}{4}$ as the fraction of variation which is heritable. However, lest this should be an over-estimate, parallel calculations have been made, assuming fractions of $\frac{1}{2}$ and $\frac{3}{4}$ as likely to straddle the true situation. Should even the figure of $\frac{1}{2}$ be too high the effect of increasing the heritable component would be correspondingly smaller. Assortative mating has been disregarded, as we may

reasonably assume that its incidence would not be affected by alteration in the amount of heritable variation, so that its relative effect would remain the same.

Calculations have been made of the effects of raising the heritable variation (V_H) to 1·25, 1·50 and 2·00 times its present value, assuming that environmental variation (V_E) remains unaltered. Thus, taking V_H to be $\frac{3}{4}$ of the total variation (V_T) we have:

$$V_H = \tfrac{3}{4}V_T = \tfrac{3}{4} \times 15^2, \text{ and}$$
$$V_E = V_T - V_H = \tfrac{1}{4} \times 15^2,$$

so that doubling the heritable variation would give us the new total:

$$V_T' = V_E + 2V_H = (\tfrac{1}{4} \times 15^2) + (2 \times \tfrac{3}{4} \times 15^2) = 1\tfrac{3}{4} \times 225 = 393{\cdot}75$$

and the new distribution of I.Q. would have a standard deviation of 19·84, the mean remaining at 100. The proportion of individuals expected to have, for example, an I.Q. of less than 70 can then be found as the area in the tail of the distribution cut off by the ordinate falling short of the mean by a normal deviate of $\frac{30}{19{\cdot}84} = 1{\cdot}512$.

The results of this and similar calculations are shown in Table 1G, where they are expressed as values relative to the proportion calculated as falling into corresponding classes with the distribution as it is now assumed to be. Thus the assumed present distribution ($\bar{x} = 100$, $s = 15$) gives 2·27 per cent of individuals with an I.Q. of less than 70. With the heritable fraction at $\frac{3}{4}$ of the total and doubled, 6·53 per cent fall below a score of 70, making a relative value of $\frac{6{\cdot}53}{2{\cdot}27} = 2{\cdot}88$. In other words, on these assumptions, doubling the heritable variation would nearly treble the number of children with an I.Q. of less than 70 (i.e. those needing to be taught in special schools, as judged by a common convention of today). In addition to the relative changes in the numbers with an I.Q. of less than 70, figures are also given for those in the classes with I.Q. less than 75 (a figure sometimes taken to indicate the need for special schooling), I.Q. between 70 and 80 (special teaching), and I.Q. over 115 (grammar school).

TABLE 1G

The effects of raising the heritable variation on the frequencies of different intelligence groups

Intelligence group	Present proportion per cent of population (assuming a normal distribution)	Assumed present heritable fraction	Factor of increase relative to present proportion with heritable variation raised to: (constant mean)			(falling mean)
			1·25	1·5	2·0	2·0
I.Q. < 70	2·27	$\frac{1}{2}$	1·31	1·62	2·26	3·24
		$\frac{3}{4}$	1·46	1·94	2·88	4·51
I.Q. < 75	4·78	$\frac{1}{2}$	1·22	1·42	1·82	2·50
		$\frac{3}{4}$	1·32	1·63	2·17	3·24
70<I.Q.<80	6·85	$\frac{1}{2}$	1·09	1·16	1·27	1·59
		$\frac{3}{4}$	1·13	1·22	1·33	1·75
I.Q.>115	15·87	$\frac{1}{2}$	1·09	1·17	1·30	1·00
		$\frac{3}{4}$	1·13	1·24	1·42	

In these calculations the variance has been assumed to change without alteration of the mean, so that the proportion of high I.Q. increases with the proportion of low I.Q. A further calculation has been made in which, as the variance increases, the mean is allowed to fall so as to keep constant the proportion with I.Q.'s of over 115. This is intended to illustrate the kind of result which would be obtained if mutation were preponderantly, but not wholly, degradatory. The relative changes for doubled heritable variation on this assumption are also shown in the table.

It should be remembered that these changed proportions would be achieved by corresponding increases in the mutation rate, only when equilibrium had been reached or at least closely approached, that is to say, after very many generations.

K. MATHER

APPENDIX H

The Doubling Dose of Radiation for Various Plants and Animals

In paragraphs 171-182 we were concerned with finding some quantitative measure of the effectiveness of radiation in causing mutations, with the purpose of using this estimate to establish maximum levels of exposure which are genetically tolerable. In this context, one must be particularly careful not to under-estimate the effects of radiation. In order to express its influence in terms of a 'doubling dose', we should try to arrive at a figure for the lowest dose of radiation which changes mutation in a way which is effectively equivalent to a doubling of the mutation rates of every gene. For those genes with very low spontaneous mutation rates a doubling of the rate will be relatively unimportant. Among the genes with relatively greater spontaneous mutation rates, some may be more sensitive to radiation than others. What we need to estimate is the dose of radiation which doubles the mutation rate of a sample of these more sensitive genes sufficiently large to be physiologically representative of mutations in general. This might be called a 'minimum representative doubling dose'.

After pointing out (paragraph 176) that this can scarcely be less than the naturally occurring dose of radiation, we discuss the possible modifications of this bedrock minimum in terms of an argument which was originally largely due to J. B. S. Haldane (1948). This consists of an attempt to estimate the fraction of the spontaneous mutation rate in man which can plausibly be attributed to natural radiation. The argument proceeds by analogy with the conditions in other organisms, and particularly those in the fruit-fly (*Drosophila*) and the mouse.

In *Drosophila,* suppose that:

f = the fraction of spontaneous mutations due to natural radiation,

r = the rate of natural radiation (in r per day),

m = the rate of mutation induced by 1 r,

s = the spontaneous mutation rate,

t = the average age at reproduction (in days).

Then we shall have:

$$f = \frac{m\,r\,t}{s}$$

Similarly if capital letters represent the same factors in man, we shall have

$$F = \frac{M\,R\,T}{S}$$

Now the spontaneous mutation rate and the rate of induced mutation (s and m) are much better known for flies than for man (S and M). Thus, the procedure is to arrive at F by first finding as good a value as possible for f and then modifying this according to the relation,

$$F = f \times \frac{M\,R\,T\,s}{m\,r\,t\,S}$$

In his original presentation of the argument, Haldane adopted for f a value (0·001) which had been calculated by D. E. Lea (1946). However, it has recently been pointed out (Spiers, 1956) that Lea based his calculation on an estimate of natural radiation of 2·2 milliroentgens (mr) per day, which is about eight times greater than that accepted at the present time (paragraph 200). The value for f has to be reduced accordingly. For the sake of simplicity we have taken it as 0·0001 (paragraph 178), although this may be a slight under-estimate.

This value for f is based on an estimate of 0·15 per cent sex-linked lethals. Lea argues, in agreement with other authors who have considered the matter, that there may be about 1,000 genes capable of mutating to sex-linked lethals in *Drosophila*; his value for the spontaneous mutation rate can therefore be expressed as $1{\cdot}5 \times 10^{-6}$ per locus. The statement (paragraph 178) that the human spontaneous mutation rate is probably about five times as great as this is based on the observation that the mutation rates of several human genes are around 10 per million per generation (cf. Table 1C).

The opinion (paragraph 179) that mouse genes are more sensitive to radiation than those of *Drosophila* (i.e. have a higher value for m) is based on the work of W. L. Russell (1954). It is disputed by some authors; but if one adopts it, and takes it to suggest the hypothesis that human genes are also more sensitive, the result is to increase the value of F, and thus to lower the estimate of the minimum representative doubling dose. In order to be as cautious as possible, we have therefore adopted these assumptions.

The estimate (paragraph 185) that one human germ cell in ten carries a new mutation is a minimum figure arrived at in calculations by H. J. Muller (1950, 1954).

Some typical figures for doubling doses derived from experiments on plants and animals are given in Table 1H. These figures are open to a wide margin of statistical error, as the number of spontaneous mutations was always small; in most instances values smaller or larger by a factor of 2 are not excluded.

There have been many other reports of experiments in which mutations were induced by ionizing radiations, especially in plants and lower organisms; however, in the great majority of these, either the control series was too small for any spontaneous mutation to be observed, or the apparent mutants found were not confirmed by genetic test. The figures quoted here are probably representative.

References

(*a*) *Text*

Haldane, J. B. S. (1948). The formal genetics of man. (Croonian Lecture.) *Proc. roy. Soc.*, Ser. B, **135**, 147.

Lea, D. E. (1946). *Actions of radiations on living cells*. Cambridge University Press, p. 181.

Muller, H. J. (1950). Our load of mutations. *Amer. J. hum. Genet.*, **2**, 111.

Muller, H. J. (1954). The manner of dependence of the permissible dose of radiation on the amount of genetic damage. *Acta radiol., Stockh.*, **41**, 5.

Russell, W. L. (1954). Genetic effects of radiation in mammals. Chapter 12 in: *Radiation Biology.*, Ed. A. Hollaender, McGraw-Hill, New York.

Spiers, F. W. (1956). Genetical effects of radiation from products of nuclear explosions. *Nature, Lond.*, **177**, 226.

TABLE 1H

Some typical figures for the doubling dose of radiation in various higher organisms

Group	Genus and species	Cell stage irradiated	Genes studied	Doubling dose (r)	Source of information (*see list of references*)
					No.
Plant	*Zea mays*	Pollen	Four recessive visibles	28	1
	Oenothera organensis ...	Pollen	Self-incompatibility	60	2, 3
	Prunus avium	Pollen	Self-incompatibility	60	2, 3
Insect	*Drosophila melanogaster* ...	Spermatozoa	Sex-linked lethals	50	4
	Drosophila melanogaster ...	Spermatozoa (aged) ...	Sex-linked lethals	140	5, 6
	Drosophila melanogaster ...	Spermatogonia ...	Sex-linked lethals	8	7
	Drosophila melanogaster ...	Oocytes and oogonia ...	Nine recessive visibles	390	8
	Drosophila melanogaster ...	Spermatozoa	White eye	60	9
Mammal	*Mus musculus*	Spermatogonia ...	Dominant visibles, semi-steriles, sex-linked lethals	50	10
	Mus musculus	Spermatogonia ...	Seven recessive visibles ...	50	11, 12

(*b*) *Table 1H*

(1) Konzak, C. F. and Singleton, W. R. (1956). The effects of thermal-neutron radiation on mutation at endosperm loci in maize. *Proc. nat. Acad. Sci., Wash.*, **42,** 78.

(2) Lewis, D. (1948). Structure of the incompatibility gene. I. Spontaneous mutation rate. *Heredity*, **2,** 219.

(3) Lewis, D. (1949). Structure of the incompatibility gene. II. Induced mutation rate. *Heredity*, **3,** 339.

(4) Spencer, W. P. and Stern, C. (1948). Experiments to test the validity of the linear r-dose/mutation frequency relation in *Drosophila* at low dosage. *Genetics*, **33,** 43.

(5) Caspari, E. and Stern, C. (1948). The influence of chronic irradiation with gamma-rays at low dosages on the mutation-rate of *Drosophila melanogaster*. *Genetics*, **33,** 75.

(6) Uphoff, D. E. and Stern, C. (1949). The genetic effects of low intensity irradiation. *Science*, **109,** 609.

(7) Muller, H. J., Herskowitz, I. H., Abrahamson, S. and Oster, I. I. (1954). A non-linear relation between X-ray dose and recovered lethal mutations in *Drosophila*. *Genetics*, **39,** 741.

(8) Muller, H. J., Valencia, J. I. and Valencia, R. M. (1950). The frequency of spontaneous mutations at individual loci in *Drosophila* and the production of mutations at individual loci in *Drosophila* by irradiation of oocytes and oogonia. *Genetics*, **35,** 125.

(9) Bonnier, G. and Lüning, K. G. (1949). Studies on X-ray mutations in the *white* and *forked* loci of *Drosophila melanogaster*. *Hereditas, Lund*, **35,** 163.

(10) Charles, D. R. (1950). Radiation-induced mutations in mammals. *Radiology*, **55,** 579.

(11) Russell, W. L. (1952). X-ray induced mutations in mice. *Cold Spr. Harb. Symp. quant. Biol.*, **16,** 327.

(12) Carter, T. C., Lyon, M. F. and Phillips, R. J. S. (1956). Induction of mutations in mice by chronic gamma irradiation. *Brit. J. Radiol. N.S.*, **29,** 106.

C. H. WADDINGTON
T. C. CARTER

APPENDIX J

The Dose of Radiation Received in Human Tissues from Natural Sources

All living organisms absorb ionizing radiation from sources which are either present in their environment or incorporated in their own tissues. The most important 'external' radiations are cosmic rays and the radiations arising from elements of the uranium and thorium series present in the earth or the air and from the potassium 40 content of the earth and of vegetable matter. The 'internal' irradiation arises chiefly from the potassium in tissues, but there is also a small contribution from carbon 14 and in some cases soft tissues within bone receive radiation from very small amounts of radium present in the skeleton. Radon in the atmosphere, besides contributing its quota to the external radiation, may also add to the internal dose, by access of the radioactive material to the tissues via the lungs. Because alpha rays and beta rays are so readily absorbed, even by the elements of low atomic number which comprise the soft tissues, the dose from external sources may be regarded as entirely due to cosmic rays and gamma rays. The dose from internally absorbed radioactive materials, however, arises in large measure from beta rays and, when present, from alpha rays.

The radiation dose to human tissues from this normal background is small compared with doses known to cause immediate somatic change, and its significance is presumably to be sought in possible long-term effects; of these the genetic and carcinogenic actions of ionizing radiation would appear to be the most likely. The critical tissues considered in this appendix are, therefore, the gonads and the osteocytes of the Haversian systems in bone. An estimate of the dose to these tissues is made as far as present data allow, and although this is attempted for a number of different localities, it must be emphasised that knowledge of the basic data is far from complete.

All doses are expressed as soft-tissue doses in rads, and where calculations have involved the quantity 'W,' the energy per ion pair formed in air, a value of 34 eV has been taken as representative of recent experimental determinations of that constant.

SOURCES OF EXTERNAL RADIATION

Cosmic rays

The cosmic ray ionization intensity in air at sea-level (and geomagnetic latitudes above 41° N) has been variously quoted in the literature over a range of from 1·5 to 2·8 ion pairs/cc/sec. The differences have arisen largely in the interpretation of high-pressure ionization-chamber measurements, and the correct method of analysis appears to be that used by Clay and his co-worker (1938), and re-examined and supported by further experiments by Burch (1954). The mean of three values given by these workers and corrected to ionization in free air is 1·92 ions/cc/sec. Converting this value to a tissue dose gives:—

cosmic ray dose-rate to soft-tissues = 0·028 rad/year.*

This dose-rate applies to any soft tissues in the body, including the gonads and osteocytes, and is typical for most locations at the earth's surface above latitude 41° N. It may be some 20 per cent less in basements of tall buildings, which absorb the 'soft' cosmic ray component; but any considerable

* 1 rad corresponds to a dose of about 1·07 r in soft tissues.

reduction in dose-rate will only occur in deep underground situations. At a height of 10,000 ft. the cosmic ray dose-rate is increased by a factor of 3 or more, but it will be seen later that cosmic rays contribute only a fraction of the total tissue dose, and that, in consequence, the tissue dose-rate at this height might be only some 40 per cent greater than at sea-level.

The dose-rate of 0·028 rad/year is less than the figure 0·035 rad/year, given, for example, by Libby (1955). For the reasons given above, the lower figure appears to be the correct one and is retained in the present analysis. It is a matter of some discussion whether or not the small component of slow particles in cosmic radiation has a higher R.B.E. than the fast-meson component, the R.B.E. of which has been taken as unity. Some mutations are reported as being less effectively produced by heavily ionizing particles; in a few other cases the R.B.E. for heavy particles has been found to be in the region of 5. In the extreme case, the slow-particle component would not appear likely to add as much as 0·01 rad/year to the effective cosmic ray dose. On the other hand, some shielding of the soft gamma ray component of the cosmic radiation by building structures occurs, which reduces the dose below the unshielded value. It would seem best, in view of this and the possible effect of a higher R.B.E., to accept the unshielded value of 0·028 rad/year as the best estimate for sea-level and latitudes above 41° N.

Local gamma rays

Under most conditions of life, gamma radiation from local surroundings is responsible for the greater fraction of the external radiation dose. Measurement of this contribution, however, has been made in comparatively few places.

Some measurements of local gamma ray dose-rates in Leeds and Aberdeen (Spiers and Griffith, 1956) are summarised in Table 1J. The results cover only limited types of situation, but the concordance between the dose-rates in brick and concrete buildings, whether in Leeds or in Aberdeen, suggests that they may be fairly representative of the dose-levels in areas which are not specially radioactive and in buildings not made of granite. The dose-rates in Leeds determined with a lightly shielded counter, are about 20 per cent higher than the background measurements previously reported, but this effect was shown to be due to the difference in shielding in the two measurements. The results in Table 1J represent the local gamma ray dose-rates under conditions of light shielding.

TABLE 1 J

Measurements of local gamma ray dose-rates in Leeds and Aberdeen (*Spiers and Griffith*, 1956)

Type of building (or 'out-of-doors')	Location	Local gamma ray dose-rate (rad/year)
I. All granite ...	(*a*) Aberdeen—laboratory	0·107
	(*b*) Aberdeen—bell tower	0·099
	(*c*) Aberdeen—entrance hall	0·101
II. Brick and concrete	(*a*) Aberdeen—rooms on various floors	0·073
	(*b*) Leeds—room in hospital building...	0·081
	(*c*) Leeds—single-storey laboratory ...	0·080
	(*d*) Leeds—various rooms in house ...	0·077
III. Out-of-doors ...	Leeds—garden of house II(*d*) above ...	0·048

Sievert and Hultqvist (1952) and Sievert (1955) have reported measurements of the total 'cosmic ray plus gamma ray' background in Swedish houses and in out-of-door situations. Some of the Swedish results are given in Table 2J where an allowance has first been made for the cosmic ray fraction and the residual ionization converted to tissue dosage. The mean dose-rates recorded were based on measurements in about 70 houses. Variations in dose-rate were most marked in Type 3 houses where, in some, values 50 to 100 per cent above the mean in Table 2J were recorded.

TABLE 2J

Measurements of the total 'cosmic ray plus gamma ray' background in Swedish houses and in out-of-door situations in Sweden (*Sievert and Hultqvist,* 1952 ; *Sievert,* 1955)

Situation		Gamma rays only (ions/cc/sec.)	Mean dose-rate (rad/year)
Indoors, centre of room	Wooden houses	4·0	0·059
	Brick and concrete houses (Type 1)	6·2	0·091
	Brick and concrete houses (Type 2)	6·5	0·095
	Brick and concrete houses (Type 3)	14·8	0·216
Outdoors	Stockholm streets	5·8	0·085
	Over igneous rocks	3·9 to 8·3	0·06 to 0·12
	Over clay	3·4	0·05

The local gamma ray dose-rate can also be estimated approximately at some places in South-west England from geiger-counter recordings reported by Peirson (1951). Taking the counting rate given by Peirson for a 'normal' background situation, and allowing for the cosmic ray fraction, a counting rate of about 1·3 cts/min. per cm^2 of projected cathode area is deduced for this 'normal' local radiation. Assuming an over-all efficiency of the counter assembly of 0·6 per cent, and a mean gamma ray energy of 1 MeV, a gamma ray flux is deduced which corresponds to a tissue dose-rate of 0·05 rad/year, a figure reasonably in accord with measurements in Leeds and in Sweden away from areas of high radioactivity. Applying the same analysis to the recorded counting rate in St. Ives and its neighbourhood, the local gamma ray dose-rate is deduced as approximately 0·25 rad/year. Great accuracy cannot be claimed for this estimation in view of the assumptions made, but it is of the same order as the values given in Table 2J for areas of known high radioactivity in Sweden. Dose-rates of 0·3 rad/year and over have been reported in some parts of Cornwall (Wood and Willey, 1954).

Atmospheric radon

Radiation from the break-down products of atmospheric radon also add to the external gamma ray dose. This effect may be expected to be small under most conditions, but so far no measurements have been made to distinguish its contribution from that of gamma rays from the solid surroundings. Peirson and Franklin (1951) have calculated that at ground level an atmospheric radon content of 3×10^{-13} c/1 produces a gamma ray flux of the order of 10 quanta/cm^2/min. Taking a mean energy of 0·8 MeV for radium B and C gamma rays, the tissue dose-rate for this flux is 0·0022 rad/year. Anderson, Mayneord and Turner (1954) have reported levels of atmospheric radon in London (in May 1953) which averaged $2\text{–}3 \times 10^{-12}$ c/1. and under these conditions the external gamma ray dose from atmospheric radon is of the order 0·02 rad/year—a contribution of nearly the same magnitude as that due to cosmic rays.

SOURCES OF INTERNAL RADIATION

Potassium 40

The following data have been used in the calculation of the dose-rate from the potassium content of the body:—

Mean potassium content of body	$= 0{\cdot}215$ per cent
Specific β-activity of K	$= 27{\cdot}4\ \beta$'s/sec./g. K
Specific γ-activity of K	$= 3{\cdot}5\ \gamma$'s/sec./g. K
Mean β-ray energy of K 40	$= 0{\cdot}605$ MeV
Mean γ-ray energy of K 40	$= 1{\cdot}46$ MeV

Because the mean range of the beta particles of potassium 40 is only some 2 mm, the dose-rate in a given organ is determined mainly by its own potassium content. In the absence of precise values for the potassium content of the gonads, the mean for the whole body, derived from Shohl's data (1939) is adopted. The total tissue dose-rate derived from the energy released per g. of tissue, is then:—

$$\text{tissue dose-rate due to K} = 0{\cdot}018(\beta) + 0{\cdot}002(\gamma) = 0{\cdot}020 \text{ rad/year.}$$

In relation to this calculation there may be doubt as to the precise value for the potassium content of the gonads. So far it has only been possible to make flame-photometric measurements on tissues taken from two post mortem examinations. The results, obtained through the kind co-operation of Dr. F. M. Parsons of the Urological Department of the General Infirmary at Leeds, are as follows:—

Case and age		*Potassium content (mg./100 g.)*	*Sodium content (mg./100 g.)*
A. 67 yr. (testes)	(i)	190	205
	(ii)	240	172
B. 28 yr. (ovaries)	(i)	188	200
	(ii)	197	204

Three results are concordant and one (Aii) is suspect in that the sodium value is so low. They suggest, however, that no great error is being made in assuming an average potassium content of 0·215 per cent, as given by Shohl. The tissues were taken from the central parts of the gonads, and the potassium content should be representative of the average value over dimensions of a few mm. of the tissues containing the germ cells.

Carbon 14

Carbon in living systems contains approximately 1 part carbon 14 in 10^{12}, and has a specific beta ray emission of 0·2 beta particles per sec per g. carbon, with a mean energy of 0·053 MeV. Taking the carbon content of tissue as 18 per cent, the energy deposition due to the carbon 14 amounts to a tissue dose-rate of only 0·001 rad/year.

Radon and its disintegration products

An estimation of tissue dose arising from the inhalation of air containing radon can be made if, in the absence of complete information on all the factors concerned, some simplifying assumptions are made. The concentration of radon in the atmosphere is regarded as uniform, and it is assumed that the break-down products (radium A, B, C and C^1) are in equilibrium with the radon and are uniformly suspended in the air. The calculation is then made in two parts: (1) for a steady level of radon (plus disintegration

products) in body-tissues via the blood in contact with the radon in alveolar air, and (2) for a steady intake into the lungs of the disintegration products formed in the air.

The solubility of radon in water at 37° C. is 0·17, and in fat the figure is about five times higher. If the concentration of pure radon in alveolar air is *C*, and is regarded for the moment as free of disintegration products, the concentration of radon in the 50 kg. of aqueous tissue will be 0·17 *C* and that in the 10 kg. of fatty tissue will be 0·85 *C*, giving a mean for the whole soft tissues (63 kg.) of 0·27 *C*. This level of radon in the tissues is maintained, and hence it will maintain its disintegration products in equilibrium with it. Taking the effective disintegration energy of the series as 20 MeV, and assuming an atmospheric radon content of 3×10^{-13} c/l (as above) the energy deposition per g. of tissue corresponds to a dose-rate of only 3×10^{-5} rad/year, mainly of alpha radiation. Using an R.B.E. of 10 to enable this alpha dose-rate to be added to the beta and gamma dose-rates already calculated, the tissue dose-rate for the dissolved radon is 3×10^{-4} rem/year.

The dose-rate from the disintegration products formed in the atmosphere and subsequently inhaled can be calculated on an assessment of the fate of the products retained. If the products are insoluble in body fluids, little if any irradiation of the gonads could occur from inhalation; if soluble, a fraction of the retained products (the retained fraction in lungs and respiratory tract is 75 per cent, I.C.R.P., 1955) would be generally disseminated in the bloodstream. The calculation has been based on the assumption that 20 per cent of the inhaled disintegration products are effective in irradiating general body-tissues, and an exact formula has been used in calculating the equilibrium energy dissipation. The total dose-rate to soft tissues due to the inhalation of air containing 3×10^{-13} c/l of radon plus disintegration products is then 1·9 millirem per year.

The total dose-rate to soft tissues due to the inhalation of air containing 3×10^{-13} c/l of radon plus disintegration products might be expected, therefore, to be about 0·0022 rem/year. At the radon concentration of 3×10^{-12} c/l reported by Anderson *et al.*, (1954) for London air the dose-rate thus calculated would be 0·022 rem/year, i.e. a figure comparable with the cosmic ray background.

Total dose-rate to the gonads

Before summarising the total gonad-dose from all sources the effect of body-shielding on the local gamma ray dose should be considered. Measurements have now been made of this shielding factor by using a water-filled tin model. A thin-walled tube was fixed in the trunk so that a small geiger-counter could be positioned at the site of an ovary. The counter was placed

TABLE 3J

The screening factors for local gamma rays: horizontal, sitting and standing postures

Position of model	Screening factors for local gamma rays			
	Female	Mean	Male	Mean
Horizontal	0·52		0·67	
Sitting	0·58	0·56	0·70	0·70
Standing	0·59		0·72	

Mean factor for both sexes 0·63

93299 O—57—pt. 2——42

outside the trunk to assume representative positions for the testes. Measurements were made in a laboratory (site IIb in Table 1J) where the background was known to be steady. The cosmic ray response of the counter was allowed for by measurements made inside a cubicle shielded by a minimum thickness of 9 in. of steel. The screening factors for horizontal, sitting, and standing postures are given in Table 3J.

The correction for the cosmic ray component inside the building could only be estimated approximately, but if this were in error by as much as ±100 per cent it would produce errors in the screening factor of not more than −8 or +4 per cent. In the male, the factor varies with the position assumed but by not more than ±8 per cent. The average ratio for the two sexes should not be in error, therefore, by as much as ±10 per cent.

Summary of dose-rates to the gonads

Table 4J summarises dose-rates which may be regarded as typical for regions in this country and possibly elsewhere, where the local rock radioactivity is not specially high, and the buildings are of brick or concrete construction not incorporating specially radioactive materials such as granite or granite chips. In arriving at the dose-rates a gonad shielding factor of 0·63 has been assumed, and the local gamma ray dose is averaged for an assumed 8 hours* out-of-doors and 16 hours indoors.

TABLE 4J

Dose-rates to the gonads for a region of 'normal' ground radioactivity

Radiation source	Dose to gonads per year (rad)
External irradiation	
Cosmic rays (sea level)	0·028
Local gamma rays (Leeds, 78 millirad/year indoors ... 48 millirad/year out-of-doors)	0·043
Radon in air, 3×10^{-13}c/1	0·001
Internal irradiation	
Potassium 40	0·020
Carbon 14	0·001
Radon + disintegration products, 3×10^{-13}c/1	0·002
Total dose per year	0·095†
Dose to age 30 years	2·85†

† Includes allowance for the R.B.E. of the alpha radiation where present, and therefore also expresses the gonad-dose in rem.

Table 5J illustrates an attempt to assess the gonad-dose to populations in three different localities and in different types of building. Estimates are given for two radon-in-air concentrations but it is not known whether levels as high as 3×10^{-12} c/1 persist for long periods.

It should be noted that higher dose-rates and greater differences between localities could be obtained by taking the extreme values observed in some of the Stockholm and Aberdeen sites, but an attempt has been made to assess as far as possible the conditions affecting large numbers of people. Thus, because even agricultural workers spend probably 8 hours per day or more

* This is a maximum figure and is probably an over-estimate.

TABLE 5J

Assessment of the gonad-dose to populations in three different localities and in different types of building

Location	Type of building	Dose to gonads (rad per year)*	
		Radon 3×10^{-13} c/l	Radon 3×10^{-12} c/l
Leeds	Brick	0·095	0·125
Aberdeen	Granite	0·108	0·138
Stockholm	Wood houses ...	0·095	0·125
	Type 2 houses ...	0·109	0·139
	Type 3 houses ...	0·160	0·190

* Gonad shielding factor, 0·63; exposure, 8 hours out-of-doors and 16 hours indoors.

in a brick house, their gonad-dose is not very different from that calculated for a Leeds town-dweller. The body-shielding factor of 0·63 considerably reduces the difference otherwise apparent in situations of differing local radioactivity.

DOSAGE IN BONE

Consideration should be given to the problem of dosage in bone from natural sources, in order that the significance of the ingestion of bone-seeking radioactive isotopes may be properly assessed. The dose-rate to osteocytes has been estimated, therefore, by a summation of the dose-rate from sources external to the bone and that from radium deposited in the skeleton itself.

Dose from sources external to bone

The dose-rates to osteocytes from the sources considered above will not be very different from the tissue doses already calculated. The dose-rate from potassium 40 will be less, because the potassium content of the bone is only about one-quarter of that for the body as a whole. The dose-rate from inhaled atmospheric radon and its products may also be expected to be greatly reduced, because even if the radioactive content of an osteocyte from these sources were the same as that deduced for soft tissues, the alpha particles would leave only a small fraction of their energy in the osteocyte itself. The reduced dose-rate to osteocytes for the conditions assumed in Table 4J might be put at about 0·08 rem/year.

Dose from radium in bone

The most likely value for the radium content of the skeleton for a region not exceptionally high in radioactivity would appear to be the mean content of $1{\cdot}2 \times 10^{-10}$ g. measured by Hursh and Gates (1950) for subjects in Rochester, New York, U.S.A., where the radium content of drinking-water is given as $0{\cdot}6 \times 10^{-16}$ g./cc. If the skeletal radium is proportional to the radium level in drinking-water, the measured level of $1{\cdot}1 \times 10^{-15}$ g./cc for tap water in St. Ives (Gleuckauf and Jacobi, 1953) would imply a body radium content of $2{\cdot}2 \times 10^{-9}$ g. Swedish waters are known to have very much higher radium contents, implying body radium contents approaching 10^{-7} g., but measurements of body gamma ray emission by Sievert (1953) do not suggest radium contents of this order. In fact a cross-check in 1953 between Sievert's apparatus and one in Leeds indicated that measured activities of persons not occupationally exposed to radium salts were about the same in Stockholm

and Leeds. These measurements, however, do not exclude small skeletal-radium burdens of the order of 5×10^{-9} g.*

A mean radium-burden of 10^{-10} g. has been taken as typical for a region not specially radioactive, and a mean burden of 10^{-9} g. of radium for an active region like St. Ives. Using methods similar to those given by the author elsewhere (Spiers, 1953) the mean dose-rates to osteocytes have been estimated as in Table 6J.

TABLE 6J

Estimation of the mean dose-rates to osteocytes

Conditions	Ra in skeleton (g.)	Dose-rate to osteocytes (rem/year)		
		Ra	External sources	Total
Not specially active region, e.g. Leeds 3×10^{-13} c/l radon in air ...	10^{-10}	0·037	0·08	0·12
Active region 3×10^{-12} c/l radon in air ...	10^{-9}	0·37	0·18	0·55

References

Anderson, W., Mayneord, W. V. and Turner, R. C. (1954). The radon content of the atmosphere. *Nature, Lond.*, **174**, 424.
Burch, P. R. J. (1954). Cosmic radiation: ionization intensity and specific ionization in air at sea level. *Proc. phys. Soc. Lond.*, A, **67**, 421.
Clay, J. and Clay, P. H. (1938). The absolute intensity of cosmic radiation at sea level. *Physica*, **5**, 898.
Gleuckauf, E. and Jacobi, R. B. (1953). Personal communication.
Hursh, J. B. and Gates, A. A. (1950). Body radium content of individuals with no known occupational exposure. *Nucleonics*, **7**, 46.
International Commission on Radiological Protection (1955). Recommendations. *Brit. J. Radiol. N.S.*, Suppl. No. 6.
Libby, W. F. (1955). Dosages from natural radioactivity and cosmic rays. *Science*, **122**, 57.
Peirson, D. H. (1951). Personal communication.
Peirson, D. H. and Franklin, E. (1951). Aerial prospecting for radioactive minerals. *Brit. J. appl. Phys.*, **2**, 281.
Shohl, A. T. (1939). *Mineral metabolism.* Reinhold Publishing Corpn., New York.
Sievert, R. M. (1953). Personal communication.
Sievert, R. M. (1955). *International Conference on the Peaceful Uses of Atomic Energy*, Geneva. Butterworth and Co., London. (In the press.)
Sievert, R. M. and Hultqvist, B. (1952). Variations in natural γ-radiation in Sweden. *Acta radiol., Stockh.*, **37**, 388.
Spiers, F. W. (1953). Alpha-ray dosage in bone containing radium. *Brit. J. Radiol. N.S.*, **26**, 296.
Spiers, F. W. and Griffith, H. D. (1956). Measurements of local gamma-ray background in Leeds and Aberdeen. *Brit. J. Radiol. N.S.*, **29**, 175.
Wood, E. E. and Willey, E. J. R. (1954). Personal communication.

F. W. SPIERS

* Recent work in America and in Germany has confirmed the value for the skeletal-radium burden as about 10^{-10}g., and suggests that burdens greater than 10^{-9}g. are unlikely.

APPENDIX K

The Genetically Significant Radiation Received from Diagnostic Radiology

The total number of X-ray examinations performed per annum

Information under this head relating to the National Health Service has been obtained from the Annual Reports of the Ministry of Health. Other information was supplied by the Services, other Government departments, and various bodies which undertake diagnostic examinations.

Hospitals operating under the National Health Service are responsible for by far the biggest proportion of the X-ray diagnostic work carried out in this country. Total figures for the number of X-ray examinations performed at these centres are available for the years 1951 and 1952; for the years 1953 and 1954 however the only information given is the number of 'units of work done'. Between 1951 and 1952 the number of examinations carried out under the National Health Service (Table 1K) increased by 13·2 per cent, and between 1953 and 1954 by 10·9 per cent. It has, therefore, been assumed that the mean of these figures, 12 per cent, would fairly represent the increases for 1952–53 and 1954–55. The number of examinations carried out in 1954 was estimated by applying to the number of 'units of work done' the ratio derived from the previous year's figures, i.e., that 1 examination equals 1·8566 'units of work done'. The figure for examinations performed in 1955, estimated on the assumption that the trend shown in the previous years continues, is, therefore, approximately 12,200,000.

TABLE 1K

The number of X-ray examinations per year carried out at National Health Service hospitals: 1951–55

Year	Number of 'units of work done'	Number of examinations
1951	—	7,738,389*
1952	—	8,756,643*
1953	18,214,310*	9,810,365†
1954	20,201,177*	10,880,506‡
1955	—	12,189,801†

*From the Annual Reports of the Ministry of Health.
†Obtained by adding 12 per cent to the figure for the previous year (see above).
‡Obtained by applying to the number of 'units of work done' the ratio derived from the previous year's figures, i.e. that 1 examination equals 1·8566 'units of work done'.

This total refers only to National Health Service hospitals. It has been assumed that hospitals outside the Health Service undertake 3 per cent of this number of examinations, i.e., 350,000, and that private medical practice accounts for a further 100,000 examinations. It is further assumed that the distribution between types of examination and sex and age of the patients examined is sufficiently similar to justify the final total of 12,650,000 being treated as a single group.

The gonad dose per examination

The values used in the calculations for radiation dose to the reproductive organs are listed in Table 2K. They are based almost entirely on the work of Stanford and Vance (1955). These workers made careful measurements on more than 1,500 patients, largely at one hospital. For males, the measuring instrument was placed close to the testes; for females, at a point on the skin over the ovaries. A subsidiary experiment on six cadavers gave the ratio of ovary-dose to surface-dose to be expected for the different kinds of X-ray examination; it did not, however, give any indication of the dose received by the reproductive organs of a foetus. Accordingly, where the site of examination is remote from the pelvis of a pregnant woman, the dose to the foetal gonads is taken to be the same as that to the mother's ovaries; where the child is in the direct beam, however, the dose has been estimated from the information given by Stanford and Vance. For salpingography and pelvimetry, the doses used are the lowest that have been published in this country. In the case of pelvimetry, it is assumed that three films are taken in each examination, although many hospitals take more. The dose for salpingography is as reported by Barnett and Bewley (1955) and that for the foetal gonads in pelvimetry by Stanford (1951).

It must be emphasised that the doses quoted in Table 2K are those produced by the techniques and methods of only one hospital; further, this hospital is one where particular care is taken to reduce the gonad doses to the minimum.

TABLE 2K

The dose of radiation received by the gonads in the course of X-ray diagnostic examination of various parts of the body

X-ray examination	Dose (mr) received by the gonads		
	Male	Female	Foetal
Head	0·8	0·2	0·2
Teeth	4·75	0·8	0·8
Shoulder	0·22	0·03	0·03
Arm, hand	0·26	0·05	0·05
Rib, sternum	0·48	0·16	0·16
Chest—large film	0·36	0·07	0·07
„ —mass miniature radiography ...	0·25	0·15	0·15
„ —special*	37	5·4	5·4
Barium swallow and meal	20	9	9
„ enema	40	20	20
Abdomen	69	200	580
Cholecystogram	1·8	15·6	15·6
Pyelogram	486	1,290	3,210
Bladder	279	690	2,610
Pelvis	1,100	210	800
Hip, femur	710	210	800
Leg, foot	3·5	0·6	0·6
Spine—cervical	1·74	0·18	0·18
„ —thoracic	22	15	15
„ —lumbar	129	713	713
Lumbosacral joint	22	220	1,540
Sacro-iliac joint	129	713	2,700
Salpingogram	—	1,700	—
Pelvimetry	—	1,280	2,680

* An average value for bronchography, tomography, etc.

TABLE 3K

The genetically significant radiation resulting from diagnostic radiology: England and Wales, 1955

Examination centres	Type of examination	Males: Examinations as per cent of total*	Males: Dose as per cent of total†	Females: Examinations as per cent of total*	Females: Dose as per cent of total†	Foetal gonads: Dose as per cent of total†
Hospitals ...	Head	3·9	0·1	3·4	n‡	n
	Teeth	0·2	n	0·3	n	n
	Shoulder	0·8	n	0·6	n	n
	Arm and hand	4·8	n	4·3	n	n
	Rib and sternum ...	0·4	n	0·1	n	n
	Chest—large film ...	11·8	0·1	12·5	n	n
	,, —special ...	0·4	0·2	0·8	n	n
	Barium swallow and meal	2·6	0·4	1·6	0·1	n
	,, enema	0·5	0·2	0·7	0·1	n
	Abdomen (including obstetric)	0·6	1·1	1·1	4·0	6·4
	Cholecystography ...	0·2	n	0·4	n	n
	Pyelography	0·7	3·0	0·6	10·7	2·3
	Bladder	0·1	0·1	0·1	0·4	0·4
	Pelvis	0·7	7·4	0·7	2·3	0·7
	Hip, femur	1·4	19·2	1·4	3·8	0·2
	Leg, foot	5·0	0·4	3·9	n	n
	Spine—cervical ...	0·4	n	0·7	n	n
	,, —thoracic ...	0·5	0·1	0·6	0·1	n
	,, —lumbar ...	1·4	2·6	1·4	10·8	0·7
	Salpingography ...	—	—	0·1	1·1	—
	Pelvimetry	—	—	0·1	3·0	15·6
	Total	36·4	34·9	35·4	36·4	26·3
	(Re-allocation of the foetal dose)§ ...		13·5		12·8	
	Total		48·4		49·2	
General Dental Service	All types	1·8	0·1	3·0	n	
Armed Services	,, ,, ...	3·4	2·2	0·1	0·1	
Mass Miniature Radiography	,, ,, ...	10·8	n	8·3	n	
National Coal Board	,, ,, ...	0·2	n	—	—	
Others	,, ,, ...	0·5	n	0·1	n	
	Total	53·1	50·7	46·9	49·3	

* *i.e.* of the total of all X-ray diagnostic examinations in England and Wales, 1955.

† *i.e.* of the total of all genetically significant radiation received from X-ray diagnostic examinations in England and Wales, 1955.

‡ n = negligible (i.e. below 0·1 per cent).

§ Allocated between the sexes in the sex-ratio at birth, 1 : 1·059.

Classification of examinations by sex and age of patient and by type of examination

Information as detailed as this is not readily available in many hospitals. It has, however, been obtained for 2 London teaching hospitals, 2 general non-teaching hospitals in the Greater London area, and a children's hospital. In the few cases where appreciable discrepancies exist, weighting was in favour of the non-teaching hospitals. Information on the number of pelvimetry and obstetric abdomen examinations carried out was obtained from 9 hospitals, 5 of them outside London. Apart from these two types of examination, the number of unborn children exposed to radiation was calculated from the number of women of childbearing age examined.

Detailed results

The results of the calculations on the genetically significant radiation received from diagnostic radiology are given in Table 3K. The number of examinations has in each case been expressed as a percentage of 17,650,000, the estimated total number of X-ray examinations of all kinds performed in England and Wales in 1955. The doses received, weighted according to the age of the patients exposed, are expressed as percentages of the total genetically significant dose from diagnostic radiology in England and Wales in 1955.

For comparison, the genetically significant radiation dose from natural radiation has been similarly calculated. Dose rates of 0·10 rad per year for males, and 0·09 rad per year for females have been assumed, and weighted according to the ages of the population as a whole. The total radiation received from diagnostic radiology is found to be 22 per cent of that received from natural radiation by the whole population, and is in addition to it.

Strictly, these figures apply only to England and Wales, but there is no reason to think that the corresponding figures for Scotland and Northern Ireland will be greatly different. Each approximation in the calculation has, however, been estimated on the low side. According to the calculations based on the present sample, therefore, the value of 22 per cent should be regarded as a probable lower limit rather than as an estimate. A realistic estimate of the radiation contribution from diagnostic radiology might be considerably greater than this figure.

A more detailed presentation of the subject than is possible here has been prepared and is to be published shortly (Osborn and Smith, 1956).

References

Barnett, E. and Bewley, D. K. (1955). Ovarian radiation during hysterosalpingography. *J. Fac. Radiol., Lond.*, **6**, 186.

Osborn, S. B. and Smith, E. E. (1956). The genetically-significant radiation dose from X-ray diagnostic radiology in England and Wales: a preliminary survey. *Lancet.* (In the press.)

Stanford, R. W. (1951). Radiation doses in radiographic pelvimetry. *Brit. J. Radiol. N.S.*, **24**, 226.

Stanford, R. W. and Vance, J. (1955). The quantity of radiation received by the reproductive organs of patients during routine diagnostic X-ray examination. *Brit. J. Radiol. N.S.*, **28**, 266.

S. B. OSBORN

APPENDIX L

The Contribution of Occupational Exposure to the Genetically Significant Dose of Radiation

In order to calculate the amount of radiation contributed to the total gonad-dose by occupational exposure, it is necessary to know (*a*) the number of persons, distributed by sex and age, who are exposed to radiation in the course of their work, and (*b*) the average gonad-dose received per person exposed. Accurate information on these points is lacking at the present time, and the following estimate is based on the best available data.

An estimate of the average gonad-dose per worker can be made by analysing the data obtained by the radiation monitoring service operated at one time by the National Physical Laboratory and now by the Radiological Protection Service; this service measures the occupational exposure to radiation for workers in the fields of medicine, research and industry, excluding the Atomic Energy Authority. It is a voluntary service and its coverage is incomplete, since many organisations, particularly hospitals, carry out their own monitoring, and there are undoubtedly others where no monitoring is done. However, the average dose recorded by the N.P.L. Service is a low one, and all the available evidence suggests that the doses recorded by the self-monitoring organisations are of a similar order. In these circumstances it has been assumed that the data obtained by the N.P.L. Service are applicable to all workers, except dentists in private practice; analysis of these data gives the figure of approximately 50 mr per week, or about 2·5 r per year, as the average gonad-dose for both males and females in all occupations.

Practically no information is available about private dentists and their assistants, but it is clear that, taking into account the number and type of examinations involved, their exposure risk is very much less than that of radiologists and radiographers; given reasonable care, the average gonad-dose they receive will be considerably lower than the figure of 2·5 r per year just quoted.

Besides those persons actually working with irradiating equipment, there are large groups of people, for example nurses in hospitals, who are exposed to some extent as a result of their work-places being near a source of radiation. These people are not regarded as being radiation workers and are not monitored; their total number however, greatly exceeds that of the radiation workers, and although the average doses are very low, the aggregate dose is undoubtedly significant.

From the Annual Reports of the Ministry of Health, it would appear that there are at present about 9,000 persons, excluding private dentists, occupationally exposed in the medical field; and it is estimated from information supplied by the Factory Department of the Ministry of Labour and National Service that there are about 5,000 persons occupationally exposed in the field of industry and research. This total of 14,000 persons is divided roughly equally between the sexes, and these are the people to whom the figure of 2·5 r per year is considered to apply. Thus, the gonad-dose received per year by this group as a result of occupational exposure, is 17,500 r to each sex. In order to allow for the group of workers exposed at a low level mentioned

above, it is considered advisable to increase this aggregate dose by about 50 per cent, i.e. to 25,000 r per year, for each sex; and a further 1,000 r should be added for each sex to cover the doses likely to be received by those private dentists who possess X-ray equipment, and by their assistants. The final total, which, it will be appreciated, is a very rough estimate, would then be 26,000 r each for males and females.

There is, unfortunately, no precise information about the age-distribution of these groups, and the best assumption that can be made at the present time is that the females are all below the mean age of reproduction (for women, about 28 years) and that the males are evenly distributed between the ages of 18 and 60 (mean age of reproduction about 32 years).

The total number of females below the age of 28 in the United Kingdom is 10×10^6. Each of these women receives a gonad dose from natural radiation of about 0·1 r per year, so that the total dose-contribution from natural radiation to this part of the female population is 10^6 r per year. Hence, at the present time, the occupational dose adds about 2·6 per cent to the genetically significant dose of natural radiation received by the female population.

For males in the United Kingdom, the population is about 5×10^6 between the ages of 18 and 32, and about 9×10^6 between the ages of 32 and 60. Only the dose-contribution for workers aged up to 32 is effective, so that the occupational exposure of genetic importance for male workers is about 9,000 r per year. The total number of males in the United Kingdom below the age of 32 is about 12×10^6, so that this section of the population receives a total dose from natural radiation of $1{\cdot}2 \times 10^6$ r per year. Thus, the occupational dose constitutes an addition of about 0·75 per cent.

Accordingly, in the United Kingdom the occupational contribution to the genetic dose from all sources except the Atomic Energy Authority is 26,000 r per year for females and 9,000 r per year for males, i.e. 35,000 r per year in all. The dose of natural radiation received by females up to the age of 28 is about 10^6 r per year, and by males up to the age of 32, about $1{\cdot}2 \times 10^6$ r per year, making a total of $2{\cdot}2 \times 10^6$ r per year. Hence, on the basis of the assumptions made, the occupational dose adds about 1·6 per cent to the genetic dose received by the population from natural radiation. It is to be noted that the contribution from the Atomic Energy Authority, reported by Farmer (1956), is 0·09 per cent.

I should like to thank Dr. G. H. Aston of the National Physical Laboratory and Mr. K. L. Goodall of the Factory Department, Ministry of Labour and National Service, who supplied some of the information on which the above estimates are based.

Reference

Farmer, F. R. (1956). Personal communication.

E. E. Smith

APPENDIX M

The Long-range Fall-out from Nuclear Test Explosions

This appendix summarises the data available on the radiation dose to human beings resulting from the fall-out from distant nuclear explosions. Continuous measurements have been made since 1951 by the Atomic Energy Research Establishment, Harwell, of the deposition on the ground of fission products from distant nuclear explosions. Measurements have also been made of the activity in the air at ground level in the United Kingdom, and on several occasions the variation of activity with height has been explored in the atmosphere up to 50,000 ft. (Stewart, Crooks and Fisher, 1955).

Diffusion and deposition of dust clouds from nuclear explosions

The dust cloud from a weapon in the 'nominal' size-range generally remains within the troposphere, reaching a maximum height of some 25–40,000 ft. when exploded in the middle latitudes. This part of the atmosphere is comparatively turbulent, and as the cloud travels downwind it diffuses both laterally and vertically and contaminates the lower atmosphere across a broad front at great distances. The dust is ultimately removed from the atmosphere by washout in rain-water and by direct deposition on to surfaces. Measurements made in the United Kingdom have shown that the former is the more important process and that the latter can be neglected in comparison; this conclusion however is not necessarily true close to the test site, where gravitational deposition may be an important factor. The United Kingdom measurements have also shown that, on the average, one half of the fine dust from the smaller type of nuclear explosion is removed from the atmosphere by rain-water in a period of 22 days. Deposition of the dust cloud from such explosions is therefore effectively complete within 3 months of the time of burst.

The behaviour of the clouds from explosions in the megaton class is markedly different. These clouds penetrate into the stratosphere, and may reach heights of the order of 100,000 ft. Diffusion is a very slow process in the stratosphere, and material returns to the lowest layers of the atmosphere at a much slower rate than in the case of the smaller type of explosion; a significant fraction of the dust generated may remain in the stratosphere for years after the weapon is exploded. Systematic surveys of the radioactive content of the air at various altitudes, taken in conjunction with fall-out measurements made at the same time, have shown that the dust from these tests was being deposited at a rate of between 10 and 20 per cent per year.

Measurement of airborne dust at ground level

The concentration of radioactive dust in the atmosphere has been measured routinely by drawing measured volumes of air through filters and counting the resultant beta activity on a suitable geiger-counter. Ground-level measurements made at Harwell since 1951 have shown that the activity present in air is of less biological significance than that deposited on the ground. The average level of activity from nuclear explosions has been found to be less than 1 per cent of that due to natural radioactivity in the air, and the maximum level has never exceeded that due to natural radioactivity. The measurements also show that over the past 7 years an individual in the United Kingdom might, on the average, have inhaled a total of $3{\cdot}4 \times 10^4$ dpm of fission products, including $8{\cdot}7 \times 10^3$ dpm of fresh fission-products

(measured at an age of 10 days) and including the particles of relatively high individual activity (Heard, 1956). The remaining $2{\cdot}5 \times 10^4$ dpm consisted of particles of low activity, owing to their low specific-activity or prolonged radioactive decay whilst airborne. For comparison, the continuous occupational permissible breathing-level for strontium 89 dust is $1{\cdot}3 \times 10^6$ dpm *per day* over a working life. Table 1M summarises information on the particles which might have been inhaled, deduced from the detailed ' particle-size and activity ' analysis. Only particles in the high and medium specific-activity groups are considered ; particles in the lower specific-activity groups are of less individual importance.

TABLE 1M

Estimate of the probable number of radioactive particles in the high and medium specific-activity groups, inhaled by any given individual in the United Kingdom over the past seven years

Particle size (μ)	High specific-activity		Medium specific-activity	
	Probable number inhaled	Total activity per particle* (dpm)	Probable number inhaled	Total activity per particle* (dpm)
1–2	7	$5{\cdot}4 \times 10$		
2–3	1	$2{\cdot}5 \times 10^2$		
3–6	0·5	$1{\cdot}4 \times 10^3$	112	14
6–9	0·07	$6{\cdot}6 \times 10^3$	14	66
9–12	0·01	$1{\cdot}8 \times 10^4$	3	180

* Immediate half-life = 6 days.

The particles carry fission-product beta activity which decays approximately inversely with time, and the activities quoted in Table 1M are those at 10 days after burst. The only significant alpha activity in the particles is that of plutonium. The activity of a 10 μ-particle may vary from about 4 disintegrations *per day* to extremely low levels. The mean concentration of this alpha activity in the ground-level air over the period has been 2×10^{-17} μc per cc compared with the occupational maximum permissible limit of 2×10^{-12} μc per cc (International Commission on Radiological Protection) for breathing insoluble plutonium dust over an occupational lifetime.

Measurements of deposition

Measurements of the deposition of the activity have been carried out at Milford Haven and at Chilton, near Harwell, since 1951, and in New Zealand since February 1955. In the current system, rain-water falling on a 12×10 ft. polythene roof is passed through a cylindrical esparto-grass filter and is collected in a tank. The beta activity of the dried filter is measured by mounting it co-axially over a calibrated cylindrical geiger-counter. Samples of the filtered rain-water are evaporated and counted on the same counting system, so that a correction can be made for the solubility of the radioactive material. This measurement is made only periodically, since experience has shown that the solubility of material from any particular bomb-series does not vary significantly with age, and it is possible to use a mean figure.

The daily deposition records at Chilton and Milford Haven are generally similar, although they occasionally differ markedly in detail. All daily deposits

of surface activity greater than 5 mc per sq. mile observed between February, 1951, and December, 1955, have been arranged in groups, and the frequencies of occurrence of the various groups are given in Table 2M.

TABLE 2M

The range of values of all daily deposits of surface activity greater than 5 mc per sq. mile, observed at Chilton and Milford Haven between February, 1951 *and December,* 1955

Range of values of deposited activity (mc per sq. mile)	Frequency of occurrence	
	Chilton	Milford Haven
5– 25	83	76
26– 50	9	5
51–100	3	1
101–150	3	—
151–200	1	—
201–250	—	1

The highest daily deposit at the sites was 190 mc per sq. mile at Chilton and 240 mc per sq. mile at Milford Haven; these occurred about the same time in heavy rain, some 5 days after the explosion of a weapon in Nevada in the autumn of 1951. The highest deposition in a single day from a thermonuclear weapon test was 25 mc per sq. mile at Chilton and 100 mc per sq. mile at Milford Haven.

The dose from deposited radioactivity

The gamma ray dose to human beings from each individual deposition of fission products has been calculated. Little difficulty has been experienced in dealing with the fission products from individual test series in which all the explosions take place within a period of a few weeks, or in interpreting the data when a series of nominal-bomb tests takes place in an atmosphere previously contaminated from thermonuclear tests; the difference in the time-scales of the deposition processes and in the decay-rates of the samples can be used to separate the components. The major difficulty arises when the stratosphere is contaminated with the fission products from thermonuclear tests widely separated in time. If, as we believe, the fine dust in the stratosphere is deposited at a rate of only 10–20 per cent per year, then it can be shown that more than 90 per cent of the integrated dose per generation is due to the single isotope, caesium 137.

The gamma ray dose from each individual deposition of fission products has been calculated, initially for the idealised case of an individual standing on an infinite flat plane. A protection factor of 3 has then been introduced to take account of the material which is washed into drains or is otherwise removed from the topmost layer of the earth's surface; this factor is believed to be conservative, since about one-half of the material already deposited has been found to be soluble in water. An additional factor of 7 has been allowed for the shielding provided by buildings* against the gamma rays from fission products; this figure has been derived from measurements carried out

* It is perhaps noteworthy that the reduction in dose from this cause is much less than the enhanced dose received from the gamma radiation from the natural radioactivity of the building materials (see Appendix J).

at the Atomic Energy Research Establishment with the gamma rays from cobalt 60, and is based on the assumption that the average individual spends 2½ hours daily out-of-doors.

The individual doses have been summed, and the total external dose to be received by the average inhabitant of the United Kingdom due to material deposited on the ground from bombs exploded before 31st December, 1955, is estimated to be 1·7 mr. About 75 per cent of this dose is associated with material which has yet to be deposited; the dose due to bomb dust suspended in the air near ground level is negligible by comparison. It has also been estimated that if the various types of weapon continue to be fired at the present rates for an indefinite period, the ultimate dose per individual per generation of 30 years will be 26 mr; this level will be reached in approximately 100 years.

Very sensitive methods of measuring the radioactivity of the human body have been developed in recent years, and there has been some indication, on the records obtained in recent months, of the presence of a 0·6 MeV gamma ray, which is suspected to be due to the fission-product caesium 137 arising from the fall-out; it is not yet possible, however, to identify this radiation rigorously. The radiation has been observed qualitatively on body-monitoring records obtained at Harwell, and has been reported in some detail in progress reports from the Argonne National Laboratory in the United States. The highest body-activity detected so far in the United States is found to be 4×10^{-3} μc. This activity would, if maintained, produce a total-body irradiation of 0·6 millirad per year, or about 1/30 of the dose due to the naturally-occurring isotope potassium 40 in the body. The caesium activity in the body may be expected to fluctuate in step with the rate of fall-out, if the biological half-life is about 20 days as suggested by the International Commission on Radiological Protection, and if this activity in human bones arises from the direct contamination of herbage; in this respect the caesium differs markedly from the strontium 90, which is cumulative. From the quantities expected in the fall-out and from the chemical and metabolic properties of this isotope, the caesium 137 in the human body due to uptake from food and water is unlikely, on the basis of present information, materially to affect the dose, calculated above, from the fission products deposited on the ground.

The accumulation in the human body of strontium 90 *from fall-out*

The rate of deposition on the ground of strontium 90 in the fall-out has been measured by radiochemical analysis and, since the spring of 1954, has been found to be approximately 6 mc per sq. mile, per year. The total at 31st December, 1955, was 11 mc per sq. mile. From the measurements of activity in the upper air, it is anticipated that the total from bombs already fired will rise in about 10 years to a maximum of approximately 45 mc per sq. mile. Should the various types of explosion continue at the present rates, the accumulated deposition is likely to rise to an equilibrium value of about 500 mc per sq. mile in about 100 years' time.

Because of its similarity to calcium, strontium is found to follow this element in the human food-chain. Measurements have accordingly been made of the strontium 90 activity per g. of calcium in samples of vegetation and soil from a number of locations in the United Kingdom (Bryant and Chamberlain, 1956). Using refined radiochemical techniques and anti-coincidence counting procedures, the activity per g. of calcium has also been measured in the bones of yearling sheep, in samples of milk, and in human bones. The representative figure for the strontium 90 activity of vegetation in the autumn of 1955 was about 35 μμc per g. of calcium. The corresponding representative

figure for sheep bones was 14 μμc per g. of calcium but in a few mountain pasture areas, values up to ten times higher have been recorded.

The maximum activity found so far in the limited number of human bone-samples in the United Kingdom is 1·2 μμc per g. of calcium in the skeletons of one-year-old children. Adult bones show lower activities, ranging from 0·05 to 0·2 μμc per g. of calcium. These levels may be compared with the maximum permissible level for strontium 90 in the body for occupational workers, recommended by the International Commission on Radiological Protection, which corresponds to about 1,000 μμc per g. of calcium. The average radiation dose to the bone from a level of 1 μμc per g. of calcium would be about 3 millirem per year, and may be compared with the dose to the bone from the background level of radium in the average person in this country (10^{-10}c), which is about 37 millirem per year, and from natural gamma radiation, which contributes about a further 80 millirem to the bone (see Appendix J).

References

Bryant, F. J. and Chamberlain, A. C. (1956). Private communication.

Heard, M. J. (1956). Private communication.

Stewart, N. G., Crooks, R. N. and Fisher, E. M. R. (1955). *The radiological dose to persons in the United Kingdom due to debris from nuclear test explosions.* A.E.R.E. Report HP/R 1701.

W. G. Marley

APPENDIX N

An Attempt to Estimate the Hazard from the Ingestion of Strontium 90

During the last fifty years, information has been accumulating about the induction of cancer in man and various species of animals by ionizing radiation and radioactive substances. Much of the evidence is qualitative and we have still very little reliable quantitative information on which to assess hazards.

The main risk from the absorption of bone-seeking isotopes is the delayed production of changes in bone, sometimes followed by osteogenic sarcoma, as well as blood changes consequent upon effects in the bone marrow.

The best human evidence is from the clinical investigation of patients to whom radium salts were administered for therapeutic purposes some twenty-five to thirty years ago, supplemented by earlier studies on industrial workers including those who ingested radioactive luminous paint. One of the most characteristic features of the production of malignant tumours of all kinds by ionizing radiations and radioactive materials is the very long latent period, often in the region of twenty years.

From a consideration of all the available evidence, the maximum permissible level of radium permanently incorporated into the skeleton has been fixed by the International Commission on Radiological Protection for persons occupationally exposed as 0·1 microcurie. From a comparison of the toxic effects of strontium 90 as compared to radium in animal experiments, the corresponding maximum permissible level for strontium 90 has been fixed at 1·0 microcurie. It must be appreciated that the biological effects of the radium and its breakdown products are probably largely due to alpha particles, whereas strontium 90 with its associated yttrium 90 emits only beta radiation. These differences in the energies and nature of the radiations, as well as differences in the patterns of distribution of the radioactive materials in the bone itself, probably cause the differences in biological effects. Strontium behaves chemically very similarly to calcium in bone formation and it is therefore natural to associate the two elements. The maximum permissible level of strontium 90 in the human skeleton corresponds to 1,000 micro-microcuries of strontium 90 per gramme of calcium in the skeleton, and it is this concentration which determines the dose level.

However, this maximum permissible level has been fixed for a group of adults educated in relation to the risks, under medical supervision, and working under carefully controlled conditions.

It is well-known that growing bone takes up more of the bone-seeking isotopes such as strontium 90, and concentrates them in the rapidly growing portions of the bone. It is also well known that rapidly growing tissues, such as those of children, are often particularly radiosensitive. We must also conclude from the available evidence that the damage produced by radioactive materials in bone is an integrated effect over the whole time and dose of the radiation. Since the radioactive half-life of strontium 90 is long (28 years), any material incorporated during childhood has a longer time to act than material taken up in later life. The danger is a little mitigated by the fact that radioactive strontium may not be retained in bone for as

long as radium. The biologically effective half-life of strontium 90 together with its daughter product yttrium 90 has been estimated by the International Commission on Radiological Protection as 2,700 days.

Another factor which suggests caution is that it is well-known that the irradiation of tissues in inflammatory conditions is more likely to induce tumour formation than the irradiation of normal tissues, and such conditions are more likely to occur in the whole population than in the specially selected occupational group.

It is difficult to fix precisely the lowest level at which tumour formation and other effects have occurred, owing to the fact that in many instances the material ingested by the worker or patient has been an unknown mixture of mesothorium and radium, and this uncertainty complicates the estimation of dose since the rates of decay of these substances are very different (mesothorium 6·7 years half-life and radium 1600 years).

Examination of the results of ingestion of radium by humans makes it clear that in the fixing of the maximum permissible level there is no great safety factor involved. A suggestive destructive lesion (in the dentine of the teeth) has been observed in a patient who carried a body burden of approximately 0·15 microcurie of 'radium' (probably a mixture of radium and mesothorium). Of 44 patients investigated by Looney *et al.*, 36 had body burdens of 0·4 microcurie or more; clinically recognisable, but not malignant, bone lesions were observed in 32 of these. The lowest level of pure radium producing a bone sarcoma in this series was 3·6 microcurie. Sarcomata were seen in five other patients included in this study, but these patients may well have ingested a mixture of radium and mesothorium. Among these five, the lowest level associated with the production of a tumour was 0·52 microcurie of 'radium' (mixture).

If we assume, in accordance with the agreed international recommendations, that 1 microcurie of strontium 90 carries the same risk as 0·1 microcurie of radium, then with all these considerations in mind, it would be unwise to fix the maximum allowable concentration of radioactive strontium in the bones of the general population, with its proportion of young children, at more than one tenth of the level agreed for occupationally exposed persons. That is, the maximum allowable concentration should not be more than 100 micro-microcuries of strontium 90 per gramme of calcium.

As we consider the possible effects which might be produced at lower dosage levels (that is, below one tenth of the maximum permissible level) our ignorance increases still further and we can only rely upon extrapolation from limited animal experience. It is still not possible to give a certain quantitative answer to the question of the relationship between dose and the frequency of bone changes, induction of tumours, and other effects of the radiation. At very low dose levels the incidence is so small in animals that the existence of a threshold below which no effect occurs has been postulated.

It appears however that each unit quantity of radiostrontium absorbed by the bone confers a certain probability of bone-tumour formation, the tumour development time perhaps decreasing and the tumour incidence increasing with the dose. On the whole, the experiments seem in favour of a proportionality between the frequency of tumours produced in a given length of time and the amount of radioactive material in the body even at low dose levels.

If we again assume, in accordance with the agreed international recommendations, that one microcurie of strontium 90 carries the same risk as

0·1 microcurie of radium, and attempt in this way to estimate the effects at one-hundredth of the maximum permissible level, we provisionally conclude that the effects are unlikely to be detectable. Nevertheless, if the concentration in human bones showed signs of rising greatly beyond one-hundredth of that corresponding to the maximum permissible occupational level it would indicate the need for immediate consideration of the problem.

Bibliography

Brues, A. M., Lisco, H. and Finkel, M. P. (1947). Carcinogenic action of some substances which may be a problem in certain future industries. *Cancer Res.*, **7**, 48.

Brues, A. M. (1949). Biological hazards and toxicity of radioactive isotopes. *J. clin. Invest.*, **28**, 1286.

Brues, A. M. (1951). *Carcinogenic effects of radiation. Advances in biological and medical physics.* Vol. 2, p. 171. Academic Press Inc., New York.

Cahan, W. G., Woodward, H. Q., Higinbotham, N. L., Stewart, F. W. and Coley, B. L. (1948). Sarcoma arising in irradiated bone; report of 11 cases. *Cancer, N.Y.*, **1**, 3.

Finkel, M. P., Lisco, H. and Brues, A. M. (1955). Toxicity of Sr89 in mice. Malignant bone tumors. Argonne National Laboratory Report No. 5378, p. 106.

Furth, J. and Lorenz, E. (1954). *Carcinogenesis by ionizing radiations.* Radiation Biology, Vol. 1, Part II, p. 1145. McGraw-Hill, New York.

Hoecker, F. E. and Roofe, P. C. (1951). Studies of radium in human bone. *Radiology*, **56**, 89.

International Commission on Radiological Protection (1955). Recommendations. *Brit. J. Radiol. N.S.*, Suppl. No. 6.

Looney, W. B. (1955). *Late effects* (25 *to* 40 *years*) *of the early medical and industrial use of radioactive materials.* Ann. intern. Med., **42**, 378.

Looney, W. B., Hasterlik, R. J., Brues, A. M. and Skirmont, E. (1955). A clinical investigation of the chronic effects of radium salts administered therapeutically. *Amer. J. Roentgenol.*, **73**, 1006.

Spiers, F. W. (1953). Alpha-ray dosage in bone containing radium. *Brit. J. Radiol. N.S.*, **26**, 296.

W. V. MAYNEORD
J. S. MITCHELL

Atomic Scientists Association

Limited by guarantee

STATEMENT ON STRONTIUM HAZARDS

In view of the widespread concern about radiation hazards, the Council of the Atomic Scientists' Association decided to appoint a committee to study the whole problem, and to follow up the implications of last year's report of the Medical Research Council in the light of more recent information. There is particular concern at the present time about the effect of the radioactive strontium produced in H-bomb tests. The Council has, therefore, requested the committee to prepare a public statement giving its current assessment of this hazard.

The report of this committee (whose membership is given below) is as follows:

When an H-bomb is exploded at a high altitude, and in an area in which there are no inhabitants—as is said to be the case with the forthcoming British tests—there is very little likelihood of immediate injury to people, or of substantial local contamination of the oceans, even if a large amount of radioactivity is produced in the explosion. This radioactivity would be taken up into the upper regions of the atmosphere from where it would spread all over the globe and gradually descend to the ground over a period of some years. By that time only the long-lived radioactive products of the bomb would remain, and it is with the possible effects of these that we are concerned.

If H-bomb tests continue at the present rate, the dose of radiation to the reproductive organs, which may cause damage to future generations, has been estimated in the M. R. C. report to be of the order of 1% of that resulting from the natural level of radiations. Of greater import, however, is the damage which may result to the present generation, mainly from one radioactive substance—strontium-90. This substance enters into our food, chiefly in vegetables and dairy products, and it accumulates in the human body in the bones where it remains for a long time. Depending on the assumptions made about the distribution of strontium in bone, we calculate (see appendix) that by the year 1970 the radiation dose to bone from all the tests carried out up to the autumn of 1956 will range from 9% to 45% of the dose received from all natural sources, including the radium which is normally present in bone.

It is known that radioactive substances concentrated in the bone may give rise to bone cancers and other damage, and that the irradiation of bone marrow may result in leukemia, a type of cancer of the blood. The induction of bone cancers by the action of strontium-90 in the bone has been demonstrated in animals; in human beings the same effect has been observed with radium, which in some ways behaves like strontium. In all these cases, however, the amounts of radioactivity present in the bone were far greater than those that are likely to accrue from H-bomb tests. The question then arises how to apply these findings to very small doses. There is here a fundamental difficulty, in that the relationship between the damage produced and the amount of radiation is not known. If this relationship is such that there exists a threshold dose below which cancer cannot be induced, then it can reasonably be inferred that the small amount of strontium-90 which will accumulate in bone from the current H-bomb tests would not result in any harm. If, however, the number of additional bone tumours resulting from radiation is directly proportional to the dose, then even a very small dose will give rise to a small but definite probability of bone cancer. This means that in a very large population a certain number of people would contract this disease as the result of their having a small amount of strontium-90 in their bones.

The evidence is as yet inconclusive. Some animal experiments have been interpreted as indicating the existence of a threshold dose. On the other hand, in man, the occurrence of leukaemia caused by radiation suggests a simple proportional relationship. Unfortunately the question cannot be settled by experiment in a short period of time, nor is there strong guidance from theory. There is one theory of the origin of cancer (that it is due to somatic point mutation) which

implies a proportional relationship. Where the effects of strontium-90 are concerned the authorities in the M. R. C. report appear to be inclined towards the proportional hypothesis, for they state "On the whole the experiments seem in favour of a proportionality between the frequency of tumours produced in a given length of time and the amount of radioactive material in the body even at low dose levels."

If the proportional relationship is accepted, it is then possible to make a rough estimate of the number of bone cancers which may result from a given H–bomb test. The calculations given in the appendix show that an H-bomb of the type tested at Bikini in 1954, if exploded high in the atmosphere, may eventually produce bone cancers in 1,000 people for every million tons of T. N. T. of equivalent explosive power. (It has been stated that bombs hitherto exploded were equivalent, in aggregate, to 50 million tons, insofar as their strontium-90 fall-out is concerned.) These thousand casualties would be spread all over the world and occur in the course of several decades. A somewhat larger number of people might suffer other bone changes, possibly without manifesting any clinical symptoms. There is also the probability of a number of cases of leukaemia resulting, but we have not enough data to estimate this number. At the same time it should be pointed out that these casualties, although large in absolute number, represent only a fraction of those due to the natural level of radioactivity; there would be no way of distinguishing one from the other.

If other types of nuclear weapons were exploded, the number of casualties would vary in direct relation to the amount of fission products released into the upper atmosphere.

In giving these estimates it must be emphasized again that, apart from the considerable margin of error due to lack of adequate data, they are based on the as yet unproved hypothesis of a proportional relationship applying to very small doses. From this point of view they represent the most pessimistic approach. On the other hand, if this hypothesis is correct, then the figures may be an underestimate of the damage since they do not allow for the radiation dose in children before or after birth. Children are known to take up much larger quantities of strontium than adults and the likelihood of producing radiation damage in them is probably much greater for the same amount of radiation.

H. S. W. MASSEY, F. R. S.,
President.

H. R. ALLAN,
Honorary General Secretary.

Membership of the Radiation Hazards Committee:
- Professor J. Rotblat, D. Sc., F. Inst. P., Chairman
- J. W. Boag, D. Sc., F. Inst. P.
- Professor A. Haddow, M. D., D. Sc.
- W. M. Levitt, M. D., F. R. C. P., F. F. R., D. M. R. E.
- Patricia J. Lindop, B. Sc., M. B., B. S., M. R. C. P.
- S. B. Osborn, B. Sc., F. Inst. P.
- Professor L. S. Penrose, M. D., F. R. S.
- Professor P. A. Sheppard, B. Sc., F. Inst. P.
- G. Simon, M. D., F. F. R., D. M. R. E.
- A. J. Salmon, Ph. D., A. Inst. P., A. M. I. E. E.

APPENDIX

Estimate of strontium-90 hazard based on an assumed linear relationship between bone tumour formation and dose

1. The measurements of the ^{90}Sr content in human bone in this country, carried out by workers of the U. K. A. E. A.[1] show that by the beginning of 1956 the average amount in all age groups from 10 years upwards, was about 0.2 S. U. (1 S. U.$=10^{-12}$ curies of ^{90}Sr per gramme of calcium).

2. If this figure is applied to the data of the world survey of strontium-90 made by the Lamont group,[2] we conclude that the ^{90}Sr concentration resulting from all the tests carried out up to the autumn of 1956 will in 1970 amount to about 4 S. U. This value is in agreement with the estimate of 4–10 S. U. made by Libby[3] for the U. S. A.

---

[1] R. J. Bryant, A. C. Chamberlain, A. Morgan, G. S. Spicer. A. E. R. E. HP/R 2056, 1956.
[2] J. L. Kulp, W. R. Eckelmann, A. R. Schulert. Science, 125, 219, 1957.
[3] W. F. Libby. Proc. Nat. Acad. Sci., 42, 945, 1956.

3. The total fission yield from the tests carried out up to the autumn of 1956 is estimated[2] to be equivalent to 50 megatons. For one nominal high-yield weapon (as defined in the Indian Government's document:[4] 20 megatons with high fission yield) exploded high in the atmosphere, one may therefore expect a concentration in bone of 1.6 S. U.

4. From Hasterlik's survey[5] of bone sarcoma in people with a radium burden, we estimate that 0.1 microcurie of radium yields about 0.5% probability of bone sarcoma. (A factor of 2 has been allowed for the possible admixture of mesotherium with the radium.)

5. Since 0.1 microcurie of radium is accepted[6] to be equivalent to 1,000 S. U., applying this probability value to the population of the whole world (2.5×10^9 people) we obtain that the number of bone sarcomas from one nominal high-yield nuclear test may amount to 20,000 all over the world ($2.5\times10^9\times0.5\times10^{-2}\times1.6\times10^{-3}$).

6. The radium normally contained in the body and the external radiation from natural sources deliver to the bone a dose equivalent to 45 S. U., or 9 S. U. if one allows for the non-uniform concenaration of strontium in bone (Spiers[7]). This means that the dose rate from strontium in bone will amount to 4/45 or 1/9 of that due to the natural background.

[British Journal of Radiology, v. 29, August 1956 : 409–417]

Radioactivity in Man and His Environment

Presidential address[1] by F. W. Spiers, D. Sc., Department of Medical Physics, The University of Leeds

A tradition, upheld almost without exception in the long history of the Röntgen Society and the British Institute of Radiology, is the delivery of an address to the Institute by the President during his term of office.

I am very conscious of the honour that falls to me to address you as President and very much aware of the responsibility of following my distinguished predecessors in the fulfilment of this obligation. In recent years, by a change of Articles of Association, the Institute has re-emphasised a characteristic that has always been implied by the nature of its membership—a wide interest in all that pertains to the science of radiation and radioactivity. Many presidential addresses given in this house bear witness to the activities and interest which spring from the Institute's constitution and to the bonds of cooperation and of service to radiology which exist between its medical and non-medical members.

It seemed appropriate, therefore, that I should speak on this occasion about radioactivity in man and his environment and give thereby a practical example of the width of interests encompassed by the Institute. Moreover, the subject I have chosen is of interest and importance to all who use ionizing radiation and is one which, although having significance in the problems of today, has also the historic interest of belonging to the scientific discoveries of the early years of this century.

The experiments of Elster and Geitel and of C. T. R. Wilson in the year 1900 first revealed the presence of a residual ionization in an electroscope and subsequent work by many investigators in the next few years showed that this ionization arose from radioactivity in the materials of which the electroscope was made, from terrestrial sources outside the electroscope and from penetrating radiation of cosmic origin. It was soon established that traces of the known radioactive elements were widely dispersed throughout the lithosphere and that few raw substances or even few metals were free of radioactivity in trace amounts. Man, it appeared, lived in a radioactive world and eventually it was to be found that, like his surroundings, man himself was also radioactive. It is certain that man has been exposed to ionizing radiations throughout his occupancy of this planet and it seems at least likely that in geologically recent times, no very great changes have occurred in the radiation intensity of his environment.

---

[4] Nuclear Explosions, Government of India, 1956.
[5] R. J. Hasterlik. Geneva Conference, Vol. II, p. 149.
[6] Recommendations of Int. Comm. Rad. Protect. B. J. R. Suppl. No. 6, 1955.
[7] F. W. Spiers. Brit. J. Radiol., 29, 409, 1956.
[1] Delivered at the British Institute of Radiology on May 24, 1956.

During this century, however, the situation has undoubtedly changed, for man now produces ionizing radiations and artificial radioactivities in abundance and turns them to his own great technical advantage. It is possible, perhaps likely, that the increase now taking place in man's radiation background will be greater than the other possible changes which have so far occurred, when for example ancestral man left his arboreal home for the cave, or later even, the Englishman entered his castle! It would seem fitting to put on record in this Institute an estimate of man's natural radiation dose, a quantity which members, by their professional activities, are bound in some measure to augment.

The radiation dose received naturally by tissues of the human body arises in two days: from external sources which include terrestrial radioactivity and cosmic rays, and from internal radioactivity acquired by the body from air, food, and water. The biological significance of this background radiation dose is properly a consideration for the biologist, but any physical analysis should be oriented to give the data required for a biological assessment. Because the background dose is in any case small compared with doses known to cause immediate somatic change, long-term genetic and carcinogenic actions may be presumed to be the likely considerations. The radioactivity data will be used, therefore, to derive the dose to human gonads, and the dose to osteocytes in bone. The dose to soft tissues in the body will not differ greatlyy from that of the gonads, with the exception of lung tissue where recent work by Hultqvist (1956) will be used to indicate the possible lung dose from the inhalation of radon, thoron, and their decay products. Because α rays and β rays are so easily absorbed, even by the elements of low atomic number comprising soft tissues, the dose to the gonads from external sources may be considered to be due entirely to cosmic rays and γ rays. The dose from internally acquired radioactivity, however, arises to a large extent from β rays and, when present, from α rays.

RADIOACTIVITY IN MAN'S ENVIRONMENT

1. Surface rocks and oceans

The radio-elements which contribute significantly to the terrestrial γ radiation are the members of the U and Th series together with the naturally occurring radioactive isotope of potassium, ^{40}K. The actinium series, ^{87}Rb, ^{147}Sm, ^{14}C and a number of radioactive isotopes, found in recent years to occur naturally, do not contribute significantly to the γ-ray background, either because of the character of their energy emission, or because they are insufficiently abundant. The radioactive constituents of some rocks listed in Table I show that for an "average" granite the γ-ray emission is contributed in roughly similar degree by the elements of the uranium and thorium series and by potassium 40. Sedimentary rocks are considerably less radioactive than granite with limestone least of those listed. The alum shale is given as an example of an exceptionally high rock radioactivity in Sweden, its excess activity being due entirely to its very high uranium content. Sea water has a low content both of uranium and potassium and furthermore exhibits a very low γ-ray equivalent radioactivity because, in deep water, the thorium isotopes, including ionium (^{230}Th), the precursor of ^{226}Ra, are almost completely removed by sedimentation following adsorption on ferric and manganic hydroxide while the uranium remains largely in solution (Rankama, 1954). The equivalent radium content of sea water is about 100 times lower, and river water some 10,000 times lower, than that of sedimentary rocks.

TABLE I.—*Radioactivity in rocks and water*[1]

Material	Radio-elements per g	γ-ray equivalent g Ra per g×10 [12]	
		Components	Total
Granite (average)	U: 4 μg Th: 13 μg K: 30 mg	1·44 2·25 2·40	6·1
Granite (alps)			12·4
Clays (Yorkshire)			4·4
Sandstone (Yorkshire)			2·0
Limestone			1·4
Brick			5
Alum shales (Sweden)	U: 170 μg Th: 1·5 μg K: 35 mg	60 0·3 2·8	63
Sea water	U: $1{\cdot}3\times10^{-9}$ g K: 0·35 mg	7×10^{-5} 28×10^{-3}	$2{\cdot}8\times10^{-2}$
Thames			$\sim10^{-4}$

[1] Data mainly from Libby (1955) and Hultqvist (1956).

TABLE II.—*Radioactivity in air*[1]

Situation	Radon content 10 [13]c/l	Thoron content 10 [13]c/l
Open air:		
London	1 to 3	
Rothamstead	0·3	
Innsbruck	4·3	
Over oceans	0·01	
In houses:		
Sweden:		
Wood	5·3	0·28
Brick	9·1	0·91
Shale-concrete	18·6	0·96
Britain:		
Laboratory	0·8	
Cellar	7·8	
Air-raid shelter	118	
Joachimstal mines	30, 000	

[1] Data mainly from Hultqvist (1956).

2. Air

The radon and thoron contents of air (Table II) depend on the escape of these gases from the ground and on the prevailing atmospheric conditions; stagnant and dusty air will retain the gases and their decay products, wind and rain will clear the air of radioactivity. The low value over oceans reflects the low concentration of the parent elements radium and thorium in sea water.

The value of 3×10^{13} c/l is often taken as representative of the radon content of air although values ten times higher have been reported by Anderson, Mayneord and Turner (1954) on some days in central London. The radon and thoron levels in houses depend very much on ventilation as well as on the building materials used.

3. Drinking water and foods

Table III lists values of the radium content in some drinking waters and foods according to some recent measurements by Hursh (1956) and Muth (1956). The radioactivity of tap water depends to a considerable extent on the purifying processes, being reduced by large factors where precipitation methods are used. High radioactivity is found in mineral waters and can occur in rural areas where water is taken from wells. Some values for the radium content of foods are given although no large survey of foods has so far been undertaken.

RADIOACTIVITY OF THE HUMAN BODY

Only in comparatively recent years has attention been given to the determination of the natural radioactivity of the body. Values of the total body radium content as high as 7.5 to 14×10^{9} g Ra, reported by Rajewsky (1941) and by Krebs (1942), have not been found in modern measurements of radium in cremation ashes. The recently obtained values given in Tables IV, varying from 0.05 to 0.32×10^{9} g Ra, show reasonable agreement between measurements made by different methods in different parts of the world. There is only a slight correlation of radium content and drinking-water activity and it seems likely that, as the data in Table III suggest, the major route for radium intake is through the radium content of food. The radium contents of soft tissues have been reported so far only by Muth (1956) and are included only tentatively, in order to assess the significance of the figure for the testis in a later calculation of the gonad dose.

TABLE III.—*Radioactivity in water and foods* [1]

Source	*Radium content 10^{16} g per g*
Drinking waters:	
London	<1
America:	
Cities	0·1 to 1·4
Rural areas	0·3 to 23
Germany:	
City	1·4 to 3·1
Mineral waters	130 to 240
Thermal springs	~1100
Foods:	
Milk	0·04 to 2·7
Cereals	10 to 39
Potatoes	67 to 125
Meat	80

[1] Data from Hursh (1956) and Muth (1956).

TABLE IV.—*Radioactivity of the body* [1]

Radio-element	Ra in tap water g/c.c. 10^{16}	Body radioactivity in in $c \times 10^{9}$
Potassium 40		13
Carbon 14		87
Radium (1)	0·01	0·047
(2)	0·36	0·12
(3)		0·14
(4)	1·4 to 3·1	0·32
(5)	34	0·24
Radium in bone		10^{14} gRa/g
Radium in soft tissues		1·4 to $4{\cdot}6 \times 10^{15}$
Radium in tests		$0{\cdot}6 \times 10^{15}$

[1] Data mainly from Hursh (1956) and Muth (1956).

The highest radioactivity in the body expressed as curies is that due to ^{14}C, the amount in Table IV being calculated from the measured specific activity of carbon of biological origin, given by Anderson and Libby (1951) as 15.3 dis min g C. The β-ray energy of ^{14}C is so low (54 kc V), however, that the dose rate to body tissues from ^{14}C is almost negligible compared with that from potassium and radium.

The potassium content of the body is now reasonably well established, at about 0.2 percent of body weight, by measurements of body γ-ray emission by Sievert (1955), Burch and Spiers (1953), and Rundo (1955). Males show higher values than females but the differences are related to the fat content of the body which contains no potassium. Anderson (1956) has shown that γ-ray measurements of total body potassium correlate well with lean body mass, as determined by measurements of body water by the tritium method.

THE EXTERNAL RADIATION BACKGROUND

1. Local γ-radiation

In most circumstances the local rock radioactivity is responsible for the major part of the external radiation background. Calculations of the dose-rate over different types of rock have been made by Libby (1955) and by Hultqvist (1956) and estimates, based on formulae given by the latter, are shown in Table V for the surface radioactivities listed in Table I. The dose-rates are of the same order as those given by Libby and accord with such direct observations as have so far been made.

The dose-rates inside houses and buildings are greater than the corresponding open-air values because the building materials are disposed to give a nearly isotropic irradiation. Some measurements reported by Sievert and Hultqvist (1952) and by Hultqvist (1956) in open-air and in houses in Sweden are summarized in Table VI, where the original values in ions per second have been corrected for the cosmic ray component and then converted to dose-rates in mrad/year. The values given are the means of observations in some 1500 apartments and houses; in each class of building dose-rates were found, exceptionally, up to twice the mean. Measurements made recently in Leeds and Aberdeen (Spiers and Griffith, 1956) are given in Table VII where the observations fall into two groups, those in granite buildings in Aberdeen and those in brick or concrete buildings in both cities. The dose-rates are lower than those reported by Sievert and Hultqvist but, with the exception of the Swedish alum-shale buildings, the difference for brick dwellings in the two countries are not very great. Open-air dose-rates in Leeds and over clay and in wooden houses in Sweden are about the same and only about half the dose-rates in brick dwellings.

TABLE V.—*Calculated dose-rates over flat ground*

Situation	*Local γ-ray dose-rate (mrad/year)*
Granite (average)	95
Granite (alps)	228
Clays (Yorkshire)	82
Sandstone (Yorkshire)	37
Limestone	26
Alum shales (Sweden)	1150
Sea	0. 5
Thames	0. 001

TABLE VI.—*Measured dose-rates in Swedish houses*

Building material	*γ-ray dose-rate at room centre (mrad/year)*
Wood	50
Brick	104
Concrete with alum-shale	171
Stockholm streets	85
Over igneous rocks	60 to 120
Over clay	50

2. Cosmic radiation

The dose-rate given by Libby (1955) for cosmic radiation is 35 mrad/year and appears to be based on an ionization intensity at sea level of approximately 2.5 ion pairs/c.c/sec. Recent work by Burch (1954) on the interpretation of high-pressure ionization chamber measurements supports the analysis given by Clay and his colleagues (1938) and leads to a lower value of the cosmic radiation ionization. The mean of three values given by these workers, corrected to ionization in free air, is 1.92 ions/c.c./sec. which converts to a dose-rate of 28 mrad/year at sea level and geomagnetic latitudes above 41° N.

This dose-rate applies to all bodily tissues. It may, however, be some 20 percent less in the basements of tall buildings which absorb the soft cosmic ray component and it will increase rapidly with altitude. At 10,000 and 15,000 feet the cosmic ray ionization is known to increase to about three and six times the sea-level rate; but because cosmic radiation is responsible for only a fraction of the total background dose-rate, the total tissue dose-rates are increased only by factors of the order of 1.6 and 2.3 respectively.

It is possible that greater significance attaches to the increase in the heavy particle component of cosmic radiation with altitude, thereby changing the relative biological efficiency (R. B. E.) of the radiation for some biological actions. Some mutations, for example, are reported as being less effectively produced by heavily ionizing particles than by X-rays; on the other hand, in a few cases the R. B. E. for heavy particles has been reported to be in the region of 5. At sea level, however, the contribution to the total ionization by slow particles and the effect of an associated increase in R. B. E. for this component would appear to be unimportant.

TABLE VII.—*Measured dose-rates from local radioactivity*

Type of building	Location	Local γ-ray dose-rate [1]
		mrad/year
1. All granite	(a) Aberdeen—laboratory	107
	(b) Aberdeen—in bell tower	99
	(c) Aberdeen—entrance hall	101
2. Brick or concrete	(a) Aberdeen, rooms on various floors	73
	(b) Leeds, room in hospital building	81
	(c) Leeds, single storey laboratory	80
	(d) Leeds, various rooms in house	77
3. Out-of-doors	Leeds, in garden of house 2(d)	48

[1] Mean values given for centres of rooms and at 1 m above floor.

3. Atmospheric radioactivity

Radiation from the decay products of radon in the air can add to the external γ-ray dose, although the effect may be expected to be small and very dependent upon atmospheric conditions which determine the degree of radioactive equilibrium. Peirson and Franklin (1951) have calculated that at ground level an atmospheric radon content of 3×10^{-13} c/l produces a γ-ray flux of the order of 10 quanta/cm^2/min. Taking a mean energy for Ra B and C γ rays, the tissue dose rate for this flux is 2.2 mrad/year. The formula used by Hultqvist (1956), which includes a factor for scattered quanta, gives a dose-rate, for the same radon level, of approximately 4 mrad/year and this figure will be used in subsequent calculations. Under most conditions this contribution to the background dose-rate is trivial but for the higher levels of atmospheric radon given in Table II (or for the higest value, 2 to 3×10^{-12} c/l., reported by Anderson, Mayneord and Turner, 1954, for London air), the γ-ray dose could be of the same order as that due to cosmis rays.

Calculations of dose-rates due to thoron in air lead to values of the same order of magnitude as those for similar levels of radon activity. The dose-rates for the thoron levels given in Table II are from 0.6 to 2 mrad/year. Apart from Hultqvist's data for Swedish houses, however, no information is available on the general level of thoron in air and no allowance for thoron has been included in the subsequent tabulation of dose-rates.

THE INTERNAL RADIATION BACKGROUND

1. Potassium.

The potassium content of the body provides the chief source of internal radiation to soft tissues. Taking the average tissue content of potassium to be 0.2 per cent and using Sawyer and Wiedenbeck's (1950) constants for the radioactivity of ^{40}K as 28.3 β particles per second per g K of mean energy 0.605 MeV and 3.6 γ quanta per second per g K of energy 1.46 MeV, the tissue dose can be calculated to be:

Tissue dose rate due to K=18 (β)+2 (γ)=20 mrad/year.

Because the mean range of the β particles of ^{40}K is only some 2 mm in tissue the dose-rate in a given organ is determined mainly by its own potassium content. With regard to the gonad dose, flamephotometer measurements on testes and ovaries taken from two post-mortem examinations gave a mean potassium content of 0.20±0.02 per cent. Although observations have been made so far on only four specimens, the results suggest that the mean content assumed in the ^{40}K calculation is sufficiently representative of gonad tissue.

2. Carbon 14

The specific activity of ^{14}C in carbon of biological origin is 15.3 β particles/min/g C (Anderson and Libby, 1951) with a mean β-ray energy of 0.15 MeV. Taking the carbon content of tissue as 18 per cent, the energy deposition due to ^{14}C amounts to a tissue dose-rate of only 1 mrad/year.

3. Radon and its disintegration products in air

An estimation of tissue dose arising from the inhalation of air containing radon can be made if, in the absence of complete information on all the factors concerned, some simplifying assumptions are made. The concentration of radon in the atmosphere is regarded as uniform and it is assumed that the breakdown products (RaA, RaB, RaC and RaC′) are in equilibrium with the radon and are uniformly suspended in the air, a condition holding probably only in still air. The calculation is then made in two parts: (1) for a steady level of radon (plus disintegration products) in body tissues *via* the blood in contact with the radon in alveolar air, and (2) for a steady intake into the lungs of the disintegration products formed in the external air.

The solubility of radon in water at 37° C. is 0.17 and in fat the figure is about five times higher. If the concentration of pure radon in alveolar air is R, the concentration of radon in the 50 kg of aqueous tissue will be 0.17R and that in the 10 kg of fatty tissue will be 0.85 R, giving a mean for the whole soft tissues of 0.27 R. This level of radon in the tissues is maintained and hence it will maintain its disintegration products in equilibrium with it. Taking the effective disintegration energy of the series Rn RaA . . . RaC′ as MeV and assuming an atmospheric radon content of 3×10^{-13} c/l, the energy deposition per gram of tissue corresponds to a dose rate of only 0.03 mrad/year, mainly from σ radiation. Using an R.B.E. of 10, to enable σ-ray dose-rate to be added to the β and γ-ray dose-rates already calculated, the mean tissue dose-rate for the dissolved radon is 0.3 mrad/year.

If the disintegration products are present in equilibrium with the radon content of the air, these are also swept into the lungs by the inspired air and, where they are deposited, build up an equilibrium level which is determined by the inspiration rate. Following the recommendations of the International Commission on Radiological Protection, the distribution of the inhaled products will be taken to be: 25 per cent exhaled, 50 per cent retained in the upper respiratory passages and subsequently swallowed and 25 per cent deposited in the lungs. Because of the short half-lives of the decay products RaA . . . RaC′ and their low transference from the gut to the bloodstream, the fraction of the products swallowed will contribute little to general body dosage. This fraction, however, would irradiate the areas in the upper respiratory tract where the deposition was initially localised. In the case of soluble compounds, all the fraction deposited in the lungs would be taken up by body fluids; in other cases perhaps half the lung deposition would be generally disseminated. It would probably be safe to assume that some 20 per cent of the inhaled disintegration products could give rise to the irradiation of the general body tissues.[2]

For the atmospheric radon content assumed and an inhalation rate of 20 m^3/day the dose-rate to soft tissues from inhaled decay products is 0.16 mrad/year or 1.6 mrem/year. The total dose-rate to the general soft tissues of the body from inhalation of air containing 3×10^{-13} c/l, of radon and disintegration products might amount, therefore, to about 2 mrem/year, a quantity which remains small compared with other dose-rates, but one which might become significant under conditions of high atmospheric radioactivity.

---

[2] It can be shown that for an influx of K c/sec. of the disintegration products RaA . . . RaC′, the energy dissipated per second at equilibrium will be:

$$\Sigma(E) = 3{\cdot}7 \times 10^{10} K \left\{ E_A\left(\frac{1}{\lambda_A}\right) + E_B\left(\frac{1}{\lambda_A} + \frac{1}{\lambda_B}\right) + E_C\left(\frac{1}{\lambda_A} + \frac{1}{\lambda_B} + \frac{1}{\lambda_C}\right) + E_{C'}\left(\frac{1}{\lambda_A} + \frac{1}{\lambda_B} + \frac{1}{\lambda_C} + \frac{1}{\lambda_{C'}}\right) \right\}$$

MeV/sec where E and λ are respectively the particle energy in MeV and the transformation constant in sec. $^{-1}$.

TOTAL DOSE-RATE TO GONADS

In summarising the total gonad dose from all sources it is necessary to allow for the shielding of the gonads by overlying body tissues. The shielding factors, which reduce the dose from the local γ radiation, have been determined by using a water-filled tin model in which measurements of the normal background intensity could be made at the sites of the ovary and testis. The screeing factors were derived from the measurements after appropriate corrections had been made to allow for the cosmic-ray contribution to the instrument response. In the female, the loyal γ radiation was found to be reduced by factors of 0.52 to 0.59 depending on the position adopted by the model. In the male the screening factors varied from 0.67 to 0.72 and a mean factor of 0.63 was derived as the average for both sexes. This factor, which is unlikely to be in error by as much as $\pm$ 10 per cent is applied to all estimations of gonad dose from local γ radiation.

Table VIII summarises the dose-rates which may be regarded as typical for regions of this country, and possibly elsewhere, where the local rock radioactivity is not specially high and the buildings are of brick or concrete not incorporating highly radioactive materials. An arbitrary division of the day has been made by assuming six hours of the 24 to be spent out-of-doors.[3] Possible small additions to the total dose-rate would be not more than 2–3 mrem/year for thoron in air and 1 mrem/year for the radium content of the testis suggested by Muth's (1956) data.

Table IX illustrates the range of dose-rates deduced for various situations in this country and in Sweden. Greater differences between localities could be obtained by taking the extreme values observed in some of the Swedish and other sites, but as far as possible an attempt has been made to assess the conditions affecting large numbers of people. Thus because even agricultural workers spend some eight hours in a brick house their radiation dose is not very different from that calculated for a town dweller in the same geological area. The body shielding factor considerably reduces the differences otherwise apparent in situations of differing local radioactivity.

TABLE VIII

Radiation source	*Dose to gonads per year*
External irradiation	
Cosmic rays (sea level)	28 mrad
Local γ rays (Leeds, 78 mrad year indoors, 48 mrad/year out-of-doors)	45
Radon in air, $3 \cdot 10^{13}$ c/l	1
Internal irradiation	
Potassium 40	20
Carbon 14	1
Radom dis. products, $3 \cdot 10^{13}$ c/l	2
Total dose per year	97 mrad [1]
Dose to age 30 years	2·91 rad [1]

[1] Includes allowance for the R. B. E. of the a radiation, where present, and therefore also expresses the gonad dose in rem.

TABLE IX.—*Gonad doses*

Location and buildings	*Dose rate mrad/year*
Leeds, brick:-	
(1) outdoor worker	90
(2) indoor worker	97
Aberdeen, granite	113
Stockholm:	
(1) wood	89
(2) brick	114
(3) concrete	146
At 15,000 ft. on granite:	
(1)	244
(2)	328

[3] The assumed time out-of-doors, which is used illustratively, is probably above average for city dwellers.

The last two entries illustrate the overall effect of the sixfold increase in cosmic-ray ionization at an altitude of 15,000 feet in a situation where the rock is granite, as for example in the Andes. Two granites have been chosen (as in Table I) and the resulting dose-rates are respectively 2·2 and 1·7 times the corresponding values over the same rock at sea level.

DOSE TO LUNGS

In addition to dosage from external sources of radiation and from ^{40}K, lung tissues will receive further irradiation from inhaled particulate matter carrying the decay products of radon and thorium. Some of this radioactivity will lodge in the upper respiratory tract and be removed in perhaps a matter of hours by ciliary action. In this time, however, irradiation will have occurred from the short-lived radon product RaA. Radioactivity on the finer dust particles ($< 1\ \mu$) will enter more deeply into the lung tissues *via* the alveoli and, unless rapidly dissolved, will produce a general irradiation of the lung tissues.

Chamberlain and Dyson (1955) and Shapiro and Bale (1953) have considered the problem of dose to the trachea and bronchi and have come to approximately the same conclusion as to the order of magnitude of the dose produced by a given atmospheric radon content. Chamberlain and Dyson have shown that for a radon concentration in air of 3×10^{-13} c/l., bronchial tissues to a depth of 45 μ (the range of the RaA α particles) receive an average dose-rate of 220 mrem/year. The total dose-rate from all sources would then be of the order of 315 mrem/year. The total dose-rate from all sources would then be of the order of 315 mrem/year to the superficial bronchial tissues, a rate about three times that calculated for the general soft tissues of the body.

If it is assumed that the 25 per cent of decay products reaching the alveoli are insoluble and remain in the lungs, calculations similar to those given in an earlier section show that the average lung dose would be about 160 mrem/year for an air activity of 3×10^{-13} c/l. A very comprehensive analysis of problem of lung dosage from both radon and thoron decay products has been given by Hultqvist (1956). The dose-rate to lung tissue due to a radon concentration of 3×10^{-13} c/l. can be deduced from Hultqvist's results as 150 mrem/year. The thoron concentrations given by Hultqvist (see Table II) are about ten times lower than the radon levels and, assuming a thoron concentration of 3×10^{-14} c/l., the resulting lung dose according to Hultqvist could be about 200 mrem/year. The total lung dose from all sources would then be about 245 mrem/year if the radon only is included and 445 mrem/year including radon and thoron. A high ventilation rate (3·5 changes of the air per hour) reduces the radon and thoron doses by a factor of nearly 4, in which case the lung dose from all sources would be reduced from 445 to 190 mrem/year. It appears, therefore, that the dose-rate to lung tissue may be from two to four times that received by other body tissues when the radon and thoron concentrations in air are respectively 3×10^{-13} c/l. and 3×10^{-14} c/l.

DOSAGE IN BONE

The bone tissues likely to receive the highest dose from natural sources are the osteocytes lying in regions of bone which incorporate radium. An average dose to an osteocyte of a given size can be deduced from the radium content of the body on the assumption of uniform radium deposition. In those cases of radium poisoning which have been studied by autoradiography (especially by α-particle track photographs) the radium occurs in localised areas where the dose-rate has been shown to be as much as 10 to 20 times the average. Nevertheless, the calculation of an average dose-rate will be a satisfactory measure of the general background irradiation of bone and will serve as a means of assessing the significance of the ingestion of bone-seeking radioactive isotopes.

The dose-rate to osteocytes from sources external to the bone will be practically the same as in the calculations for soft tissues, but the potassium contribution to the dose-rate will be less because its concentration in bone is about four times less than in muscle. The resulting dose-rate from sources other than radium can them be put at about 82 mrem/year (Table X).

TABLE X.—*Background dose-rates to bone*

Conditions	Radium in skeleton	Dose rates to osteocytes in mrem/year		
		Radium	External sources	Total
Not specially radioactive region Radon in air 3×10^{-13} c/l.	10^{10} g	39	82	121

The radium body burdens listed in Table IV suggest that for a region not specially radioactive a representative value for the radium content of the skeleton might be 10 ^{10}g Ra. Using methods of calculation similar to those given by Spiers (1953) the mean dose-rate to an osteocyte of diameter 5μ would then be 39 mrem/year, making a total from all sources of 121 mrem/year. The extent to which this might vary from site to site in bone connot be estimated with any confidence because the deposition of radium in the skeleton due to a low continuous intake has not so far been studied.

ACQUIRED RADIOACTIVITY TO PRODUCE THE EQUIVALENT OF THE BACKGROUND DOSE-RATES

At a time when the accidental ingestion of radioactive materials must be regarded as at least a possible accompaniment of nuclear energy development, it is of interest to consider what quantities of acquired radioactive elements will produce dose rates equal to those already being received by body tissues from natural sources. Two fission products would seem to be particularly important in that they are long lived and are capable of being taken into the body structure. Radioactive caesium, ^{137}Cs, may be expected to follow potassium in its behaviour and distribution and strontium 90 is known to be taken up in bone.

In the case of ^{137}Cs, which emits β rays of 0.55 MeV and 1.21 MEV energy and a γ ray of 0.66 MeV, a body burden of 0.6 μc is required to give the soft tissues of the body a dose-rate equal to the background rate of 100 mrem/year. This comparative figure is relevant to any discussion of the significance of an acquired ^{137}Cs activity.

In the case of ^{90}Sr with its radioactive decay product ^{90}Y, a body burden of 45×10^{-9} c would result in an average dose-rate to osteocytes equal to that received from natural sources including a radium burden of 10^{-10} g. If, however, it is conceded that the concentrations of strontium in bone can be five times higher than those observed with radium (I. C. R. P. 1953), the introduction of this further factor of 5 reduces the amount of ^{90}Sr+^{90}Y equivalent to the background radioactivity to 9×10^{-9} c. Assuming the calcium content of the skeleton to be 1000 g, the ^{90}Sr contents can be expressed respectively at 45×10^{-12} and 9×10^{-12} c of ^{90}Sr per g Ca.

DISCUSSION

The importance of an estimate of the dose-rate from natural radiation lies in the fact that it is the inevitable accompaniment to life and has been so throughout man's evolution. It must form one basic element of comparison in any judgment of what is a tolerable level of human dosage from the new radiation sources. It would seem that the natural dose-rate, if not an essential factor in man's environment must be at least a tolerable one. Furthermore, a comparison of the natural background dose-rate with dose-rates known to be harmful will give some indication of the range within which "permissible" rates must be set—although it is beyond the proper scope of this discussion to consider what those rates should be.

In relation to genetic effects, comparison must be made with the dose received by the gonads from background radiations. It seems likely that this dose-rate is not far from 0.1 rad per year or 3 rad in a generation time of 30 years, and that, even in houses incorporating highly radioactive shales in their structure, the dose level is not much more than 50 per cent greater than this. Although reservation is necessary, in view of the paucity of information, great variations in the background dose-rates to large populations seem rather unlikely, because local γ radiation provides only part of the background dose and its contribution to the gonad dose is reduced by the shielding of overlying tissues.

The relationship of a gonad dose of 3 r in a generation to that required to double the human spontaneous mutation rate is uncertain because the radiosensitivity of human genes is unknown. The most recent estimate that can be quoted is that of Muller (1955) which suggests that the so-called doubling dose is unlikely to be much more than ten times this background dose.

The dose-rate to the soft tissues in bone is only slightly in excess of that to the gonads because the contribution from the small natural radium content of the skeleton is partly off-set by a reduced contribution from potassium. A dose-rate of about 0.12 rad per year or about 6 rad in 50 years appears to be a reasonable estimate. This is some 2000 times less than dose-rate to osteocytes which have been estimated in cases where small accidental radium burdens (of about 0.4 μg) have ben associated with radiographically detectable but not necessarily malignant—changes in bone.

It has been shown that the dose rates from natural sources to the upper respiratory tract and to the general tissues of the lung may be as much as three times the gonad dose-rate for an assumed normal atmospheric radon content of 3×10^{-13} c/1. The dose in 50 years, 15 rad, is from 100 to 1,000 times less than the order of dose estimated in cases where the occurrence of cancer has been associated causally with the irradiation of body tissues. It is probably 10,000 times less than the lung dosage in the uranium miners of Joachimstal amongst whom the incidence of lung cancer was so marked.

It is evident, therefore, that the natural background dose stands nearest in relationship to those doses thought to be genetically significant. Several orders of magnitude lie between the natural dose and those associated with carcinogenesis. Clearly in any assessment of the significance of population dosage, these differences of magnitude must be recognized and taken into account.

REFERENCES

ANDERSON, E. C., Report on Conference on the Measurement of Body Radioactivity, Leeds 1956, in the press.

ANDERSON, E. C. and LIBBY, W. F., Phys. Rev., 1951, lxxxi, 64.

ANDERSON, W., MAYNEORD, W. V., and TURNER, R. C., Nature, 1954, clxxiv, 424.

BURCH, P. R. J., Proc. Phys. Soc. Lond. A., 1954, lxvii, 421.

BURCH, P. R. J., and SPIERS, F. W., Nature, 1953, clxxii, 519

CHAMBERLAIN, A. C. and DYSON, E. D., Brit. J. Radiol., 1956, xxix, 317.

CLAY, J. and CLAY, P. H., Physica, 1938, v, 898.

HULTQVIST, B., K. svenska Vetensk, Akad. Handl., 1956, vi, No. 3.

HURSH, J. B., Report of Conference on the Measurement of Body Radioactivity, Leeds, 1956, in the press.

International Commission on Radiological Protection, Brit. J. Radiol. Supplement No. 6.

KREBS, A. T., Strahlentherapie, 1942, lxxii, 164.

LIBBY, W. F., Science, 1955, cxxii, 57.

MULLER, H. J., Bull. Atomic Scientists, 1955, ix (9), 329.

MUTH, H., Report of Conference on the Measurement of Body Radioactivity, Leeds, 1956, in the press.

PEIRSON, D. H. and FRANKLIN, E., Brit. J. appl. Phys., 1951, ii, 281.

RAJEWSKY, B., Strahlentherapie, 1941, lxix, 438.

RANKAMA, K., Isotope Geology, 1954 (London, Pergamon Press Ltd.), 405.

RUNDO, J., J. Sci. Instrum., 1955, xxxii, 379.

SAWYER, G. A., and WIEDENBECK, M. L., Phys. Rev., 1950, 1xxix, 490.

SHAPIRO, J., and BALE, W. F., Univ. of Rochester Report U. R., 1954, 242.

SIEVERT, R. M., International Conference on the Peaceful Uses of Atomic Energy, Geneva (London, Butterworth and Co.), in the press.

SIEVERT, R. M., and HULTQVIST, B., Acta Radiol., 1952, xxxvii, 388.

SPIERS, F. W., Brit. J. Radiol., 1953, xxvi, 296.

SPIERS, F. W. and GRIFFITH, H. D., Brit. J. Radiol. 1956, xxix, 175.

[Reprinted from Nature magazine, April 13, 1957]

BIOLOGICAL EFFECTS OF RADIATION

An open meeting was held on March 7 at the Institute of Biology under the titl "Aspects of Radiotoxicity," with the aim of clarifying some of the problems arising from the increasing level of radiation. Dr. H. O. J. Collier, in the chair,

set the evening's challenging tone to the scientist by opening the discussion with the quotation, "Man, proud man, drest in a little brief authority, . . . plays such fantastic tricks before high heaven, as make the angels weep". This challenge was taken up by Prof. J. Rotblat, who spoke on the physical aspects of radiation damage, and by Prof. L. Penrose, a member of the Medical Research Council Committee (1956) for the study of radiation hazards, who discussed biological damage, including the genetic effects.

Prof. Rotblat began by stressing the complexity of the problem. On the physical aspect alone, and confining the discussion to ionizing radiations, there is a multitude of particles to consider, from electrons to fission fragments, with widely differing energies, penetrating powers and types of secondary reactions produced. Compared, however, with the chemical, biochemical and biological processes contributing to radiation damage, the nature of which is still obscure the physics of radiation damage is fairly well understood. The passage of radiations through matter gives rise to excitation and ionization of atoms and dissociation of molecules. All these contribute to the biological action and for this reason it is necessary to consider the total energy transferred to matter rather than ionization alone. The unit employed is the linear energy transfer (sometimes referred to as the L. E. T.), which is the amount of energy transferred per unit length of path of the ionizing particles, usually measured in keV. per micron of tissue. This does not depend on the mass of the ionizing particle but only on its charge and velocity. The very marked variation with velocity explains the observed difference between the damaging action of alpha particles, protons and electrons, of the same energy. But although different particles with the same value for the linear energy transfer produce the same biological effect, the relationship between the linear energy transfer and biological damage, or the relative biological effectiveness (R. B. E.), is not at all straight-forward. Prof. Rotblat quoted examples of biological processes, in some of which the biological damage increases with linear energy transfer, in others decreases with it, and in still others first increases and then decreases. These variations indicate the existence of different mechanisms of biological action.

Turning to the doses of ionizing radiations to which man is nowadays exposed, Prof. Rotblat made a survey of the natural background of radiation, pointing out that this will differ depending on whether one considers the gonad dose, the dose to bone or to other tissue. In Great Britain the gonad dose is 0·1 r. per year, or 3 r. in thirty years; the bone dose is slightly greater and amounts to 8·5 r. during seventy years of life. Of the man-made radiations, the largest contribution is from diagnostic radiological examination. The Medical Research Council report gave a figure of 22 per cent of the natural background, but more recent evidence indicates that the actual value may be 5–10 times higher, which would bring it in line with that in the United States. The fall-out from nuclear test explosions has up to now contributed very little so far as external radiations are concerned, but internal radiation gives rise to some concern. The effect of cæsium-137 in the body, although quite small, has perhaps been dismissed too lightly, but the concentration of strontium-90 in bone is the more worrying factor.

All these doses appear quite safe when compared with the maximum permissible dose (M. P. D.) established by the International Commission on Radiological Protection, which amounts to 0·3 r. per week, or 150 times the natural background. It has, however, to be remembered that the maximum permissible dose was set up for people occupationally exposed, and on the assumption that only a small fraction of the population is so exposed. For the whole population the figure may have to be reduced by a factor of 10 or even 50, but there may be other reasons for lowering the maximum permissible dose. It is now becoming evident that the doubling dose of leukæmia may be much less than was thought. Another factor, which came to light only recently, is the shortening of life-span due to an acceleration of the natural processes of ageing. A survey of radiologists in the United States, showing a reduction of life-span by five years, may be statistically uncertain, but there is no doubt about this effect from experiments on animals. These experiments show that every irradiation results in some irreparable damage, and that the lethal dose gradually decreases with age. If this is extrapolated to human beings, it leads to an inherent limit of life-span due to the natural background of radiations. This limit may be two hundred years or more, but the grim significance of this sort of speculation becomes apparent when applied to the present maximum permissible dose, which would reduce the life-span to about thirty years from the time exposure began. There seems, therefore, to be a good case for lowering the maximum permissible dose, quite apart from the genetic hazards.

Prof. Penrose opened his discussion of the biological damage due to radiations by directing attention to the ignorance there is of the intermediate steps in their action, stating that one could only speculate with the knowledge available. He pointed out that the major damage to populations exposed to an atomic explosion would be due to heat and blast, and not to radiation. Ionizing radiations were, however, damaging even in small intensities. Their effects could be divided into immediate, during the first few weeks, and delayed. The immediate effects, such as burns and anæmia, are well known. Their relation to the dose received is not linear, lower doses producing apparently no effect. With the delayed effects, such as cataract or leukæmia, the picture is different. From the evidence on survivors at Hiroshima and Nagasaki, as well as from the findings of Court Brown and Doll on patients treated with X-rays for anklyosing spondylitis, it is clear that the incidence of leukemia increases linearly with the dose. This linear relationship between exposure and delayed effects emerged toward the end of the Medical Research Council Committee's deliberations and was the reason for the demand for a reconsideration of the maximum permissible levels.

Another point to be clarified in the public mind is the difference between somatic and genetic effects. Direct damage to the fœtus by radiations has been studied on pregnant rats and mice as far back as 1931, and the findings have been supported by observations on human fœtuses. It was found that large doses to the mother's abdomen could clearly cause fœtal damage not previously suspected. Apart from miscarriage, this could result in the birth of babies with arrested development of the central nervous system leading to a special type of microcephaly. These risks, which had been known for some time, were definitely confirmed in the populations exposed at Hiroshima. Dr. Alice Stewart and her colleagues at Oxford claim to have observed delayed deleterious effects on the fœtuses exposed at any time during pregnancy to a much smaller dose of X-rays, such as occurs during diagnostic pelvic X-ray of the mother. In these offspring there was found to be a significantly higher incidence of leukæmia, which developed one, six, or even ten years after birth. Prof. Penrose hesitated to accept these findings as fully proved, but felt that although the damage occurred only in a small proportion of the mothers X-rayed in pregnancy, nevertheless it was important.

The genetic hazard can only be considered by looking at a long-term picture. There are two processes to be considered—chromosome breakage, which disturbs nuclear harmony, and point mutations, both of which may be the result of ionizing radiations. When a chromosome breakage occurs, both theory and experiment show that the effect is proportional to the square of the dose because, to produce an effect, there must be two breakages with the possibility of joining up again to reproduce. In the case of point mutations there is a direct linear effect, and this has been established by work with *Drosophila*. In man, such mutations are far from difficult to study, and this has in part produced the impression that the genetic hazard results only in an increase in the number of achondroplasic dwarfs. Actually, an increase in the rate of mutations will increase many other far more serious conditions. Referring to the nuclear test explosions, Prof. Penrose stated that the number of fresh mutants would be small and difficult to demonstrate, and in this sense only is it insignificant, counted over the whole population the number would certainly not be negligible.

Prof. Penrose then produced some evidence of the part played by radiation on the natural mutation-rate in man. One method is to study the incidence of diseases believed to arise by spontaneous mutation in relation to the ages of parents. For chondrodystrophy and acrocephalosyndactyly the age of the father, but not that of the mother, is strikingly high. This could be explained by assuming that the gene loci concerned were not very sensitive to radiation but mutated in response to other factors. For the most sensitive loci, mother's and father's age would both be only slightly raised, as seems to be the case in retinoblastoma neurofibromatosis and epiloia. Mongolism falls into a quite different group, its incidence increasing with an increasing maternal, but not paternal, age, and it is clearly not due to point mutation.

In answer to questions on the haphazard use of radioisotopes therapeutically and experimentally, both speakers stressed that nowadays every care is taken in the use of these materials. Prof. Rotblat explained that in the case of radioiodine therapy in thyroid disease, treatment is now limited to patients of the older age group where tumour induction after many years is of less importance

than in the younger age group. Physicians in Britain have been more conservative in accepting the possession of a Geiger counter as essential for their practice than has the medical profession in the United States. The contribution from short-lived isotopes used in medicine has been calculated to be very small.

The chairman concluded the meeting by a revision of the old rules of health, suggesting that one should stay indoors to be shielded from cosmic rays, to stop drinking milk to limit the intake of cæsium 137, and to keep away from the doctor, to avoid being X-rayed.

P. J. LINDOP.

[Britsh Medical Journal, London, Saturday, March 30, 1957]

STRONTIUM-90 IN MAN

Whenever a nuclear weapon is exploded, or atomic energy is released in a reactor, over 200 radioactive isotopes are produced. All these can be harmful to man, but the main villain of the piece appears to be strontium-90. Its notoriety is due to several reasons. First, it has a high yield: one in every twenty fissions results in the production of an atom of strontium-90. Secondly, it is long-lived: its physical half-life is 28 years. Thirdly, it finds its way from the soil into food. Fourthly, it is a bone seeker. And, fifthly, it is retained in the human body for a long time; the biological half-life is about 7½ years. The last two properties lead to the possibility that the β-rays emitted from radiostrontium may produce bone lesions and sarcomas as well as leukaemias.

Although the hazards from the deposition of strontium in bone have long been recognized, too little has been known about its distribution and metabolism to assess the hazards quantitatively. Recent papers [1, 2, 3] giving results of a world-wide survey of strontium content in man throw some light on the subject. Strontium-90 is an inevitable by-product of nuclear power production, and by the time most of our power requirements have been met from atomic energy there will be an embarrassingly large quantity of strontium-90 to be disposed of; but this does not present a hazard at the moment, since the strontium is not allowed to escape into the open. Even in a full-scale nuclear war strontium-90 would not be the main radiological poison; its damaging action would be small compared with that of the shorter-lived isotopes strontium-89 and iodine-131. It is the relatively small amount of strontium-90 accumulating from test explosions that causes some worry. Furthermore, strontium-90 does not present a genetic hazard, since it emits only β-rays, which do not penetrate to the gonads. It is the somatic hazard, the probability of inducing bone tumours, that is the main concern.

When a nuclear weapon is exploded in the air the radioactive fragments are carried by the upward draught high into the stratosphere. In the case of a high-altitude explosion—as the next Britsh H-bomb test is supposed to be—there is very little local fall-out, and practically all the radioactivity is taken up into the stratosphere. The rate of descent from there is very low, about 10% per year, and this allows the radioactivity to spread out over the globe, so that when it does come down, mainly with the rain, it is nearly uniformly distributed all over the world. By the time the active material has descended the short-lived radioactive elements have practically all decayed and only the long-lived ones, mainly strontium-90 and caesium-137, are deposited on the ground. Here strontium, being chemically similar to calcium, enters into the soil–plant–animal–human chain of calcium. The first step in the chain, the uptake of strontium by plants, depends on the root depth and on the calcium content of the soil. The latter varies greatly; differences by a factor of 100 are quite common. Since the uptake of strontium by the soil will be the greater the less calcium it contains, the concentration of strontium-90 per gramme of calcium in the soil, and thus the concentration in plants, may vary enormously. There is also a large variation with depth, most of the strontium being deposited in the top 4 in. (10 cm.) of soil. The consumption of vegetable food is one way of introducing strontium-90 into the body. But since most of the calcium

---

[1] Libby, W. F., Proc. nat. Acad. Sci. (Wash.), 1956, 42, 945.
[2] Kulp, J. L., Eckelmann, W. R., and Schulert, A. R., Science, 1957, 125, 129.
[3] Booker, D. V., Bryant, F. J., Chamberlain, A. C., Morgan, A., and Spicer, G. S., Radiostrontium and Radiocaesium Measurements in Biological Materials to December 1956, Atomic Energy Research Establishment, 1957, London.

in our diet comes from dairy products the next step in the chain, the uptake of strontium by grazing animals, is the most important one. Fortunately there is a large discrimination factor, of about 7, against strontium consumed by the cow appearing in the milk. Furthermore, there is another discrimination factor, of about 8, in going from food to the deposition of strontium in bone, so that the amount of strontium-90 per gramme of calcium in the human bone is very much smaller than that in the soil.

The concentration of strontium-90 is expressed in terms of so-called sunshine units (S. U.), one such unit being one-millionth of a microcurie per gramme of calcium. A recent survey[2] has shown that the content of strontium-90 in the human skeleton, resulting from all H-bomb tests carried out until the autumn of 1956, will eventually reach a value of about 2 S. U. This is an average value with a very wide spread. The uptake in different bones in the skeleton varies by as much as a factor of 15, the largest concentration being in the vertebrae and sternum, the lowest in the skull. There is also a big variation with age; children aged 0–4 years take up about five times more than adults, and they have also a greater sensitivity to radiations. Finally, there are large variations in individuals of the same age group, probably due to differences in diet.

The main problem is how to assess the hazard from a given amount of strontium-90 in bone. The maximum permissible concentration of strontium-90 is 1,000 S. U., which is 500 times the average amount which will accrue from test explosions. But such a comparison is misleading, since all maximum permissible levels were established for people occupationally exposed to radiations. When whole populations are exposed the maximum level must be greatly reduced. The assessment of this hazard hinges on a fundamental question: how does formation of bone tumours vary with dose? If a threshold dose exists below which tumours are not formed, then the small doses resulting from test explosions are probably harmless. But if there is no threshold, and tumour formation is a linear function of dose, then even the smallest dose will impart a small but finite probability of a bone tumour being formed. The available evidence is meagre, and, moreover, it entails comparison of strontium with raduim, since only after exposure to radium has production of bone sarcoma in man actually been observed. There is growing evidence that the relation between the amount of exposure to radiations and the probability of developing long-term effects is of a linear nature. This was the view accepted by the M. R. C. in its report,[4] which states that "each unit quantity of radiostrontium absorbed by the bone confers a certain probability of bone tumour formation." The consequence of a linear relationship is that every H-bomb test by causing an increase of the content of strontium-90 in bone will increase the frequency of bone lesions. But much more research work will be required before an accurate assessment of the hazard can be made.

Leukaemia and Natural Background Radiation

Sir,—It is now well established that ionizing radiations can cause leukaemia in man. The detailed survey carried out recently by the U. K. Medical Research Council[1] of the incidence of leukaemia among patients treated for ankylosing spondylitis with x-rays, as well as the earlier investigations by the U. S. Atomic Bomb Casualty Commission[2] of the survivors of Hiroshima and Nagasaki during the period 1947–55, have definitely shown that the incidence rate of leukaemia increases with the strength of the radiation dose received. Although the exact relationship between the two is not definitely established, it may not be unreasonable to assume—and there is some recent evidence in support[3]—that the probability of incidence of leukaemia is directly proportional to the strength of the exposure (at least for low and moderate doses).

In the analysis carried out by the U. K. Medical Research Council, the x-ray dose was estimated in two different ways—first, by calculating the total amount of energy absorbed by the whole body, and, secondly, by calculating the maximum dose of radiation received (at a point in the spinal marrow). The whole-body dose is more relevant in the present discussion.

[4] Medical Research Council, The Hazards to Man of Nuclear and Allied Radiations, 1956, London.
[1] The Hazards to Man of Nuclear and Allied Radiations, 1956. H. M. S. O., London.
[2] Pathological Effects of Atomic Radiation, 1956. National Academy of Sciences, U. S. A.
[3] British Medical Journal, 1956, 2, 704.

In the table given below, the figures in the last column are calculated from the corresponding data in columns 1 and 2, which are reproduced from the U. K. Medical Research Council's report. Thus, for instance, the whole-body integral dose of 7.5–14.9 megagram-roentgens (average 11.2) would correspond to a whole-body dose of about 160 roentgens for the "standard man" (mass 70 kilograms). The leukaemia incidence rate for this dose is 4.4 cases per 10,000 men per year (if we exclude the natural rate of 0.3 cases per 10,000 per year). Thus the incidence rate per roentgen per year per million persons comes out to be 2.8.

Whole-body Integral Dose in Megagram-roentgens	Crude Incidence per 10,000 Men per year	Incidence per Million per Year per Roentgen
0	[1] 0·3	
<7·5	0·7	>0·4
7·5–14·9	4·7	2·8
15·0–22·4	5·1	1·8
22·5–37·4	11·3	2·6
37·5–52·4	22·6	3·5
>52·5	60·2	<8·0

[1] The rate has been estimated from the national vital statistics for all forms of leukaemia excluding lymphatic leukaemia.

It may be seen that the incidence rate of leukemia is roughly 3 per million per year per roentgen.

An analysis of the Japanese data leads to a figure of roughly 1 per million per year per roentgen. This represents a lower limit to the incidence rate, since the gamma-ray doses have been estimated for a person in the open. Apart from the uncertainty in estimating the dose in this case, the actual radiation dose received by the survivors must have been much less, by a factor of 2 to 3, due to shielding. Thus the incidence rate is again seen to be very roughly of the order of 3 per million per year per roentgen.

Let us assume that the natural incidence of leukemia is due entirely to the action of the background radiation (to which everyone is exposed). The average natural death rate due to leukemia is of the order of 15 per million per year. In England and Wales the annual death rate from leukemia was 11 per million in 1920, and 49 per million in 1954; the reason for this increase is not yet clear. The Japanese death rate is about 16 per million per year.) A person receives a whole-body dose of 0.1 to 0.15 roentgen per year from natural background radiation—that is, from the naturally and normally present radioactivity in his own body and the radioactivity and cosmic rays in the environment. This is equal to a dose of 3 to 5 roentgens up to adult life (say, 30 years).

On the assumption that the natural incidence of leukemia is due entirely to background radiation, the figure of 3 leukemia cases per million per year per roentgen and a natural radiation dose of 5 roentgens up to adult life would lead to a natural adult incidence rate of 15 per million per year. The close agreement between the incidence rate as calculated above and the observed rate may, no doubt, be partly accidental, but it points to the high plausibility of the assumption that the natural incidence of leukemia could be due entirely to the background radiation.

Our thanks are due to Professor D. S. Kothari and Dr. M. L. N. Sastri for stimulating discussions.—We are, etc.,

SANTOSH KUMAR MAZUMDAR.
A. NAGARATNAM.

NEW DELHI.

[Reprinted from British Medical Journal, April 20, 1957]

STRONTIUM-90 IN MAN

Sir,—Strontium-90 is now frequently being discussed all over the world, and your leading article (*Journal*, March 30, p. 752) will no doubt be quoted in lay fora as an expression of the best medical opinion of this country. It is important that some of the weaknesses of this, in most ways excellent, short summary of the situation should be noted.

"The main problem is how to assess the hazard from a given amount of strontium-90 in bone." Most would agree with this quotation.

Permissible Body Burden for the Occupationally Exposed.—This has been given by the International Commission on Radiological Protection and the Medical Research Council's Committee on Protection against Ionizing Radiation as 1 μc of strontium-90 or approximately 1,000 S. U. (strontium units, $\mu\mu$c per gramme of calcium), and was derived as follows. From industrial experience in the luminizing industry it was considered that the minimum toxic body burden of radium—a chemical analogue of calcium and strontium—was 1 μc. Early results with experimental rodents in Chicago had indicated that the toxic dose of strontium-89 relative to radium was about 10:1. Strontium-90 and its daughter yttrium-90 liberate per disintegration about twice the energy of strontium-89. Therefore the ratio of strontium-90 to radium should be about 5: 1 as administered; but radium decays in the body to radon, a gas, much of which escapes in expired air, more in the case of recently deposited radium than from long-standing depots, and more in the case of experimental rodents than man. In terms of body burden of effective radiactive material in human bone it was to allow a factor of 2 for this, bringing the ratio strontium-90: radium=10: 1. The calculated, minimum toxic permanent body burden for strontium-90 is thus 10 μc. Allowing a safety factor of 10 a maximum permissible body burden of 1 μc was derived.

The following uncertainties are relevant. (1) Industrial radium used in the luminizing industry was a mixture containing variable amounts of mesothorium and other radioactive elements, too short-lived for their contribution to the observed toxic manifestations to be assessed at the time when the body burdens of luminizers were estimated. However, a few subjects who had received injections of relatively pure radium for medical (*sic*) reasons have been discovered: in them the minimum toxic body burden appears so far to be about 3 μc[1] as compared with recent estimates of about half a microcurie of "radium" in ex-luminizers. The minimum toxic body burden of radium cannot therefore be indicated with precision. (2) The ratio of toxic doses of radium and strontium-89 was derived from injected animals. After more chronic administration these radioactive materials may be expected to be more widely distributed in bone. (3) The comparative metabolism of radium and strontium may differ significantly in man from the rodent because of different size and bone structure.

Permissible Body Burden in General Population.—In spite of the uncertainties the maximum permissible body burden for strontium-90 remains one of the best authenticated among those derived for occupational purposes. However, because of the uncertainties it is not justifiable to use this figure as a yardstick when one is considering a general population as Libby[2] and Kulp *et al.*[3] have done. Your leading article and the Medical Research Council[4] have given the most cogent reason—the presence in a general population of foetal, neonatal, and adolescent subjects who have greater avidity for and greater sensitivity to radioactive strontium. On the other hand, with the homoeopathic doses they receive daily the young will distribute strontium much more uniformly in bone than will the adult. How much this latter offsets the former is unknown.

This matter of distribution of strontium is worthy of stress. The values given by Kulp *et al.*[3] for strontium–90 in human skeletons were not those directly measured but figures "normalized" for unevenness of distribution in different bones. Now, unless I am in error, the factor for normalization was derived from the single administration of strontium–85 to adults in the terminal phase of killing diseases. An ill adult patient, probably bed-ridden, given a single dose of a radioactive marker and dying 3 to 125 days later, is no criterion for the metabolism of a daily ingested calcium analogue, and to use this criterion to "normalize" observed data, particularly from children, is, to say the least, unscientific. In fact field studies on domestic animals[5] indicate that gross radioactivity from strontium–90 is fairly uniformly distributed throughout the bones. Moreover, the analysis of stable strontium in human bones from subjects of all ages shows a similar uniformity.[6] Strontium is a normal though apparently

---

[1] Looney, W. B., Hasterlik, R. J., Brues, A. M., and Skirmont, E., Amer. J. Roentgenol., 1955, 73, 1006.
[2] Libby, W. F., Proc. nat. Acad. Sci. (Wash.), 1956, 42, 945.
[3] Kulp, J. L., Eckelmann, W. R., and Schulert, A. R., Science, 1957, 125, 219.
[4] Medical Research Council, The Hazards to Man of Nuclear and Allied Radiations, 1956. London.
[5] Cox, G. W., and Morgan, A., personal communications.
[6] Sowden, E. M., and Stitch, S. R., A. E. R. E. MRC/R.2030, 1956.

non-essential metabolite, and radioactive strontium will follow the same pathways.

Radioactive strontium–90, now universally distributed over the surface of the world, should be considered in comparison with the naturally occurring radium found in all bone, human and animal. Calculations of acceptable risks for a population from body-burdens of radioactive strontium are best made therefore using natural radium contamination as the criterion. This approach is being pursued with vigour.

Dose-response Relationship.—Meanwhile, "How does formation of bone tumours vary with dose?" Is there a relationship between natural radioactivity of bone and natural incidence of bone sarcoma? Though this has been carefully looked for, the only conclusion is that the evidence is too scanty.

Is there "growing evidence that the relation between the amount of exposure to radiations and the probability of developing long-term effects is of a linear nature"? In so far that formerly there was none at all, while recently a linear relationship has been suggested between dose to a point in the spinal marrow and the incidence of leukemia in irradiated spondylitics (Appendix B[4]), this statement is true. But radiation-induced leukemia is an odd thing. It has a much shorter latency than other radiation-induced malignant states. Some would even yet argue that it is not a malignant condition. In the experimentally induced lymphoma of the mouse the relation between total dose and fractionation of the dose is most complex and certainly not simply linear.[7]

It would be unwise therefore to use leukaemia as a model for all radiation-induced malignancies and as a justification of the thesis of somantic mutation as the cause of cancer. It is wrong moreover, to attribute to the M. R. C. report[4] opinions which it did not express. The statement, "Each unit quantity of radiostrontium absorbed by bone confers a certain probability of bone tumour formation," was not in the report but one of its signed appendices. Furthermore, the statement would suggest a linear relationship only if each unit quantity conferred the *same* probability.

In point of fact, the quantitative evidence of the carcinogenic properties of radiostrontium, and there is not much of it, suggests that the relationship is not linear and that there is some form of threshold, or effective latent period.[8][9] Therefore your conclusion, "The consequence of a linear relationship is that every H-bomb test by causing an increase of the content of strontium-90 in bone will increase the frequency of bone lesions," is as yet without foundation. The correct conclusion in the present state of scientific knowledge is that a linear relationship sets the limit and indicates the worst possible conditions, so that it would be prudent to go easy until the real facts are ascertained.

Strontium in Animal Metabolism.—*Pari passu*, it is right and proper that investigations be made on the uptake of both natural radium and the now universal strontium-90. Your leading article notes that we derive most of our calcium and strontium from milk and certain of its products. A safety factor of about 7—the cow—is built into this route by which we ingest strontium-90. Your leading article quotes a second safety factor of about 8 in going from food to the deposition of strontium in human bone. The combination of 7×8 would indeed be impressive, but is unfortunately an overstatement. Sowden and Stitch[6] find the strontium content of human bones to be, in round figures, 100 p. p. m. of ash, of which about 40% will be calcium. The strontium : calcium ratio is thus about 1:4,000. In a general diet one can derive from the data of Harrison *et al.*[10] that strontium : calcium is about 1:500. Here is the factor 8, but this includes the factor for the cow insofar as milk and its products are part of the diet of the average adult. Moreover, Harrison *et al.* calculate that the discrimination against strontium in favour of calcium by the human gut gives a factor of about 2. Unpublished observations from the same source suggest that the human bone absorbs calcium and strontium from the blood without discrimination. The best figure at the moment for the human safety factor is thus only 2. It is likely to be rather higher for alkaline earths of vegetable origin than for those derived from milk, since lactose and some amino-acids cause reduction in the factor of discrimination.[11]

---

[4] Medical Research Council, The Hazards to Man of Nuclear and Allied Radiations, 1956. London.
[7] Mole, R. H., in Progress in Radiobiology, 1956, edited by J. S. Mitchell, B. E. Holmes, and C. L. Smith. Oliver and Boyd.
[8] Finkel, M. P., Radiology, 1956, 67, 665.
[9] Brues, A. M., J. clin. Invest., 1949, 28, 1286.
[10] Harrison, G. E., Raymond, W. H. A., and Tretheway, H. C., Clin. Sci., 1955, 14, 681.
[11] Wasserman R. H., Comar, C. L., and Nold, M. M., J. Nutr., 1956, 59, 371.

Strontium in Soil and Plant.—Your leading article infers that all the strontium-90 in plants is derived from the soil. But the leaf of the plant is a very good surface for the adhesion of small particles of "fall-out" and for the absorption of strontium as well as other mineral ions.[12, 13] Thus the present contamination may be much more related to the rate of fall-out than to integrated total "fall-out."—I am, etc.

Didcot, Berks J. F. LOUTIT.

SIR.—Your leading article (*Journal*, March 30, p. 752) states that strontium-90 does not present genetic hazards since it emits only β-rays which do not penetrate to the gonads. In view of the great importance of this subject at the present time, it would be interesting to know whether this statement is based on experimental evidence or merely on supposition. If the latter, then it is difficult to accept the argument without further investigation. It may be that the writer assumed that all the available strontium-90 is deposited in the bone and therefore is unlikely to affect the gonads. However, even on this assumption the β-particles can traverse distances of up to about a centimetre in soft tissues:[1] when the testes are in the inguinal canal, they must be less than one centimetre distant from bone at some stage in their journey. This may also apply to the ovaries during development.

A second possibility which may not be excluded is the effect of any strontium-90 prior to its deposition in the bone or after subsequent mobilization. If there is any similarity between the effect of strontium and lead it may be that strontium does in fact have an effect on the soft tissues in general. Lead is well known to have a special affinity for bone, but its main impact is on soft tissues, such as the nervous system and the gut. Until, therefore, these possibilities have been excluded by experiments of suitable length in mammals, such a statement as that in your leading article may convey a false sense of security.—I am, etc.

London, S. W. 17.

BRIAN H. KIRMAN.

A. E. R. E. HP/R 2017

ATOMIC ENERGY RESEARCH ESTABLISHMENT

THE RADIOLOGICAL DOSE TO PERSONS IN THE U. K. DUE TO DEBRIS FROM NUCLEAR TEST EXPLOSIONS PRIOR TO JANUARY 1956

By N. G. Stewart, R. N. Crooks, and Miss E. M. R. Fisher

1. INTRODUCTION

The size distribution of the radioactive dust particles in the cloud from a nuclear explosion depends both on the power of the weapon and on the manner in which it is exploded, whether on the ground, on a tower or high in the air. The larger particles fall to earth relatively close to the site of the explosion, but in every type of burst a considerable fraction of the total radioactivity generated is contained in fine dust particles whose rate of fall under gravity is small and which may therefore remain airborne long enough to diffuse widely throughout the atmosphere and affect large areas of the earth's surface.

The United Kingdom, being remote from all test sites used up to the present time, has been free from local effects and has provided a useful observation area in which to study the global contamination problem. In this paper, an account is given of the methods that have been used to measure the amount of radioactive material in the air above U. K., and the amount deposited on the ground, as a result of all weapons exploded between January 1951 and January 1956. Based on these measurements, an estimate is made of the integral gamma ray dose received by the average inhabitant of this country during his lifetime from all nuclear tests held to date. An estimate is also made of the dose that will ultimately be received per individual per generation if bombs continue to be exploded at the present rate.

---

[12] Tukey, H. B., and Wittwer, S. H., in Progress in Nuclear Energy VI: Biological Sciences, 1956. Pergamon Press.

[13] Russell, R. S., Squire, H. M., and Martin, R. P., A. E. R. E./S. P. A. R. 3, 1955.

[1] Medical Research Council, The Hazards to Man of Nuclear and Allied Radiations, 1956, p. 5. London.

2. PROPERTIES OF THE DUST PARTICLES

2.1 The particles reaching U. K. from the smaller type of nuclear explosion are spherical in shape, having been formed by the condensation of material which has been vaporised in the explosion. They consist mainly of fused silica or metal oxides, depending on whether the weapon has been exploded near or remote from the surface of the earth, but both types are impregnated with radioactive elements more or less uniformly throughout their volume. The particles from the thermonuclear tests over the Pacific atolls consist mainly of calcium carbonate or calcium oxide but they are shapeless, possibly due to the refractory nature of the coral on which the tests have taken place. The vast majority of the particles encountered in U. K. from all types of explosion have diameters of less than $10\mu (1\mu = 10^{-4}$ cm.).

2.2 The radioactive content of explosion dust falls into three categories:
(i) Fission Product Activity (β, γ)
(ii) Neutron Capture Activity (β, γ)
(iii) Alpha Activity

Fission Product Activity

The fission products contained in the fine dust are fairly representative of those generated in the parent explosion although, under certain circumstances, some may be preferentially deposited along with the larger particles near the test site. Different types of weapon produce the same assemblage of fission products in slightly different proportions, but the rate of decay of the gross activity is almost independent of these differences and varies approximately as the inverse 1.2 power of time. Thus the activity decreases by a factor of 10 for a 7-fold increase of time as measured from the instant of the explosion. Table I shows the percentage contributions made by some of the more important fission products to the beta-ray activity from the slow neutron fission of U^{235}. Only those contributing more than 5% to the total activity at any time are included in the list. At the foot of the table are given Hunter and Ballou's computed figures[1] for the beta activity associated with the fission of 10^8 atoms of U^{235}. It is often convenient to express the fission product activity of dust in terms of the number of fissions from which this activity is derived. Thus a sample which is disintegrating at the rate of 620 disintegrations per minute (dpm) twenty days after the time of the parent explosion may be said to contain 10^8 fissions (see Table I).

Activity from Neutron Capture

Although several different radioactive elements may be created by the capture of neutrons in materials close to the reacting core of a weapon, the only significant reactions to produce gamma-ray emitters are those associated with the natural uranium which may be used as the tamper material of the bomb. This uranium is subjected to intense neutron bombardment during the nuclear explosion and the following reaction takes place:

$$92^{U238} + {}_0n^1 \longrightarrow 92^{U239} \xrightarrow[23.5m]{\beta-} 93^{Np239} \xrightarrow[2.33d]{\beta-} 94^{Pu239} \xrightarrow[\text{long}]{\alpha} \ldots \quad (1)$$

Chemical analysis of the debris shows that in general about one neutron is captured in this way for every fission that occurs, both in nominal bombs and in thermonuclear explosions. The U^{239} decays completely before reaching the U.K. but at four days after time of burst the Np^{239} disintegration rate reaches a peak relative to that of the fission products and accounts for about 60% of the observed activity at that time.

In addition to this, a smaller number of the neutrons in a thermonuclear explosion undergo an (n,2n) reaction with U^{238} to form 6.7 day U^{237} which is also a (β, γ) emitter.

Despite the fact that Np^{239} and U^{237} contribute much to the gross activity of the dust particles in the first few days after burst, their short half-lives and the softness of their gamma rays prevent them from adding significantly to the hazards associated with the fission products.

Alpha-Particle Activity

By far the most important alpha-particle emitter in the explosion dust is Pu^{239}. Consider first a nominal bomb of efficiency 20% with plutonium as the

[1] Hunter, H. F. and Ballou, N. E. ADC-65.

fissile material. Since one plutonium atom is used per fission, there will be a residue of four atoms/fission after the explosion. But since (equation 1) we may assume that one atom of plutonium is created per fission by neutron capture in U^{238}, the dust particles from this type of explosion will contain five plutonium atoms per fission. If U^{235} is used as the fissible material, the content will be one plutonium atom per fission; this same figure has been found to hold for thermonuclear weapons.

2.3 Between 20% and 30% of the activity contained on the spherical particles collected from the smaller nuclear explosions has been found to be soluble in water. The mean figure for the particles from thermonuclear tests over the Pacific atolls is 55%.

3. MEASUREMENTS OF DUST CONTENT OF AIR

The atmosphere has been sampled in a routine manner for radioactive dust since 1948 by attaching air filters to the aircraft which carry out the standard meteorological flights from Aldergrove, Northern Ireland. The frequency of sampling has varied throughout the period from daily to three times per week. The aircraft fly for about eight hours on a triangular course over the Atlantic; the main flying on each sortie is done at 1700 feet and at 18,000 feet with occasional descents to sea level and thus a reasonable sample of the dust content of the lower atmosphere is obtained. Each plane carries a cylindrical filter of length six inches and diameter 3½ inches consisting of a mat of esparto grass paper inside a wire cage. The collection efficiency of this arrangement is about 100% for particles greater than 3μ but this drops to 70% for the finest sizes created by nuclear explosions.

The average amount of air sampled during each flight has been measured by comparing the activity on a cylindrical filter with the activity collected on a disc filter of the same material flown at the same time, the disc being mounted in a long tube containing an orifice plate so that the air flow could be determined during flight. In this way it has been estimated that each routine filter sample contains the active dust from 1530 kg. of air.

The air has also been sampled since early 1952 over successive 24 hour periods at ground level near Harwell by fitting standard cylindrical filters to a powerful blower which provides a daily throughput of 7400 kg. of air.

The beta activity of the filters is measured by mounting them coaxially over a calibrated cylindrical geiger counter type B12E. The wall of this counter is thick enough to eliminate effectively the soft beta radiations of Np^{239} and U^{237} and the system thus provides a measure of the fission product activity alone. The measured disintegration rate of a sample of known age can be expressed in terms of fission units by using the computed curves of Hunter and Ballou. The Np^{239} and U^{237} activities are determined separately by chemical analysis.

4. RESULTS

4.1 *General.*—When a nominal bomb is exploded in, say, Nevada, the dust cloud rises to a height of perhaps 10,000–12,000 metres and travels east with the prevailing westerly winds. As the cloud travels it diffuses both laterally and vertically and ultimately contaminates the lower atmosphere across a broad front at great distances. Although on the average the cloud from a Nevada explosion crosses U. K. on the fifth day after time of burst and thereafter every four to seven weeks as it circulates round the world, the behaviour of any one cloud can depart considerably from this average and to obtain a general idea of the way in which the concentration of dust varies with distance downwind or with time, it is convenient to consider the concentrations over U. K. of the fission products from a series of explosions whose times of burst are close enough together to allow them to be considered as a single event, at least throughout a period starting some three to four weeks after the mean time of burst.

The specific activity of air above U. K. in the period following the Russian and American tests in the autumn of 1951 has been plotted in Fig. 1A. In this diagram, time is measured from the mean date of the individual explosions, and the specific activity of the air, as determined from the Aldergrove flights, has been averaged over periods of approximately fourteen days to give a figure representative of a significant fraction of the circulation path of the air, and to smooth out short-period variations. In this presentation, earlier points are

omitted since the method cannot reasonably be used within the actual period when the explosions are occurring. A smooth curve has been drawn through the experimental points and it has been found by experience that the shape of this curve is quite characteristic of explosions of near nominal size which take place in the middle latitudes of the northern hemisphere. The curve has been corrected for the radioactive decay of the fission products according to a composite decay curve computed from the sum of the individual decay curves, giving the dotted line in Fig. 1 which can then be taken to represent the decrease with time of the specific air content of the dust itself or of the longer-lived isotopes.

The decrease with time of the specific dust content of the air over the U. K. can be due to increased spread of the cloud or to deposition or both. Vertical spread above 12,000 metres is prevented by the very low diffusion rates in the stratosphere (see below). There is also a barrier of stable air in the lower atmosphere (troposphere) reaching as far as 25°N of the equator which prevents widespread diffusion to the south. Finally, direct observations of the lateral spread of Nevada clouds by sampling planes based in Gibraltar and in Scotland suggest that lateral diffusion is virtually complete by the time the clouds have made one circuit of the world. All the points in Fig. 1 refer to clouds which satisfy this criterion and we must conclude that the dotted line in Fig. 1A represents the rate of removal of atmospheric dust from the atmosphere by deposition alone and that half the material is removed in approximately 22 days.

The behaviour of the clouds from thermonuclear explosions is quite different. The fortnightly averages of the specific air activity obtained from the Aldergrove flights following the Pacific tests in the spring of 1954 have been plotted in Fig. 1B. There is no sharp decrease of airborne activity such as was obtained after the Nevada explosions, and when the correction is put in for radioactive decay it will be seen that the concentration of dust has in fact been increasing throughout the ten months following the mean date of the explosions. The explanation is that a thermonuclear dust cloud penetrates into the stratosphere and reaches a height of the order of 30,000 metres soon after the explosion. Diffusion is a very slow process in the stratosphere and material returns to the lowest layers of the atmosphere at a much slower rate than in the case of the nominal bomb cloud. The gap in the record in Fig. 2 occurs during the period when the lower atmosphere was further contaminated by the explosion of several Russian nominal-sized bombs in the late Autumn of 1954. This contamination decreased in the manner shown in Fig. 1A for this type of bomb, enabling measurements to be resumed on the residual thermonuclear contamination in January and February, 1955.

Direct confirmation of the hypothesis that the thermonuclear explosion dust is held back in the stratosphere has been provided by a series of experiments conducted in the Autumn of 1954 and repeated in the Spring and Autumn of 1955. In these experiments the concentration of activity was measured at various heights in the atmosphere up to 15,000 metres. The mean concentrations, averaged over a period of two months in the Autumn of 1954, are shown in Fig. 2 which demonstrates quite strikingly the existence of a large accumulation of fine dust in the stratosphere several months after the tests. The origin of the dust was identified by chemical analysis of the fission products.

4.2 *Mean Concentration of Beta and Gamma Activity in Ground Level Air.*— The dust clouds from Nevada and Russia pass over U. K. on their first circuit of the world before they have diffused to ground level, so that the activity does not reach high values in the surface air over this country. The peak concentrations, averaged over 24 hours, are generally in the region of 5 dpm of beta activity per cubic metre of air, but individual peaks of 25, 15, 10 and 8 dpm/m^3 have been observed. Thermonuclear explosions produce smaller concentrations because of the slow rate of diffusion from the stratosphere, the highest observed figure being 0.5 dpm/m^3. The average concentration of activity from all bombs exploded within the period April 1952, to January 1956 was 0.5 dpm/m^3, and most of this came from explosions in Nevada. The mean concentration in the eight months following the 1954 Pacific thermonuclear tests was only 0.1 dpm/m^3. Although much of the material from the thermonuclear explosions has yet to reach ground level, the average over the next few years due to bombs already exploded is not expected to exceed this figure of 0.1 dpm/m^3.

The external gamma dose rate corresponding to 0.5 dpm/m^3 of fission product activity in air has been computed by applying Chamberlain's method[2] to the gamma energy spectrum of the fission products. Including the dose rate due to Np^{239} and U^{237} the total figure is 10^{-3} milliroentgen (mr) per year to a person fully exposed to the radiation.

It is useful to compare the figures just given with those for the natural radioactive content of air. The combined disintegration rate of the natural RaB and RaC in the atmosphere at Harwell, averaged over the period November 1950 to March, 1951 was 128 dpm/m^3. The corresponding gamma dose rate is about 0.4 mr per year. The Harwell values for the RaB and RaC disintegration rates in air are, on the whole, smaller than those found elsewhere; mean values of 220 dpm/m^3 and 470 dpm/m^3 have been found at Windscale[3] and Cambridge[4] respectively while much higher figures are implied by recent Radon measurements in London.[5] Moreover, under stable atmospheric conditions, the RaB and C content of the air near ground level may reach values greatly exceeding the mean levels. The highest individual peak in the Harwell observations was 1800 dpm/m^3 which is 14 times the average value; similar values have been observed by other workers.

4.3 *Mean Concentration of Sr^{90} and Pu^{239} in Ground Level Air.*—No routine measurements have been made of the concentration in air of any individual isotope but it is possible to estimate the figures for those with relatively long half-lives. This will be done for the important isotopes Sr^{90} and Pu^{239}.

The mean fission product content of the air in the period April 1952 to January 1956, derived by calculation from the observed beta activity, was 5×10^5 fissions per cubic metre. Using an apparent fission yield of 4%, which is not inconsistent with radiochemical evidence, it follows that the mean concentration of Sr^{90} in the air within this period was 4×10^{-16} μc/c.c. The I. C. R. P. occupational m. p. l. is 2×10^{-10} μc/c.c.[6]

An upper limit for the mean plutonium concentration has been calculated in a similar manner, by allowing five atoms of Pu^{239} per fission from an atomic weapon in the nominal size range and one atom per fission from thermonuclear weapons. The mean concentration over the same period is thus found to be 3×10^{-17} μc/c.c.; the I. C. R. P. occupational m. p. l. is 2×10^{-12} μc/c.c.

5. DEPOSITION OF RADIOACTIVE DUST: SAMPLING METHODS

Dust particles which are too small to have a significant falling speed in air are removed from the atmosphere in two ways:—

(i) by washout in rainwater

(ii) by dry deposition directly onto surfaces.

Continuous measurements have been made since early in 1951 of the fission product content of rainwater falling on roofs at Milford Haven, Pembrokeshire, and Chilton, Berkshire. The roofs have been coated with chlorinated rubber paint which is chemically inactive and provides a smooth surface over which the rainwater and the entrained dust flow readily. The water is passed through cylindrical esparto grass filters of heavier gauge than is used for air filtration. The filters are exposed for 24 hour periods during rain and, after removal, are dried and their activity measured on the standard counting assembly; a suitable interval of time is allowed for the short-lived natural Radon and Thoron daughter products to decay before counting is started. Allowance is made for the solubility of the material when deriving the true deposition figures from the measured counting rates; check measurements of the solubility are made at intervals during a particular series of explosions.

The problem of the direct deposition onto surfaces is a complex one and some aspects of it are now being studied by Chamberlain at Harwell.[7] Owing to the complexity of the earth's surface it is obvious that realistic measurements of the amount of radioactive dust from nuclear explosions deposited in this way would be impossible. But since Chamberlain's early work has shown that, under certain conditions, this mechanism of deposition can be as important as rainwater deposition, it cannot be lightly ignored. It is thought, however that in the cir-

[2] Chamberlain, A. C. A. E. R. E. Report HP/R. 551.
[3] Dunster, H. J. Private Communication.
[4] Satterly, J., Phil. Mag. 16, 584 (1908).
[5] Anderson, W., Mayneord, W. V. and Turner, R. C., Nature, 174, 424 (1954).
[6] Recommendations of the International Commission on Radiological Protection. B. J. R. Supplement No. 6, 1955.
[7] Chamberlain, A. C. A. E. R. E. Report HP/R 1261.

cumstances peculiar to deposition from the fine dust clouds from atomic and thermonuclear explosions, deposition by rain is by far the more important factor. The reasons for this view are given in the following section.

It will be shown in para. 831 that on the average 80% of the integrated gamma dose recived by an individual in U. K. from radioactive material deposited by *rain* after a Nevada explosion is due to the deposition that takes place while the cloud is crossing U. K. for the first time. It has already been stated that the concentration in the air at ground level during this first passage is generally low, and, in fact, the transfer rate from from air to ground would have to be impossibly high before it could add significantly to the rain deposition. To investigate what happens on subsequent circuits of the cloud, when vertical diffusion is complete, an experiment was carried out in which the deposition by rain in four successive fortnightly periods was compared with the dry deposition onto a glass plate covered by a film of vaseline. The results obtained in the four periods were very consistent and showed that the deposition by normal rainfall was four times that due to dry deposition on the sticky surface. It was therefore concluded that since the rain deposition from the cloud during its second and subsequent circuits of the world was less than that during the first, the rainwater measurements alone should provide a fair measure of total deposition. In any case, the method of sampling adopted ensures that any radioactive dust deposited on the collecting roofs during dry spells is washed off by subsequent showers and included in the rainwater activity measurements.

The weighting factor in favour of rain deposition is even greater in the case of dust from thermonuclear explosions. Since the dust is fed slowly into the troposphere from the stratosphere and is subsequently removed by deposition, a gradient of activity is maintained in the lower atmosphere and the concentration near ground level is very small (Fig. 3). The rain brings down activity from regions where the concentrations are comparatively high and it is estimated that for these reasons, rainwater deposition exceeds dry deposition by a factor of about twenty.

6. DAILY DEPOSITION LEVELS AT CHILTON AND MILFORD HAVEN

The daily deposition records at Chilton and Milford Haven are similar in general although occasionally differing markedly in detail. All daily deposits of surface activity greater than 2 mc/km² observed between February 1951 and January 1956 have been arranged in groups and the frequencies of occurrence of the various groups are given in the following table:

Range of Values of Deposited Activity (mc/km²)	Frequency of Occurrence	
	Chilton	Milford Haven
2–10	83	76
10–20	9	5
20–40	3	1
40–60	3	----
60–80	1	----
80–100	----	1

The highest daily deposit at the sites was 73 mc/km² at Chilton and 93 mc/km² at Milford Haven; these occurred in heavy rain about the same time, some five days after the explosion of a weapon in Nevada in the Autumn of 1951. The highest deposition in a single day from a thermonuclear weapon test was 10 mc/km² at Chilton and 39 mc/km² at Milford Haven.

7. DEPOSITION FROM THE 1952 NEVADA TESTS

7.1 Before proceeding to the final stage of calculating the gamma dose from the fission products deposited to date, it is important to consider in some detail the deposition measurements from one particular series of explosions in order to check that the results are in fair agreement with expectation. It is particularly suitable to choose for this purpose the Nevada tests in the period April–June 1952 since the U. S. A. E. C. have published a paper [8] summarising

[8] Eisenbud, M., and Harley, J. H. Science 117, 141 (1953).

their own deposition measurements within the U. S. during this series and a useful comparison of results can be made.

7.2 *U. K. Measurements in 1952.*—The 1952 Nevada tests consisted of eight explosions between April 1st and June 5th, the mean date of burst being May 6th. During this period and up to the time in late November when the atmosphere was further contaminated by radioactive dust from a Pacific test, the usual daily measurements were made at Chilton and Milford Haven of the activity deposited in rainwater. The quantity to be computed, to simplify comparison with the U. S. results, is the cumulative value of the deposited activity on December 6th, i. e. seven months after the mean date of burst, when deposition was virtually complete. The approximate method used was to add up the total deposition for each individual month and to calculate the probable value of this on December 6th using the Hunter-Ballou decay curve for the fission products from U^{235}. The only refinement to this method was that each heavy deposition resulting from the passing over U. K. of a single identifiable cloud on its first transit of the earth was followed through individually on its own decay curve.

The calculations show that the activity deposited at Chilton and Milford Haven amounted to 6350 dpm/m² and 8200 dpm/m² respectively on 6th December, 1952. The lower figure at Chilton is due to the fact that during an important part of the deposition period the rainfall was much lower there than that at Milford Haven, where it was very close to the average for U. K. The higher figure may therefore be taken as representative for this country.

7.3 *Comparison of British and American Results in 1952.*—In the U. S. the dust is collected on horizontal sticky plates which have the property of retaining the dust brought down by rain in addition to that deposited directly from the air. A paper has been published [8] giving an analysis of the measurements made at a network of 121 stations within U. S. during the period following the 1952 Nevada etsts. The average values of the deposited fission product activity are given at several distances between 300 and 2,400 miles downwind from the test site. The quoted figures refer to the cumulative values of the deposited activity on 1st January 1953, but using the conversion figures given in the report, these have been adjusted to a reference date of 6th December 1952 and are plotted in Fig. 8. One additional point not included among the original figures has been derived for the distance of 17,000 miles by averaging the deposition figures obtained at several places on the U. S. West coast and shown in the map in Fig. 1 of the report. This additional figure will include not only the deposition of material which has travelled once around the world but also that due to any temporary "back-flow" from the test site, and it represents therefore an upper limit for deposition at 17,000 miles downwind.

The U. K. figures have been plotted for comparison in Fig. 8, and the agreement is seen to be fairly good. No rainfall figures are given in the U. S. report for the period in question, but the difference in the mean rainfalls of U. S. and U. K. is not large enough to invalidate the comparison.

7.4 *Check on the Order of Magnitude of the U. K. Figures.*—The deposition of active material from bomb explosions in Nevada is likely to be confined to the Northern Hemisphere since the diffusion of matter across the stable air barrier is slow and is not likely to reach significant proportions in the time required for the bulk of the material to be deposited. It is therefore possible to estimate the average ground surface activity on the assumption of uniform distribution.

The number of fissions in a nominal bomb explosion is approximately 2.4×10^{24}.[9] The Hunter-Ballon decay curve shows a disintegration rate of 3.5×10^{-7} dpm per fission at +7 months. The area of the Northern Hemisphere is 2.5×10^{14} m²

$$\text{Calculated specific ground activity} = \frac{3.5\times10^{-7}\times2.4\times10^{24}}{2.5\times10^{14}}$$

$$\therefore \quad \text{per nominal bomb at } +7 \text{ months} = 3350\,dpm/m^2$$

The observed representative figure was 8200 dpm/m² from eight bombs exploded. Two of the bombs were described in the Press as very small and it may

[9] The Effects of Atomic Weapons, U. S. Government Printing Office, Washington 25, D. C. (1950).

be assumed as an approximation that the fission product release during the series was equal to that from six nominal bomb explosions.

Observed specific ground activity = 8200/6

$\therefore$

per nominal bomb at +7 months = 1367 dpm/m^2

The observed figure is therefore of the expected order of magnitude, and the U. K. deposition measurements thus show satisfactory agreement with both the U. S. result and with the calculated values.

8. COMPUTATION OF THE GAMMA RAY DOSE FROM DEPOSITED FISSION PRODUCTS

8.1 *General Procedure.*—The gamma ray dose received over a period of 50 years due to all bombs exploded before January 1956, will first be calculated for the case of a man standing on an infinite plane surface. This will be done by calculating the total dose to the man from each single deposition and summing the series. The figure thus obtained will be larger than the dose received in practice since the shielding provided by buildings against gamma rays and the removal of activity from the earth's surface by drainage and by weathering have been ignored. Suitable adjustments to take account of these factors will be considered later in the report.

8.2 *Method of Calculation.*—Consider first the dose rate at a point above a plane uniformly contaminated with fission products of some particular age. The fission products emit a large number of gamma rays whose energies and rates of emission can be obtained from the literature. The dose rate at the point can therefore be regarded as the sum of the dose rates from a number of uniform deposits each of which emits gamma rays of one energy only. But even this simplified problem has not been solved numerically for the general case owing to the complex nature of gamma ray scattering and absorption. However, Chamberlain [2] has devised an approximate method for estimating the dose rates at various heights above a plane for all gamma energies, and this has been shown in recent measurements at A. E. R. E.[10] to give an accurate result for the particular case of Co^{60} gamma rays. There is no guarantee that the method is equally accurate at all other energies, but the error cannot be large enough to vitiate the present calculations.

Dale, Kendall and McKendrick [11] have applied Chamberlain's method to the gamma rays emitted by the fission products of U^{235} and Pu^{239} and have computed the dose rates received by a man standing on an infinite plane uniformly contaminated with those products at times between one hour and three years. The two sets of dose rates do not differ by much and those for U^{235} have been used in the computations which follow; additional values have been worked out for times between 3 and 50 years. Some of the more important quantities derived from Dale's data in the intermediate stages of computations have been listed in Table II as they illustrate several features of the fission product gamma activity. Column 2 shows how the dose rate from a specified surface activity varies with the age of the fission products; thus the dose rate from a deposit of fission products one day old is four times as great as that from an equally active deposit of products three years old. Column 3 shows how the dose rate from a particular deposit varies with time; the decrease is due mainly to the radioactive decay of the fission products. Column 4 gives the total dose received within a period of 50 years from depositions which occur at various times after an explosion; these figures allow the integrated dose from any deposition of known age to be rapidly assessed. The increase of the figures with time is due to the fact that old fission products decay less rapidly than young and therefore, for a given initial surface activity, deliver a greater integrated dose. The last column shows the integrated dose between various times T and 50 years from a deposit whose specific activity at one day after time of burst is 1 c/km². This information has been plotted in Fig. 4 which demonstrates the rate at which the dose from a particular deposit is delivered. Since no deposition has yet taken place in U. K. until after the fourth day from the time of an explosion, the earlier points are of academic interest only. The graph shows that 50% of the total dose from a deposition occurring on the fourth day after an explosion is received within the following 26 days.

---

[10] Gale, H. J. Private Communication.

[11] Dale, G. C., Kendall, R. A., and McKendrick, J. C., A. W. R. E. Report HER-H7/53.

8.3 *Results of the Dose Calculations*—

8.3.1 *Atomic Bombs of approximately Nominal Size.*—Since the deposition from these bombs is completed within a few months, the calculation of the integrated dose by the procedure outlined above is a simple matter. The calculations have been done in two stages. The integrated doses from the individual identifiable depositions from clouds crossing U. K. for the first time have been calculated by combining the surface activity of these deposits with the data given in column 4 of Table II. The dose from activity deposited subsequently is determined in a similar manner, but here the depositions have been summed over fortnightly periods and the effective age of the mixed products measured from the mean time of burst of the weapons. The results of the calculations are given in some detail below for the eight Nevada explosions in 1952.

	No. of Bomb	Integrated Dose within 50 years (μr)	
		Chilton	Milford Haven
Deposition from clouds on first circuit of world	1	12	17
	2	0	0
	3	76	20
	4	8	8
	5	114	6
	6	14	166
	7	13	39
	8	59	71
Total, first time round		296	327
Residual, from subsequent deposition		56	91
Total integrated dose		352	418

The resultant integrated doses at the two sites do not differ by much, but it will be observed that the main contributions at each place come from different bombs. About 80% of the total dose is produced by deposition from clouds crossing U. K. for the first time.

The results obtained from seven test series of similar type involving the explosion of some 60 weapons are summarised in the top part of Table III. The average integrated dose from the deposited activity is 66 μr per explosion, and on this basis an allowance has been made for another 15 explosions from which dust was probably deposited in U. K. but for which no measurements were made. The figures for the number of weapon exploded have been obtained from press reports. The British tests in Australia have been neglected as the clouds from these were not detected in the North Hemisphere. The integrated dose from all bombs in the nominal size range exploded up to January 1956, based on the mean of the measurements at Chilton and Milford Haven, is 4.9 mr.

8.3.2 *Thermonuclear Explosions.*—The computation of the integrated dose in U. K. due to dust from thermonuclear tests falls naturally into two parts. Firstly, there is that part of the dose arising from material which is already on the ground and secondly, that due to material which has yet to be deposited and is at present airborne.

The first part of the calculation is a repetition of that already carried out for the nominal weapons, but there are some differences worthy of note. The integrated dose is no longer dominated by the deposition which takes place in the first few days after burst. The rate of deposition of activity is a slowly decreasing function of time of the type shown in Fig. 1B, and the mean daily deposition one year after burst adds more to the integrated dose than that at any earlier time. Again, since the time scale of the deposition is long, the records are frequently interrupted when the lower atmosphere is temporarily contaminated by dust from nominal bomb tests and a certain amount of interpolation is necessary; this presents no serious difficulty since the deposition rates are slowly varying. The results of the computations for the thermonuclear test series in the Pacific in 1952 and 1954 are given in the lower part of Table III; the figures have been separated into those based on direct measurements of deposited activity and those based on interpolation.

In order to forecast the integrated dose from active dust still to be deposited, estimates are required of the total amount of activity in the atmosphere and of its rate of descent.

An estimate of the fission product content of the atmosphere above U. K. in September 1954 can be obtained by extrapolating the graph of Fig. 2 and integrating under the curve to obtain the total fission product content of an infinite vertical column of cross-section 1 square metre. This extrapolation has been done by producing the curve of Fig. 2 linearly to a height of 10mb—the estimated initial height of the cloud—and assuming the concentration above that level to be constant. The value of the integral thus determined is 3×10^{12} fissions/m^2. The deposition rates at Chilton and Milford Haven during the same period were equivalent to 4.7×10^{11} and 2.6×10^{11} fissions/m^2/year respectively, or 16% and 9% of the content of the column per year. The same integration carried out with data obtained in January and February 1955 showed that the content of the column had increased since September to a value of 10^{13} fissions; the deposition rate had risen in proportion and was 12% per year at both sites. Whether the increased concentration is a seasonal effect or represents a gradual downwind or lateral diffusion into the atmosphere above U. K. from regions of higher concentration cannot be answered confidently. It is hoped that further observations will help to clarify this point. The present view is that the increase may be due to the slow gravitational settling of the fine dust in the rarified air of the stratosphere and that the higher levels of the stratosphere may now be depleted of activity. On this hypothesis, the value of the integral obtained in January and February 1955 may well be an overestimate of the content of the column, but it will be accepted here as the basis of the remaining dose calculations. A rough check can be made on the magnitude of the figure. It is reasonable to suppose that by the beginning of 1955 the lateral spreading of the clouds from the 1954 Pacific explosions was complete (activity reached U. K. within 12 days of the explosion on March 1st) and that the air activity above U. K. was therefore a fair sample of the world distribution. The total fission content of the atmosphere can then be calculated by summing the content of the column above U. K. (10^{13} fissions/m^2) over the area of the surface of the earth (5×10^{14} m^2); the result is 5×10^{27} fissions in the atmosphere in February 1955. This number of fissions is associated with the release of 30 megatons of energy (in the conventional units, a megaton of energy is the energy derived from the explosion of a million tons of TNT). Nothing is known of the total energy released in the 1954 Pacific tests but a typical thermonuclear weapon is commonly suposed to have a power of about 10 MT. The computed figure of 30 MT, and by implication the air activity figure from which it was derived, is therefore of the right order of magnitude and in the absence of definite weapon information, no more than this can be claimed.

The second part of the dose calculation, then, is based on an atmospheric content of 10^{13} fissions per unit column in early 1955 from the 1954 Pacific tests. A small factor of safety is introduced by assuming that deposition takes place at the rate of 20%. The computed integrated dose from the activity yet to fall is then 20.3 mr. Observations of deposition from the 1952 tests were too frequently interrupted for a similar calculation procedure to be adopted. It has been found experimentally, however, that at comparable times after burst the air concentrations and deposition rates from the 1952 and 1954 test series are in the ratio of 1:3.6. The residual integrated dose given in Table III for the 1952 tests is based entirely on the calculations for the 1954 series, using this ratio.

The figures for the Russian thermonuclear explosion in 1955 are based on the Russian statement of the power of the airburst bomb as being in the megaton range.

8.3.3 *Estimate of a Realistic Integrated Dose for U. K.*—The estimated total dose received by a hypothetical inhabitant of the U. K. standing on an infinite plane is about 35 mr in the course of 50 years from all explosions up to December 1955 (Table III); thermonuclear explosions account for about 30 mr of this total. In practice, only a small fraction of this estimated dose will be received by the average inhabitant of this country. Much of the activity brought down by rain will be washed into drains, particularly in built-up areas, or washed down crevices in the open country. This is particularly true of the activity from thermonuclear explosions, 55% of which is soluble. A factor of three to take account of this aspect would appear to be conservative. Much protection is provided by ordinary buildings against the gamma rays from the fission products which do remain in the surface of the ground. Measurements have recently been carried out at A. E. R. E. of the protection provided by a conventional semi-detached house against a uniform deposit of Co^{60} on the ground.[13] The average

[13] Stewart, N. G., Crooks, R. N., Chisholm, J. M., Gale, H. J., A. E. R. E. Report HP/R. 1782.

protection factor was 20. It can be shown by calculation that the gamma rays from Co^{60} are suitable substitutes for fission product gamma rays in this type of experiment. The protection afforded by multi-storey buildings could be greater than this but, to remain conservative, we choose a representative protection factor of seven based on the occupant of a semi-detached house who spends an average of 2½ hours per day out-of-doors. This brings the overall protection factor to 21. A more realistic estimate, therefore, of the total dose to be received within the period 1945–1995 by the average inhabitant of this country due to deposition from all bombs exploded up to January 1956 is 1.7 mr.

It is useful to calculate the probable dose per generation on the assumption that nuclear weapons continue to be exploded at the present rate for a very long time. Under these conditions, the dose within any 30-year period will build up to a flat maximum several generations hence. For the purpose of this calculation we assume, arbitrarily, that all the fission products released up to 31st December, 1955 were released within a period of three years. It can be shown rigorously that if the dose to *infinity* from a single year's explosions is X mr, then the 30-year generation dose in the equilibrium state will be 30 X mr. The calculations summarised in Table III have been extended to include the period between 50 years and infinity, and the total dose to infinity from all bombs fired to date is then found to be 56.5 mr for a person in the open. On the argument just described, it follows that the ultimate generation dose will be 565 mr under the same conditions. For the average individual (protection factor of 21) the estimated generation dose will be 27 mr.

9. DEPOSITION OF SR^{90} AND PU^{239}

9.1 *Levels of Sr^{90} and Pu^{239} on U. K. Soil.*—Since May 1st 1954, rainwater samples have been obtained at Milford Haven by collecting the rain falling within successive three-weekly periods in a polythene funnel of area 0.22 m^2. These samples have been analysed radiochemically for Sr^{90} by R. G. D. Osmond at the A. E. R. E. Woolwich oustation, and the cumulative curve of deposition of Sr^{90} is shown in Fig. 5 in units of milicuries of Sr^{90} per square kilometre. The level of 0.77 mc/km^2 in April 1954 has been deduced from previous gross fission product measurements, using an Sr^{90} yield of 4%. The mean rate of fall over the period May 1954–March 1956 is 2.3 mc/km^2.

Using our figure for the mean rate of deposition of stratospheric dust—12% per year—this implies that the ground content of Sr^{90} due to bombs exploded before January 1956 will pass through a maximum of 14 mc/km^2 in 1968. If the present rate of firing continues indefinitely, the Sr^{90} will build up to an equilibrium ground value of about 0.2 C/km^2.

By way of comparison, Eisenbud [13] has reported that the top foot of soil in U. S. contains an average of 0.4 C/km^2 of Ra^{226} and a similar figure is expected to hold for the soil in this country.

The amount of Pu^{239} deposited in the U. K., deduced from the gross fission product measurements, is found to be 0.08 mc/km^2 in December 1955. This is expected to rise to a value of 0.4 mc/km^2 from bombs exploded up to the end of 1955. If the present rate of firing continues, the plutonium content of the soil will increase steadily and will reach a value of about 0.1 mc/km^2 for each year of firing.

9.2 *Levels of Sr^{90} and Pu^{239} in Rainwater.*—The mean concentration of Sr^{90} in Milford Haven rainwater between December 1952 and December 1955 was 1.7 $\mu\mu$c/litre. The mean concentration in 1955 was 3 $\mu\mu$c/litre and if the present rate of firing continues indefinitely an equilibrium level of 8 $\mu\mu$c/litre will be reached in about 10 years time. The concentration of Sr^{90} in drinking water is likely to be much below that in rainwater because of filtration and adsorption in soil. The occupational m. p. 1. for Sr^{90} in water is 800 $\mu\mu$c/litre.[6]

The average concentration of Pu^{239} in rainwater during the three year period ending December 1955 has been computed from the gross fission product deposition figures, the value being .03 $\mu\mu$c/litre. An equilibrium value of .08 $\mu\mu$c/litre will be reached in ten years time if firing continues at the present rate. The occupational m. p. l. for Pu^{239} in water is 3000 $\mu\mu$c/litre.

10. THE PROBLEM OF C [14]

From the official data on the nominal bomb given in the Effects of Atomic Weapons (9) it is safe to assume that, of all the neutrons which are not used

[13] Hearing before Joint Committee on Atomic Energy of U. S. Congress, April 15th, 1955.
[14] Libby, W. F., Radiocarbon Dating, Univ. of Chicago Press.

up in the fission chain reaction, not more than one escapes into the air for every fission that takes place. Most of these escaping neutrons are ultimately captured in Nitrogen to form 5600 year C^{14}. As an upper limit we may therefore presume that one atom of C^{14} is formed per fission. This implies that about 300 curies of C^{14} are formed in a nominal bomb explosion. The estimated amount of 28 year Sr^{90} formed at the same time by fission is 2500 curies. But since the m.p.l.'s for C^{14} in air and water are greater than those for Sr^{90} by factors of 7×10^3 and 10^5 respectively ([8]), it would appear that the amount of C^{14} in the air presents a relatively minor hazard.

The long-term effect can best be studied by comparing the amount of C^{14} created in nuclear explosions with that which is naturally present in the world as a result of cosmic ray reactions. It follows from our previous assumptions that the present rate of firing is adding no more than .07% annually to the natural world content of C^{14}. ([14]) The importance of this may be gauged from the fact that the natural C^{14} in the world contributes about 1% of the normal background dose to the human body.

11. SUMMARY

The size distribution of the radioactive dust particles in the cloud from a nuclear explosion depends both on the power of the weapon and on the manner in which it is exploded, whether on the ground, on a tower or high in the air. The larger particles fall to earth relatively close to the site of the explosion, but in every type of burst a considerable fraction of the total radioactivity generated is contained in fine dust particles whose rate of fall under gravity is small and which may therefore remain airborne long enough to diffuse widely throughout the atmosphere and affect large areas of the earth's surface. These particles are removed from the atmosphere mainly by rain.

The most important radioactive components of the dust are the numerous fission product nuclei which decay at various rates and emit both beta and gamma rays of a wide range of energies. As time proceeds the shorter-lived isotopes in a representative sample of fission products decay into insignificance in comparison with those of longer half-life, and after about eight years nearly all the gamma activity stems from the single isotope Barium 137 daughter of Caesium 137 which has a half-life of 30 years. The gamma rays from fission products suspended in the air or deposited on the surface of the ground add to the general level of background radiation and increase the external radiological dose to the human body. Some of the individual isotopes may enter the human body through biological chains and produce internal effects. The most important of these is generally considered to be 28-year Strontium 90 which follows Calcium in biological systems and is deposited in bones from which it is removed very slowly.

The amount of activity deposited in rain has been measured at Chilton, (Berkshire) and Milford Haven (Pembrokeshire) since January 1951, by passing rainwater through filters whose radioactive content is then measured on a conventional Geiger counter assembly. There is no significant difference between the aggregate results obtained at the two sites. An example, given in some detail in the report, shows that the measured total deposition in the United Kingdom from the 1952 test explosions in Nevada is in good agreement with the very full data which the United States have published for the series.

The atmosphere between 500 metres and 5500 metres above the U. K. has been sampled for radioactive dust about three times weekly since 1948 by attaching air filters to the aircraft which carry out routine meteorological flights from Northern Ireland. In addition, the air at ground level near Harwell has been monitored continuously since early 1952.

The records reveal one clear and important difference between the clouds from weapons in the nominal size range (similar to that exploded at Nagasaki) and those from the large thermonuclear explosions in the Pacific. It is found that the concentration of fine dust in the atmosphere from nominal size explosions decreases in a regular and characteristic manner with time and that half the dust is removed by deposition in 22 days. The fine dust from a thermonuclear explosion, on the other hand, enters the stratosphere soon after burst and from there it diffuses downward so slowly that only 10–20% of the dust is deposited annually. Much of the activity from the 1952 and 1954 thermonuclear tests in the Pacific is therefore still in the stratosphere, and in order to determine the possible effects of these explosions in the U. K. it is necessary to form an estimate of this amount. This has been achieved by extrapolating the results obtained from systematic surveys of the radioactive content of the air between

ground level and 15,000 metres carried out in September 1954, February 1955 and September 1955.

The gamma ray dose from each individual deposit of fission products has been calculated initially for the idealised case of an individual standing on an infinite flat plane. A protection factor of three has then been introduced to take account of the material which is washed into drains or is otherwise removed from the topmost layer of the earth's surface. This factor is believed to be conservative, since about one half of the material already deposited has been found to be soluble in water. An additional factor of seven has been allowed for the shielding provided by buildings against the gamma rays from fission products. This figure has been derived from measurements carried out at A. E. R. E. with the gamma rays from Cobalt 60, and is based on the assumption that the average individual spends 2½ hours daily out-of-doors.

The individual doses have been summed, and the total external dose to be received by the average inhabitant of the U. K. due to material deposited on the ground from bombs exploded before 1st January 1956 is estimated to be 1.7 mr. About 70% of this dose is associated with material which has yet to be deposited. The dose due to debris suspended in the air near ground level is negligible by comparison. It has also been estimated that if weapons continue to be fired at the present rate for an indefinite period, the ultimate dose per individual per generation will be 27 mr; this level will be reached in approximately 100 years.

The concentration of Sr^{90} in the ground in the U. K. has been obtained from radiochemical analysis of the rainwater falling at Milford Haven. Based on the results of this analysis an estimate has been made of the possible future levels of Sr^{90} in U. K. soil should weapon testing continue at the present rate for an indefinite period.

All the important computed figures are given in the table at the end of this summary.

Summarised results

Gross Fission Product Activity in Air		
1. Mean conc. of activity in ground level air (1952–1955).	0.5 dpm/m³	Mean activity due to the natural RaB and RaC in atmosphere (measured at Harwell) = 128 dpm/m³.
2. Peak conc. in same period	25 dpm/m³	
3. Mean Gamma dose rate to a person fully exposed to this radiation.	10–3 mr/year	Peak value = 1,800 dpm/m³.
Gamma Dose from Deposited Activity		
1. Estimated dose to the average person in U. K. in a period of 50 years from all bombs exploded before 1.1.56.	1.7 mr	
2. Estimated equilibrium generation dose, reached in about 100 years time.	27 mr	
Strontium 90		
1. Mean conc. in air (1952–1955).	4×10^{-16} μc/cc (2×10^{-6} mpl[1])	1. Mean conc. in drinking water is likely to remain well below that of rainwater because of filtration and absorption by soil.
2. Equilibrium conc. in air (reached in 10 years[2]).	2.4×10^{-15} μc/cc (1.2×10^{-5} mpl)	
3. Mean conc. in rain (1952–1955).	1.7 μμc/l (.002 mpl)	2. Top foot of soil contains about 400 mc/km² of Radium 226, an element with similar chemical properties to Strontium.
4. Equilibrium conc. in rain (reached in 10 years).	8.0 μμc/l (.01 mpl)	
5. Conc. on ground on 1.1.56	4.5 mc/km²	
6. Max. conc. on ground due to bombs already exploded (this will be reached in 1969 approx.).	14 mc/km²	
7. Equilibrium value, reached in approximately 100 years time.	200 mc/km²	

[1] The m. p. l's used are the occupational m. p. l's recommended by the I. C. R. P.

[2] Equilibrium figures are those which will ultimately apply if the present rate of weapon firing continues indefinitely.

ACKNOWLEDGEMENTS

The authors would like to record their gratitude to the Royal Air Force for the part they played in the experimental work, and to Mr. R. G. D. Osmond of the A. E. R. E. outstation at Woolwich who carried out the rediochemical analysis of Sr^{90} in rainwater.

TABLE I.—*Principle components of fission product beta activity at various times*

Mass No.	Isotopes	Percent of Total Activity at Various Times after Fission								
		4d	10d	20d	50d	100d	1y	2y	5y	10y
38	Sr^{89}			5	9	10				
	Sr^{90}							5	15	22
39	Y^{90}							5	15	22
	Y^{91}			6	10	13				
40	Zr^{95}			6	11	15	7			
41	Nb^{95}				9	20	15			
42	Mo [illegible]	12	7							
44	Ru^{103}				7	7				
45	¹Rh^{103}				7	7				
52	Te^{132}	8	5							
53	I^{131}	5	7	6						
	I^{132}	8	5							
54	Xe^{133}	10	11	6						
55	Cs^{137}							5	12	18
56	Ba^{137}							5	12	18
	Ba^{140}	6	10	12	7					
57	La^{140}	5	12	14	8					
58	Ce^{141}		6	10	11	8				
	Ce^{143}	8								
	Ce^{144}					7	27	30	8	
59	Pr^{143}	5	10	12	8					
	Pr^{144}					7	27	30	8	
61	Pm^{147}						6	13	22	16
Total activity ($dpm/10^8$ fissions)		3, 200	1, 200	620	250	110	13	4. 7	1. 4	0. 8

TABLE II.—*Basic data on dose rates from deposited fission products*

[A: Deposit of Specific Activity 1 c/km² measured at time T; B: Deposit of specific activity 1 c/km² measured at 1 day]

1	2	3	4	5
Time measured from instant of explosition **T**	Dose rate in μr/day from deposits A or B		Integrated Dose in μr between T & 50 years from deposits A or B	
	A	B	A	B
1 day	25	25	91	91
2 days	24	9. 3	188	75
3 days	23	5. 8	274	68
4 days	23	4. 2	349	63
5 days	23	3. 2	436	60
7 days	22	2. 2	556	54
10 days	22	1. 5	699	48
15 days	23	1. 1	896	42
20 days	23	0. 81	1, 030	37
30 days	24	0. 58	1, 260	30
50 days	20	0. 27	1, 600	22
100 days	17	0. 10	2, 400	15
200 days	17	0. 034	4, 320	9
1 year	12	0. 009	8, 340	6
2 years	6. 9	0. 0019	17, 200	5
3 years	4. 6	0. 00073		4
5 years	5. 0	0. 00046		4
10 years	5. 8	0. 00031		3
50 years	8. 9	0. 00016		

TABLE III.—*External gamma doses over 50-year period to an unprotected person in the U. K.*

Explosion Series	Cumulative Dose in milli-röntgen					
	Chilton			Milford Haven		
	1st time round	Resid-ual	Total	1st time round	Resid-ual	Total
Nevada, Spring 1951	0.06	0.10	0.16			0.16
Nevada and Russia, Autumn 1951	1.05	.10	1.15	1.06	0.10	1.16
Nevada, Spring 1952	.30	.06	.36	.33	.09	.42
Nevada, Spring 1953	1.13	.32	1.45	.35	.31	.66
Russia, Autumn 1953	.29	.09	.38	.25	.09	.34
Russia, Autumn 1954	.38	.18	.56	.32	.25	.57
Nevada, Spring 1955	.20	.08	.28	.19	.03	.22
Total from above	3.41	.93	4.34	2.50	.87	3.53
Estimated dose from other explosions in same size range			1.0			1.0
Total Integrated Dose from explosions in the nominal range of sizes			5.34			4.53
Pacific, 1954:						
Deposition 13.3.54–16.9.54			.92			.82
Interpolated 20.9.54–6.1.55			.92			.92
Deposition 6.1.55–15.2.55			.38			.61
Extrapolated (para 8.3.2)			20.30			20.30
Pacific, 1952:						
Deposition 31.10.52–20.3.53			.16			.16
Residual, based on comparison with Eniwetok, 1954			6.10			6.10
Russia, 1955:						
Estimated, based on size as announced			1.0			1.0
Tota Integrated Dose from thermonuclear explosions			29.78			29.91
Total Integrated Dose from all explosions prior to 1st January 1956			35.1			34.4

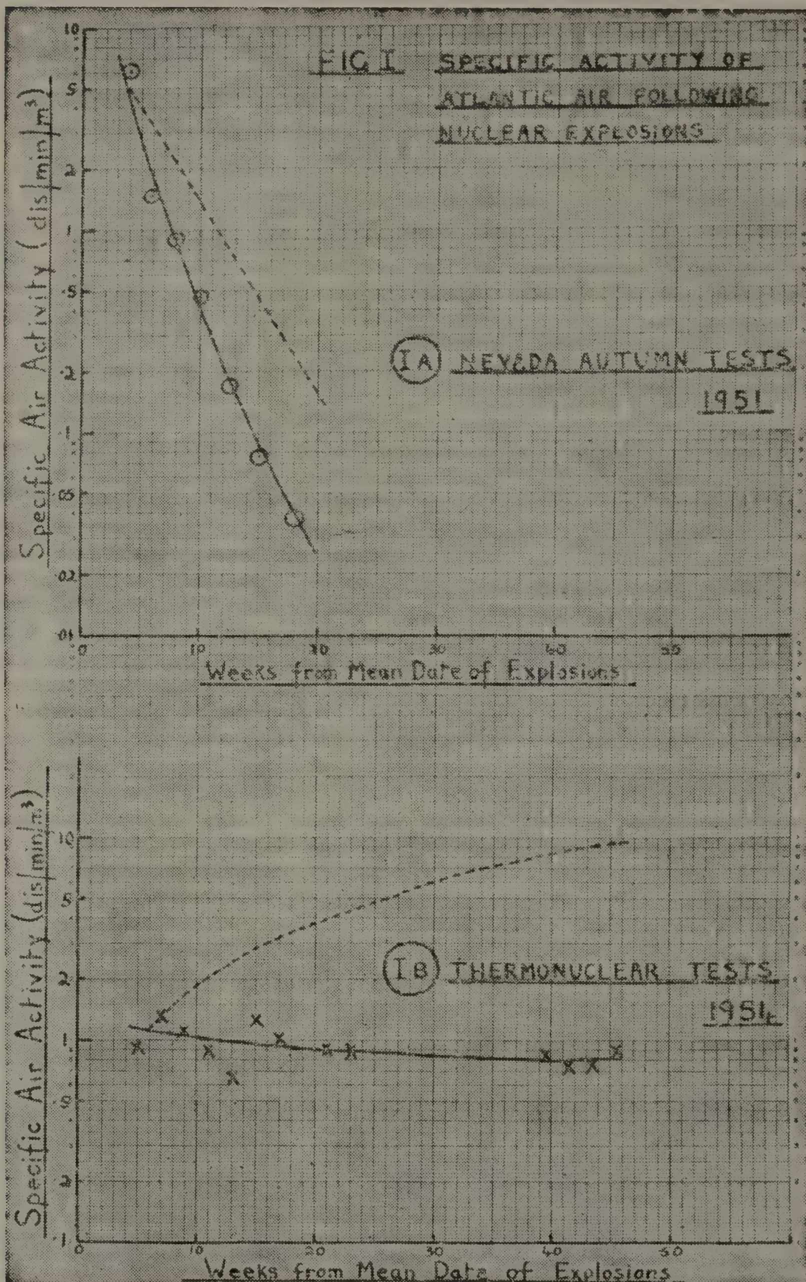


FIG I
SPECIFIC ACTIVITY OF ATLANTIC AIR FOLLOWING NUCLEAR EXPLOSIONS
IA NEVADA AUTUMN TESTS 1951
Specific Air Activity (dis/min/m³)
Weeks from Mean Date of Explosions
IB THERMONUCLEAR TESTS 1954
Specific Air Activity (dis/min/m³)
Weeks from Mean Date of Explosions

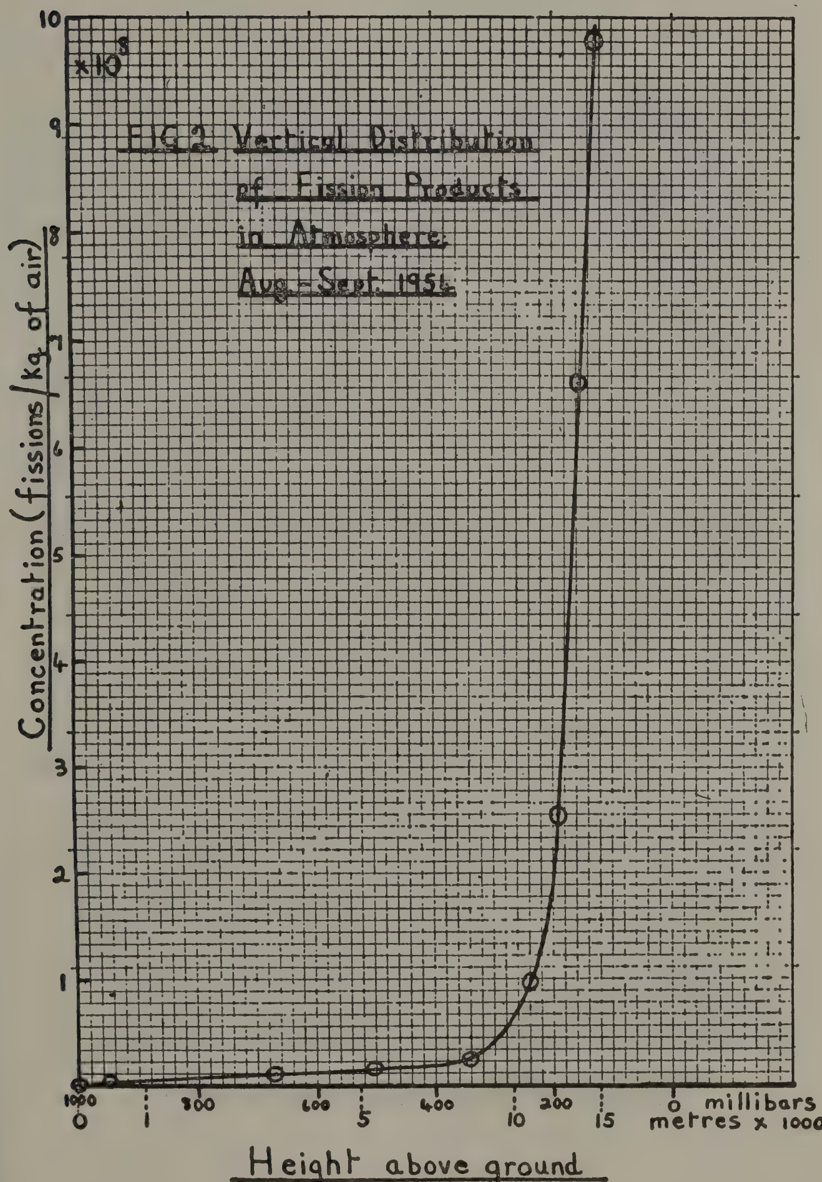


FIG 2. Vertical Distribution
of Fission Products
in Atmosphere
Aug.-Sept. 1956
$\times 10^5$
Concentration (fissions/kg. of air)
10
9
8
7
6
5
4
3
2
1
1000
800
600
400
200
0 millibars
0
1
5
10
15
metres x 1000
Height above ground

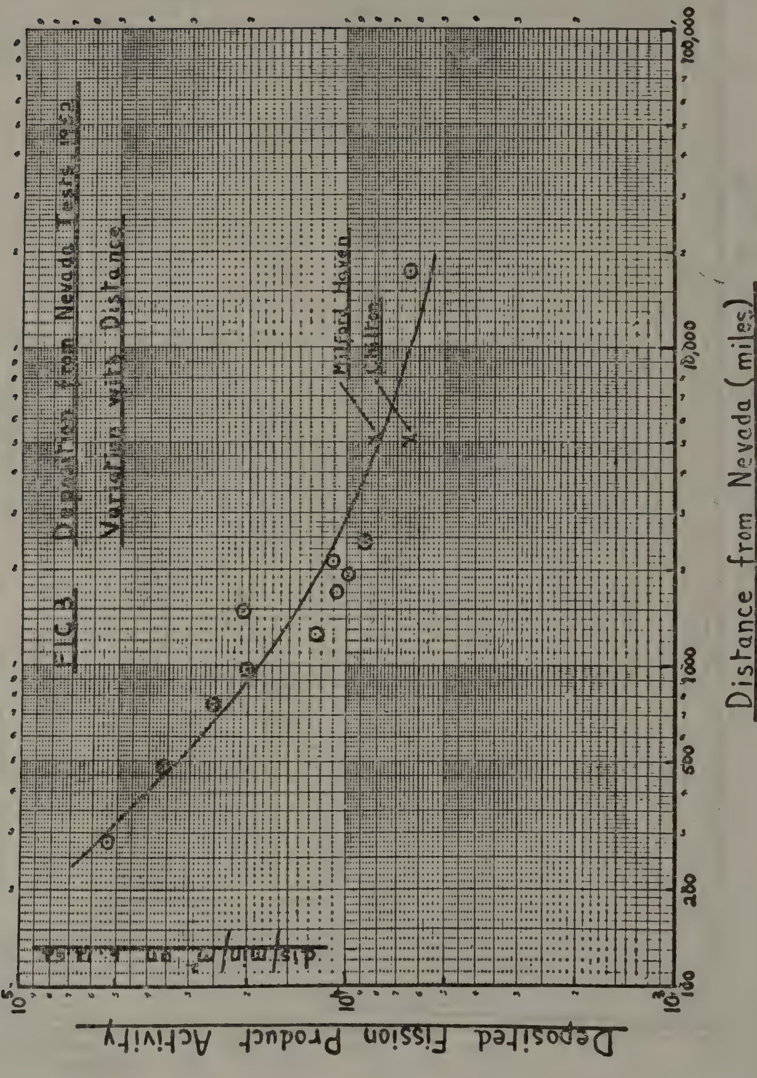


FIG. 3 Deposition from Nevada Tests 1952
Variation with Distance
Milford Haven
Chilton
Distance from Nevada (miles)
Deposited Fission Product Activity
100
200
500
1000
10,000
100,000

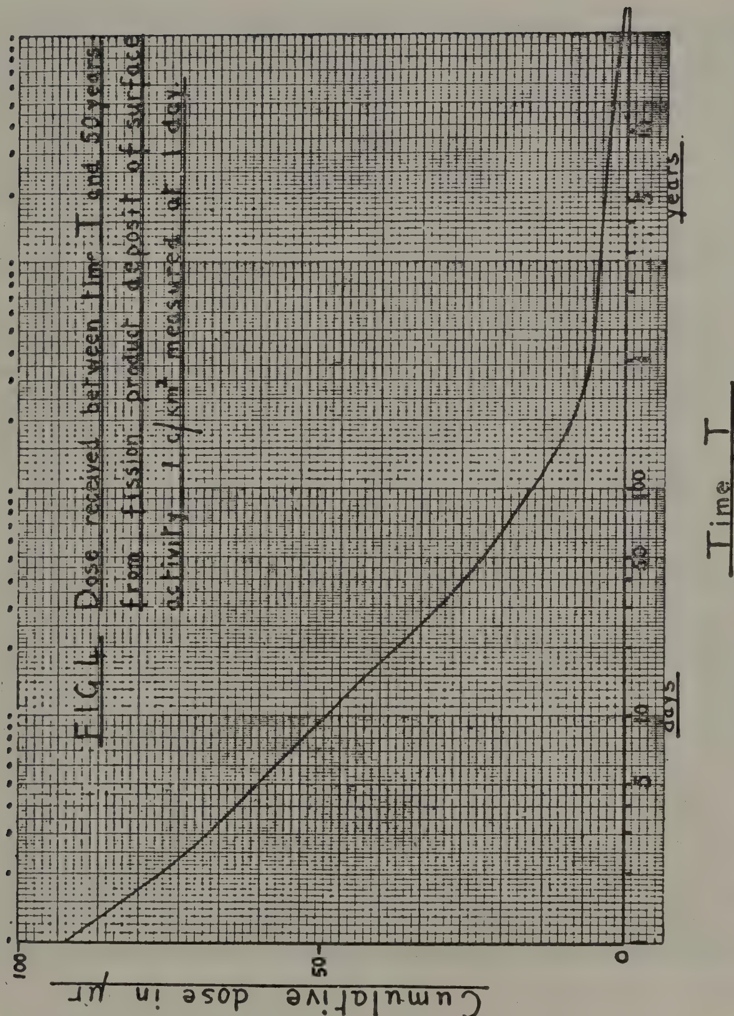


FIG. 4. Dose received between time T and 50 years from fission product deposit of surface activity 1 c/km² measured at 1 day.

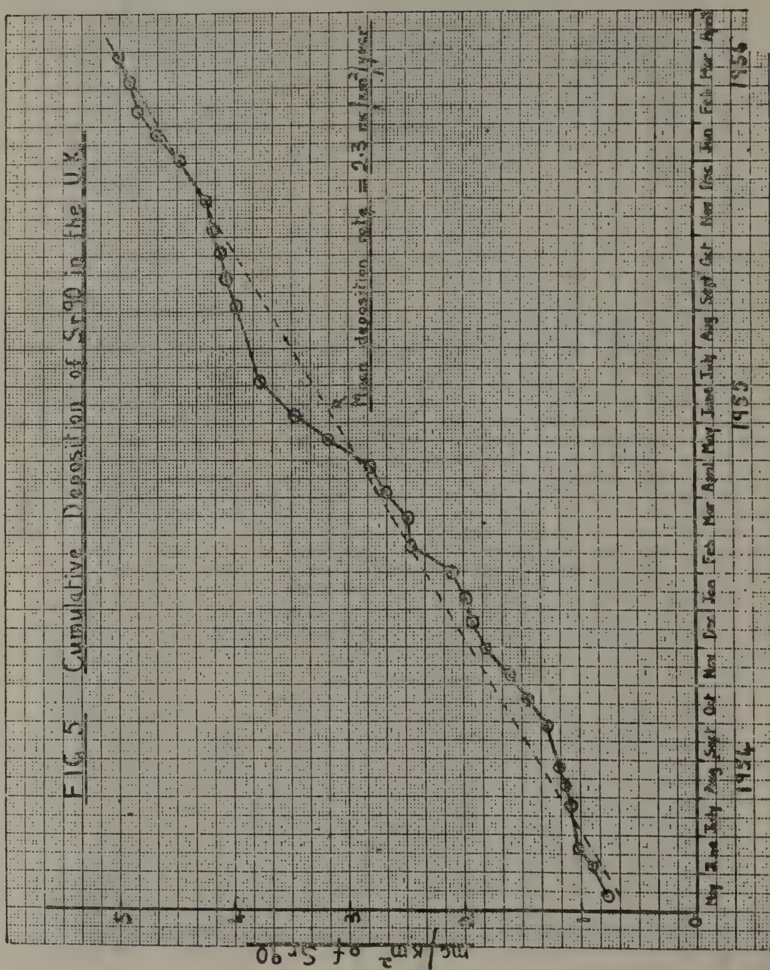


FIG. 5. Cumulative Deposition of Sr90 in the U.K.
Mean deposition rate = 2.3 mc/km²/year
mc/km² of Sr-90
0
1
2
3
4
5
May June July Aug Sept Oct Nov Dec Jan Feb Mar April May June July Aug Sept Oct Nov Dec Jan Feb Mar April
1954
1955
1956

A. E. R. E. HP/R 2056

Radiostrontium Fallout in Biological Materials in Britain

By R. J. Bryant, A. C. Chamberlain, A. Morgan, and G. S. Spicer, Chemistry and Health Physics Divisions, A. E. R. E.

1. INTRODUCTION

The general problem of the nature and effects of fallout of radioactivity from nuclear weapon tests has been considered by the Medical Research Council (1956) in Britain and the National Academy of Sciences (1956) in the U. S. Details of the physical geographical and meteorological aspects have been given by Eisenbud and Harley (1953, 1955, 1956) Stewart et al. (1955 & 1956) and Libby (1956). Libby (loc. cit.) and Martell (1955 and 1956) have described the U. S. work on the analysis of Sr^{90} in soils and biological tissues. Booker et al. (1956) have issued a preliminary report on the early results of the British work, and Bryant, Packman and Spicer (1956) have given an outline of the analytical method used at Woolwich for strontium analysis.

A variety of units have been used for reporting fallout data. It is best to use metric units where possible. The specific activity of Sr^{90} relative to calcium is expressed in micromicrocuries Sr^{90} per gram calcium and denoted by the letters S. U. Thus, in soil, vegetation, milk or bone:—

$$1 \text{ S. U.} = 10^{-12} \text{ curies } Sr^{90} \text{ per gram Ca}$$

The following physical data on the long range fallout from tests are relevant to an understanding of the uptake and distribution in biological tissues.

(i) All nuclear explosions produce Sr^{90} together with other fission products. The fission product activity is usually roughly proportional to the power of the explosion. Since Sr^{90} and Sr^{89} have gaseous precursors, they are not necessarily produced in the same physical form as the bulk of the fission product activity, and their distribution as between local and long range fallout may be different.

(ii) The physical form and distribution of the debris depends on the mode of firing and the power. In general, H bomb fission products are distributed in smaller particles, are found higher in the atmosphere, remain there longer and fallout more uniformly over the earth's surface than A bomb fission products.

(iii) The greater part of the cumulative fallout to date (1956) has been due to H bomb tests. The ratio of H to A bomb fallout is higher in the U. K. than the U. S. The ratio will increase in both countries with time, since much of the activity from H bombs already exploded yet remains to fall to earth, having a mean hold-up time in the stratosphere of the order of a decade. Fallout over temperate latitudes in the Northern Hemisphere is grossly uniform except for an excess within a few thousand kilometres distance from test sites.

(iv) Most of the fallout occurs in rain.

2. OBJECTIVES OF PRESENT WORK

Immediate objectives:

(i) Development and verification of accurate and economical analytical methods for low Sr^{90} levels in soils and biological materials.

(ii) Determination of the cumulative deposited activity, by total strontium analysis of soils, for comparison with estimates from rainfall and other methods.

(iii) Determination of current levels in human and animal tissues and food, on a sufficiently wide statitical and geographical basis.

(iv) Observations of the trend of the above with time.

Long term objectives:

(v) Investigation of the natural history of Sr^{90} in soils, and the uptake from soils to plants.

(vi) Investigation of the foliar retention of fallout by plants.

(vii) Investigation of the metabolic chain:—plants—animal tissue—milk—human tissue.

The practical importance of objectives (v) and (vii) is that this information is needed to enable present levels to be extrapolated to future conditions.

8. SAMPLING METHODS

The British sampling was begun in 1954 in consultation with Dr. L. T. Alexander of the U. S. Dept. of Agriculture. The sampling was and largely still is done by the regional staff of the Ministry of Agriculture, Fisheries & Food.

The two most important factors influencing the levels in biological materials were originally thought to be the amount of rainfall, as affecting the total fallout, and the soil conditions, as affecting the uptake from the soil to plants. It was not realised until later that foliar retention of Sr^{90} might be the dominant factor, at least on some soils. It was decided to use the sheep for sampling animal bone, on the grounds of cheapness and range of habitat.

Five farms were chosen, two in Suffolk with low rainfall and calcareous soil, and three in Wales with high rainfall and well leached acid soil. Details of these sites are given in Appendix A1, numbers 1 to 5.

All the farms carried sheep grazing permanent pastures, but the system of management of hill and lowland sheep differs. On the hill farms in summer the animals range over the mountains, where the grazing areas are often small patches of grass on hillsides interspersed with bracken. The mountain pastures are usually not limed, and may be very deficient in calcium. In the winter the sheep may be brought down to the valleys, or sent away to other areas. Some hay or other supplementary feeding may also be given.

The management of lowland sheep is different. Their grazing is usually controlled so that an area is cropped and then left to make new growth. In winter they may be folded over roots or beet tops, or given hay.

It will be seen that the amounts of rainfall, types of soil and systems of grazing the sheep are all interrelated. It will not be possible to ascribe differences in the levels of Sr^{90} in hill and lowland sheep to one factor or the other, until the underlying processes are understood.[1]

The 1954 sampling system called for soil, vegetation and sheep bone samples taken in the spring or summer. The soil samples were in fact taken from the areas grazed by the sheep—in the hill areas from the mountain pastures—but the vegetation was from hay fields. This had two effects.

(a) Hay in Wales is grown in the mountain valleys where the soil is less acid than on the hills, so that the soil conditions were not the same as for the sheep and soil samples.

(b) Hay in Suffolk is exposed to foliar absorption during the whole of the spring growing season (2 to 3 months) whereas the grazing cycle on the pastures is 2 or 3 weeks.

Thus, for different reasons, the hay samples in neither Wales nor Suffolk were representative of what the sheep were eating during the summer.

The difficulties in arranging a simple but representative sampling system are apparent. In the circumstances it is perhaps surprising that the relative activity in British samples (i. e. the ratio of vegetation; sheep bone; milk; human bone) agrees as well as it does with that found elsewhere, (para. 7.6 below).

In the 1956 routine sampling procedure, of which details are given in Appendix A2, the difficulties (a) and (b) above will be overcome by ensuring that the vegetation samples are from the same plots as the soil samples, and that the period of exposure to foliar retention of activity of the samples is similar to that of the actual pasture. It will not however be possible to distinguish the effects of rainfall, soil type, and grazing systems independently.

The decision to take samples from Welsh hill areas has introduced many difficulties, but it has been valuable in throwing up at an early stage the degree of variation in results which must be allowed for in any policy decisions affecting fallout.

Not much progress can be made with the long term objectives of the work until the relative importance of foliar and root uptake is understood. To study this, 4 sampling positions have been selected within 15 miles of Harwell, with approximately the same rainfall, but on differing soil types. At each site grass is cut from the same area every 3 weeks, and the activity compared with the fallout in rain measured over the same period. It is also planned to do growth experiments using soil from these areas. The results from these investigations are not yet complete.

---

[1] Had cattle been used instead of sheep there would have been the same sort of problem. though not perhaps in so acute a form. Milk cattle are rarely nowadays grazed on unimproved soils, but the systems of management differ considerably in different areas. Moreover dairy cows are often given cattle cake or other foods not grown on the farm.

Milk sampling in the U. K. has so far been confined to a regular sample of dried, skimmed milk from a factory at Yeovil, Somerset. Use of factory dried milk has the disadvantage that it is not referable to any particular pasture, but the advantage that the bulking process smooths out variations from farm to farm. The samples are now being extended to other areas.

4. ANALYTICAL METHODS

4.1 *Methods used in Woolwich laboratories of Chemistry Division A. E. R. E.*—All methods ultimately depend on the separation of active strontium, with added carrier, as nitrate in strong nitric acid solution and include ferric hydroxide and barium chromate scavenges to remove contaminating activities. The separated strontium is stored in the presence of yttrium carrier for at least 14 days and the yttrium precipitated as hydroxide. The yttrium hydroxide is then converted to oxalate for mounting and counting.

Strontium is precipitated as carbonate, mounted and counted after allowing the yttrium to grow in again.

The method described above can be applied directly to bone ashes but for hay and vegetation ash and soil the initial concentration of the calcium and strontium in a suitable form (carbonates or phosphates) is necessary.

Full details of the methods are given in Appendix B.

Appendix B1. The determination of radiostrontium in soil by hydrochloric acid extraction.—This method is aimed at determining the total activity and carrier is therefore added at the extraction stage. The original work was done using 3M acid but 6M is now preferred. Using 3M acid, extraction of calcium tended to be low in some cases although there is no evidence that the total activity was not determined.

Calcium and strontium are precipitated as oxalates from the extract, any iron and aluminum removed as hydroxides and the calcium and strontium finally precipitated as carbonates to which the nitric acid method is applied.

Appendix B2. The determination in soil by fusion with sodium hydroxide and sodium carbonate.—Initial attack with sodium hydroxide and subsequent addition of sodium carbonate is preferred to fusion with sodium carbonate alone as a lower temperature can be used, the attack on the crucible is less and the melt readily disintegrates in water. After removal of silica from the insoluble carbonates, the method is essentially similar to that applied to the acid extract. Carrier is added to the soil before fusion. The maximum amount of sample which can be conveniently dealt with by this method is 100 gm. whereas 500 gm. can be used in the acid extraction.

Appendix B3. The determination in soil by ammonium acetate extraction.—As the aim here is to determine strontium available to the plant, carrier is not added until the extract is separated from the soil. Calcium and strontium are precipitated as carbonates which are treated as in the acid extraction method.

Appendices B4 and 5. The determination in animal and human bone ashes.—The nitric acid separation is applied directly. In the case of human bones where the amount of sample is limited and the activity low it is impracticable to measure the separated yttrium and the strontium + yttrium count is used. In a few favourable instances it has been possible to check the accuracy of this procedure by counting the separated yttrium.

Appendix B6. The determination in dried milk.—Direct application of the nitric acid separation usually gives low strontium yields. The calcium and strontium carrier are therefore concentrated by an initial phosphate precipitation.

Appendix B7. The determination in vegetable ash.—The ash is treated for the removal of silica and calcium and strontium precipitated as phosphates before the application of the nitric acid method.

Chemical yields for strontium vary from 60–80% for bone, vegetable and milk ashes and high calcium soils and from 40–60% for low calcium soils.

The yttrium yields are about 95%.

Work on this project is done in laboratories remote from others dealing with activity and specially reserved for this purpose. Frequent blanks are carried through the procedures to check the reagents, carriers etc.

All results obtained using these methods in the Woolwich laboratories of Chemistry Division will be described as Woolwich (or W) results.

4.2 *Analytical Methods used in the Health Physics Laboratory, A. E. R. E. Harwell.*—In this laboratory the determination of radiostrontium is at present confined to vegetation, animal bone and milk samples.

The chemical methods used are based on the same principles as those used at the Woolwich Outstation and only differ in minor details. They are described briefly below:

(a) The determination in ashed vegetation.

The calcium and strontium are extracted from the ashed vegetation by three successive leachings with hot 6M hydrochloric acid. The iron and aluminum are removed from the combined leachings as the hydroxides, after which the calcium and strontium are precipitated as the carbonates and purified by precipitation. Strontium carrier is added to the purified carbonates before the fuming nitric acid separation of strontium from calcium. Subsequently other radio isotopes are removed by barium chromate and yttrium hydroxide scavenges and the purified strontium stored with yttrium carrier for 18 days. Finally the yttrium is separated, mounted as the oxalate and the decay followed. The strontium is precipitated as the carbonate, weighed, mounted and the decay followed after the yttrium has grown in, in order to determine the $strontium^{89}$.

(b) The determination in bone ash and milk ash.

These are both treated in the same way and after the addition of strontium carrier, the fuming nitric acid procedure is applied directly. Otherwise the methods are identical with that for ashed vegetation.

The counting methods are identical with those used by the Woolwich Laboratories and described in the next section.

The counters were calibrated independently and interchange of sources with Woolwich has shown good agreement.

5. COUNTING METHODS (WOOLWICH LABORATORY)

Precipitates for counting are filtered on 2.1 cm. filter papers in perspex filter holders. After suitable washing and drying the papers are mounted on aluminium trays using a dilute solution of Gelva (polyvinyl acetate resin).

Three types of counter are in use.

(a) An anti-coincidence set-up with special low background G.M.4. counting tubes (7mgm/cm^2 window) surrounded by a complete ring of 9 Type G.E.21 brass bodied guard tubes. The assembly is shielded by 4 inches of steel and the background is 0.4–0.5 cpm. depending on the G.M.4. tube. The total, coincidence and anti-coincidence counts are scaled and the accuracy of the anti-coincidence count is thus continuously monitored. The efficiency for counting Y^{90} with 25 mgm. sources is about 25%.

(b) Three position anti-coincidence counters in which the background and two samples can be measured. These are normally set to count one hour in each position and the individual hourly counts and total counts from each position are recorded. The counting tubes are either E.H.M.2 (2 mgm/cm^2 window) or special G.M.4 tubes with 13 Type G.M.5 glass envelope guard tubes arranged at 180°. The assemblies are shielded by 2 inches of lead and normal backgrounds and efficiencies for Y^{90} counting are

E.H.M.2	1.5–2.0 cpm.	efficiency c. 30%
G.M.4	1.0–1.3 cpm.	efficiency c. 25%

(c) Standard counting arrangement using E.H.M.2 counting tubes and 2 inch lead shield with a background of 7 cpm. The efficiency is about 20% and this set up has been modified to permit the direct counting of bone ashes.

Type (b) is used for most of the work, type (a) being reserved for the lowest activities and type (c) for the more active animal bones.

The counters are calibrated using virtually carrier free solutions standardised by the Isotope Division, A. E. R. E. Suitable aliquots are taken, and precipitated with appropriate amounts of carrier, mounted and counted. From these results efficiency versus source weight tables (or curves) can be prepared which are used in converting cpm. to dpm.

When significant amounts of $strontium^{89}$ are present the $Sr^{90}+Y^{90}$ contribution in a mixed $Sr^{89}+Sr^{90}+Y^{90}$ source is calculated from the Y^{90} count and the Sr^{89} count obtained by difference.

With the exception of the results for human bones, the $strontium^{90}$ content is based on the counting of the $yttrium^{90}$ source, the radiochemical purity of which is checked by following its decay. The decay of the mixed strontium and yttrium sources is also checked.

6. INTERCOMPARISON SAMPLES EXCHANGED WITH U. S.

6.1 *Bone ash samples sent to U. S.*—U. K. sheep bone ash samples, from yearling animals killed in the spring of 1955 were sent to U. S. and analysed at Chicago. Results of three laboratories (two U. K. and one U. S.) are as follows, expressed in S. U.

TABLE I.—*Intercomparison of U. K. bone ash*

U. K. Ref	A. E. R. E. results		U. S. results [1]
	Harwell	Woolwich	
B3	3.0	13	31.4
B4	12.7	15	13.1
B5	5.7	5.7	5.2
B6	56	59	60.6
B7	13.4	17	18.3

[1] Martell (1955). These results are also given in Libby (1956) figure 3b, but are there wrongly ascribed to 1954;

Agreement is good, except for one sample.

6.2 *Intercomparison samples suplied by Health & Safety Laboratory, New York.*—Samples of sheep bone, hay and milk ash, and also of soil, collected from the New York area in September/October 1955, have been analysed by the A. E. R. E. (Woolwich Outstation) New York, Chicago and Pittsburgh laboratories as follows, results being in S. U.

TABLE II.—*Intercomparison of HASL samples*

	Woolwich	New York	Chicago	Pittsburgh
Sheepbone	5.6	5.8	4.45	6.98
	5.6	5.1	5.14	4.45
Hay	19.5	20		19.7
	19.8			17.9
Milk	3.1	3.3	2.4	2.4
	3.0	3.1		2.6

Agreement on the bone, hay and milk samples is excellent, especially between the Woolwich and New York laboratories.

On the soil sample, Woolwich have given results as follows but results from the other laboratories have not yet been received.

TABLE III.—*Woolwich results on HASL soil cample*

Initial attack	Sample weight	%Ca	Sr^{90} activity		S. U.
			dpm/ft²	$\mu\mu c/m^2$	
$NaOH+Na_2CO_3$ fusion	100 gm	0.39	620	3050	17.3
2×500 ml. 3M HCl	200 gm	0.28	640	3150	25
	400 gm	0.24	580	2850	26

7. RESULTS ON U. K. SAMPLES

A check list of results on all British samples on which analysis is complete (whether analysed in the U. K. or the U. S.) is given in Appendix C.

7.1 *Soils.*—The 1955 soil samples have been analysed at Woolwich, by three methods, ammonium acetate extraction, hydrochloric acid extraction, and fusion with sodium carbonate. The agreement on total Sr^{90} between the last two methods is very fair, taking into account the low specific activity (20 to 50 dpm/kg soil) and the presence of some activity in particulate form, which makes it difficult to ensure that samples are representative.

There seems to be significantly more total Sr^{90} in the top 4″ of the Vyrnwy and Cwmystwyth soils than in those from the Suffolk stations. This may well be associated with the high rainfall (see Appendix A), but it must not be forgotten that these soils are very well leached. Stewart et al (1956) have measured the Sr^{90} fallout in rain at Milford Haven, Pembrokeshire over 3 weekly periods since the spring of 1954. Without assuming anything about the absolute levels found, their curve can be used to normalise soil estimates at different dates in 1955 to a fixed date which will be taken as 1st October, 1955. The U. K. soils were sampled in late March and early April. The factor of increase on Stewart's curve between 1st April and 1st October is 1.5. In Table I the results of the 1955 U. K. soil samples (fusion and HCl results averaged) from Appendix Cl are multiplied by the factor 1.5 and compared with three sets of results on U. S. soils, namely:—

(i) The soil taken at Ithaca N. Y. in September, 1955, depth 0–2″, by HASL for intercomparison purposes. The Woolwich results on aliquots of this soil were given in para 6.2 above.

(ii) The 17 U. S. soils (0–2″) sampled between 23rd September and 20th October, 1955 and analysed for Sr^{90} by HCl extraction by Hardy and Morse (Libby, 1956, Table 6). The mean of these samples was 2800$\mu\mu c/m^2$ (578 dpm/ft²) and the two New York samples among them averaged 2200$\mu\mu c/m^2$ (450 dpm/ft²).

(iii) The 6 Illinois/Wisconsin soils sampled by Alexander in late September 1955, extracted with ammonium acetate at Beltsville and analysed by Martell (1956) with mean Sr^{90} activity of 4800±900$\mu\mu c/m^2$ (970±180 dpm/ft²). These soils were sampled to depth 8″, 80% of the activity being found in the top 2″ of unploughed soil.

TABLE IV.—*Sr^{90} in soils at 1st October 1955*

	Annual rainfall (inches)	Soil depth	Sr^{90} activity	
			$\mu\mu c/m^2$	mc/mile²
U. K. Measurements:				
Cwmystwyth & Vyrnwy	80	0–4″	4000	10.0
Talgarth	35	0–4″	2500	6.5
Suffolk	25	0–4″	1300	3.3
Ithaca, N. Y.	42	0–2″	8070	8.0
U. S. Measurements:				
U. S. (av)	------------	0–2″	2800	7.2
New York	42	0–2″	2200	5.7
Wis/Ill	33	0–8″	4800	12.4

There is good agreement between the total Sr^{90} in the top 4″ of Cwmystwyth and Vyrnwy soils and the absolute value of the cumulative rainfall activity at Milford Haven, Pembrokeshire. Stewart et al (1956), give the total corresponding to 1st October 1955, as 4100 $\mu\mu c/m^2$ (10.5 mc/mile²). However, the profile of Sr^{90} activity in the Welsh soils is so far unknown, and too much reliance cannot be placed on this comparison.

7.2 *Vegetation Samples.*—The results are given in Appendix C2. To express the results in S. U. ($\mu\mu c/Sr^{90}$ per gram Ca) is appropriate as long as uptake from the soil is considered the major mode of contamination. When foliar retention is considered $\mu\mu c$/kg or $\mu\mu c/m^2$ of ground are sometimes more helpful units.

The notable feature of the 1955 samples (D4 to D13 and D20) was the uniformity of the results as between samples, despite the widely varying geographical and soil status of the sites. With two exceptions, all lay in the range 25 to 53 S. U. All were mature hay samples and therefore all had approximately the same period of growth and exposure to fallout.

The five samples D14a to D18/2 were taken at Chilton, Berkshire, where the soil is very calcareous (circa 100 grms, Ca per kg. soil). Rough grass, some of it dead, was collected from an ungrazed area of the former airfield. The activity ranged from 24 to 64 S. U. as compared with a S. U. value in the soil of about 0.2, thus strongly suggesting foliar retention on the dominant mode. About 25 percent of the vegetation activity was removable by washing with distilled water. Expressed per unit area of ground, these samples ranged from 50 to 85 $\mu\mu c/m^2$. From the rainfall measurements of Stewart et al (1956) the rate of fallout of

Sr^{90} during the early spring of 1956 was about 200 $\mu\mu c/m^2$/month. The Chilton results could therefore be explained without reference to soil uptake if, for example, 25% of the fallout was retained on the leaves for an average period of about 6 weeks.

Samples D24A and D25A were taken at similar airfield sites after the new season's growth of grass was available. The levels in terms of S. U. are a little lower than those of the preceding samples. In terms of $\mu\mu c$/kg and $\mu\mu c/m^2$ they are considerably lower, reflecting the lower calcium content of the spring flush of grass.

Samples D21 and D22 were taken at Cwmystwyth from a hillside sheep run similar to that on which the sheep B6 and B10 had grazed. The season was late, and the grass sparse. The activities of 370 and 510 S. U. found in the grass are in about the same ratio of 10:1 to the general level at that time as were the sheep bones taken in 1955 from Cwmystwyth to the general level in sheep bones.

7.3 *Sheep bones.*—The results of Sr^{90} analysis on 21 long bones from British sheep are given in Appendix C3.

The results for lowland sheep in 1955 and early 1956 were fairly uniform, the range of 8 samples being from 7.5 to 15.4 S. U. and the median 14 S. U. This compares with 15 S. U. estimated by Martell (1956) for the Chicago area in late 1955.

With hill sheep the results are more variable, levels as high as 59 and 183 S. U. (Cwmystwyth) 35 S. U. (Welshpool) and 65 S. U. (Haslingden) having been found. As shown in the section on sampling, it is not yet possible to say what weight to attach to excess rainfall, soil type or grazing habits in contributing to these results.

7.4 *Milks.*—Seven samples of dried milk made at a factory at Yeovil, Somerset, during the period March-July, 1955 have been analysed at the Health and Safety Laboratory, New York Operations Office, AEC, through the kindness of Dr. J. H. Harley. The mean level was 3.2 S. U. and this compares with means of 2.5 and 3.3. S. U. respectively for New York and Chicago milks over the same period as deduced from Libby (1956, Figure 1c). Similar samples dated April, 1955 and March 1956 respectively analysed at Harwell gave 4.3 and 3.8 S. U.

In samples from all three localities (New York, Chicago and Yeovil) there is evidence of a peak of Sr^{90} activity in May and June 1955 (fig. 1). Fallout was relatively heavy about this time, and the effect of this may have been accentuated by the change in the feed of the cows from hay and similar stored foods to open grazing at that time of year.

7.5 *Human bones.*—28 British human bone samples dating from October 1955 to February 1956 have analysed at Woolwich to date. Details are given in Appendix C5 and a graph of activity against age at death in Fig. 2.

The earlier samples were mostly ribs, but in future femurs will be used whenever possible. Geographically the samples are mainly from the Midlands and South East of England, with a few from Carlisle to represent the West Coast.

The highest value found so far is 1.3 S. U. in an eleven weeks old child (Carlisle), closely followed by 1.2 and 1.1 S. U. in two Birmingham one year olds, and 1.05 S. U. in a 3½ year old from Dudley. Bones from one stillborn infant have been analysed, and gave 0.45 S. U.

Adult bones show consistently low values of 0.2 S. U. or less. Much of the calcium in adult bones was of course laid down before fallout became significant.

The range of levels in British bones is almost indistinguishable from that found in the U. S. and reported by Libby (1956). The maximum in his series is 1.7 S. U. with several others just over 1 S. U. Two 1955 stillbirths from New England show 0.4 and 0.5 S. U.

7.6 *Relative levels in vegetation, bone and milk.*—The results of three series of samples are given below, median values being given except for the child, for which the maximum is used. For comparative purposes the results have been normalised to make the vegetation level equal 100, but the results in S. U. are also given in parentheses.

The three series are:

(1) U. S. results of October 1953, as given in Table I and II of Bugher (1955) and in Table 1 of Libby (1956).

(2) U. K. results of 1955

(3) The New York samples of September/October 1955, referred to in paragraph 6.2 above, with human bone results from Libby (1956).

Also for comparison, experimental results of Comar (1956) are included.

TABLE V.—*Relative Sr^{-90}/Ca ratios normalised to vegetation=100*

[S. U. values in parentheses]

	Vegetation	Animal bone	Milk	Child (max.)	Stillbirth
(1) U. S. 1953	100	30	16	8	1.4
	(9)	(2.7)	(1.4)	(0.7)	(0.13)
(2) U. S. 1955	100	28	15	8.5	2.0
	(20)	(5.6)	(3.0)	(1.7)	(0.4)
(3) U. K. 1955	100	40	9	3.7	1.3
	(35)	(14)	(3.3)	(1.3)	(0.45)
Comar (exptl)	100	37	13	-----	-----

8. SUMMARY AND CONCLUSION

The good agreement between independent determinations by different laboratories (paragraph 6) shows that the analytical methods of determining Sr^{90} in biological materials are adequate. The error is small in comparison with the biological variations between samples.

The soil determinations cannot yet be considered final, but it is unlikely that the estimation of total Sr^{90} by the hydrochloric acid and fusion methods is seriously in error.

There is good evidence that from the spring of 1955 onwards the levels of Sr^{90} in biological materials in the U. K. and the U. S. have been similar. This is particularly true of milk samples and human bones, but appears also in the results for animal bone and vegetation.

As Libby (1956) has stated:

"A Sr^{90} fallout probably derived from megaton weapons and nearly uniform over the world, except for local effects due to rainfall variations and to fallout from submegaton weapons, seems clearly established."

9. ACKNOWLEDGEMENTS

We are greatly indebted to Mr. K. H. Jones and all the members of the staff of the Ministry of Agriculture, Fisheries & Food who have helped in the collection of samples for this work. In particular Dr. Rice Williams and Mr. T. H. Caldwell have given most helpful advice and have provided invaluable assistance in arranging subsidiary experiments.

10. REFERENCES

Booker D. V., Bryant F. J., Chadwick R. C., Chamberlain A. C., and Morgan A., (1956) "Radiostrontium Fallout in Soil, Plant and Bone up to December, 1955." A. E. R. E. HP/M. 108.

Bryant F. J., Packman G., and Spicer G. S., (1956) Paper to XVth International Congress of Pure & Applied Chemistry, Lisbon.

Bugher J. C., (1955) "Effects of Fission Material on Air, Soil and Living Species" Paper to Harwell SPAR Conference.

Comar C., (1956) "Radioisotopes in the Study of Mineral Metabolism" (in the press) (quoted by Libby, 1956).

Eisenbud & Harley (1953) "Radioactive Dust from Nuclear Detonations", Science *117*, 14 1 (1955) "Radioactive Fallout in the United States", Science *121*, 677 (1956) "Radioactive Fallout through September 1955" Science *124*, 251

Libby W. F., (1956) 'Radioactive Strontium Fallout". Proc. Nat. Acad. Sci. *42*, 365.

Medical Research Council (1956) "The Hazards to Man of Nuclear and Allied Radiations" London H. M. S. O.

Martell, E. A., (1955) Project Sunshine Bulletin No. 11, and Supplements 1 and 2, University of Chicago, Chicago, Ill.

Martell E. A., (1956) "The Chicago Sunshine Method" University of Chicago, Chicago, Ill.

National Academy of Sciences (1956) "The Biological Effects of Atomic Radiation." Washington (1956).

Stewart N. G., Crooks R. N., and Fisher E. M. R., (1955) "The Radiological Dose to Persons in the U. K. due to debris from Nuclear Test Explosions." AERE HP/R. 1701.

(1956) Revised version AERE HP/R. 2017.

Appendix A1

Principal U. K. sampling sites

No.	Site	Soil	Total Ca in soil gms/kg dry wt.	Altitude (feet)	Annual rainfall (inches)
1	Earl Soham Suffolk	Boulder clay	16	90	25
2	Boulge Suffolk	Sandy clay loam	3	120	25
3	Talgarth Brecon	Free draining soil on old red sandstone	2.5	1,050	35
4	Cwmystwyth Cardigan	Peat on shale (free draining)	0.3	1,200	80
5	Vyrnwy Montgomery	Peat on shale (free draining)	2	1,100	78
6	Boxworth Cambridge	Dark brown loam with chalk particles		157	20
7	Norwich Norfolk	Sandy loam with gravel		85	25
8	Princetown Devon	Sandy peat on granite		1,300	85
9	Rookhope Durham	Peaty sandy loam with podsol		1,600	56

NOTE.—All these farms carry sheep on permanent pastures. Nos. 1 to 5 were the original (1954) sites. From 1956 onwards Nos. 4 to 9 will be used, supplemented with additional samples as required.

Appendix A2

DETAILING OF SAMPLING PROCEDURE

1. Soil and vegetation

Samples or herbage and soil will be taken in the first fortnight of July each year, beginning in 1956. Half acre sites will be used on the chosen sampling farms, the same half acre being used each year.

Grass.—On the half acre, 10 plots, as near to one square yard as possible will be selected. The grass from each of the 10 plots will be cut with shears to about "lawn-mower" length, i. e. short enough to get the vegetation that a sheep would graze but long enough to avoid contaminating the grass sample with soil. The grass from the 10 sites will be bulked. If the bulk sample falls appreciably below 5 lbs. in weight, more yard plots will be cut, the total number being recorded.

Mat.—After the grass has been cut, a core of any grass mat there might be present will be taken from each of 10 plots, and bulked. A sampling tool will be used giving a 4′′ diameter core.

Soil.—A 4′′ deep core of soil will be taken from the spot where the mat had been removed. In practice, a core of mat and soil exceeding 4′′ in depth may be taken, the mat removed and kept separate and the top 4′′ of the core retained as the soil sample. The sample from the 10 plots may be bulked. The exact surface area must be noted.

In each future year, new square yard plots as near as possible to the original plots, but not overlapping them, should be chosen.

2. Sheep

The sheep to be taken should be in the region of 15 months old. It will have to be accepted that some sheep would spend part of their lives on pastures possibly remote from the sampling site. It is important, however, that the sheep should have spent at least the last few months in grazing pastures on or near the sampling site. Long bones only will be required.

Bones should be cleared of surface meat but need not be otherwise treated.

3. Other information

Samplers will be asked to provide:

(a) Details of terrain, rainfall, etc.

(b) Details of sheep management (i. e. whether sheep had grazed in sampling pasture continuously, and breed).

(c) Photographs of the site, and a "close-up" of the herbage.

4. Other matters

(a) Reasonably level plots should be chosen for herbage and soil samples.

(b) The plots need not be fenced off in any way unless the yield of grass per square yard is likely to be less than ½ lb. In any case, cages should not be put in position more than 3 weeks before the sampling.

(c) Sites should remain unploughed for at least 5 years.

* * * * * * *

APPENDIX C

Check list of results

SOILS

Ref.	Date	District	Ca in soil gms/kg	Sr^{90} activity to depth 4″					
				NH₄Ac		HCl		Fusion	
				$\mu\mu c/m^3$	S. U.	$\mu\mu c/m^3$	S. U.	$\mu\mu c/m^2$	S. U.
UK9	3/54	Earl Soham	26	370	0.7	210	0.15	150	0.2
UK10	3/54	Earl Soham	16.5	270	0.8	440	0.7	440	0.7
UK11	3/55	Earl Soham	16	≤410	≤0.9	880	0.6	1020	0.7
UK12	3/55	Earl Soham	8	310	0.6	690	0.9	<340	0.6
UK13	3/55	Boulge	3	≤930	≤2.7	1300	3.3	1000	2.7
UK14	3/55	Earl Soham	26	580	0.7	510	0.15 0.16	780	0.2
UK15	3/55	Earl Soham	11	490	0.8	920	0.8	1000	1.0
UK16	3/55	Vyrnwy	2	1700	16	2900	22	2900	22
UK17	3/55	Vyrnwy	2	1300	11	2500	16	2900	18
UK18	3/55	Cwmystwyth	0.3	1300	160	2300	150	2500	170
UK19	3/55	Cwmystwyth	0.3	1500	280	2800	150 180	2700	170
UK20	3/55	Talgarth	2.5	11 0	6.9	2000	8.1	1300	4.9

NOTE.—1000 $\mu\mu c/m^2$ = 1 mc/1m² = 2.6 mc/mile² = 204 dpm/ft².

VEGETATION

Ref.	Date	District of sampling	Sample	Sr^{90} Activity			Lab	Remarks
				S. U.	$\mu\mu c/kg$	$\mu\mu c/m^2$		
UK/D1	8/54	Earl Soham Suffolk	Hay	5.8	35		H	
D2	9/54	Talgarth, Brecon	Hay	7.8	10		H	
D4	6/55	Boulge, Suffolk	Hay	52	140		H	
D5	6/55	Boulge, Suffolk	Hay	43	110		H	
D6	6/55	Boulge, Suffolk	Hay	41			H	
D7	7/55	Earl Soham Suffolk	Hay	5.5	24		H	
D8	7/55	Earl Soham Suffolk	Hay	29	110		H	
D9	7/55	Earl Soham Suffolk	Hay	25	100		H	
D10	7/55	Earl Soham Suffolk	Hay	33	120		H	
D11	8/55	Vyrnwy, Montgomery.	Hay	35	250		H	
D12	8/55	Vyrnwy, Montgomery.	Hay	51	335		H	
D13	8/55	Cwmystwyth Cards.	Hay	53	270		H	
D20	8/55	Talgarth, Brecon	Hay	8.3			H	
D14A	12/55	Chilton, Berks	Grass	24			H	Rough grassland, including dead grass from previous year's growth.
D14B	2/56	Chilton, Berks	Grass	40		85	H	
D16/3	3/56	Chilton, Berks	Grass	64	490	80	H	
D17/2	3/56	Chilton, Berks	Grass	61	360	57	H	
D18/2	3/56	Chilton, Berks	Grass	44	380	50	H	
D24/A	5/56	Culham, Oxon	Grass	41	165	21	H	Rough grassland, new growth.
D25/A	5/56	Chilton, Berks	Grass	37	185	13	H	
D21	4/56	Cwmystwyth Cards.	Grass	370	1010	84	H	Sparse grass from sheep run.
D22	4/56	Cwmystwyth Cards.	Grass	510	1100	290	H	
US	9/55	Ithaca, N. Y.	Hay	19.7			W	Intercomparison sample.

SHEEP BONES

Ref.	Date	Age (years)	District of origin	Sr^{90} Activity (S. U.)		Remarks
				Woolwich	Harwell	
UK/B1	3/54	1	Earl Soham, Suffolk	1.9	1.4	Lowland farm.
B2	3/54	1	Earl Soham, Suffolk	1.7	1.2	Lowland farm.
B3	3/55	1	Boulge, Suffolk	13	3	Lowland farm.
B4	3/55	1	Boulge, Suffolk	15	12.7	Lowland farm.
B5	3/55	1	Talgarth, Brecon	5.7	5.7	Hill farm.
B6	3/55	1	Cwmystwyth, Cards	59	56	Hill farm.
B7	4/55	1	Vyrnwy, Mongtomery	17	13.4	Hill farm.
B8	4/55	----	U. S. (Alexander)	4.1	----	
B9	4/55	----	U. S. (Alexander)	4.4	----	
B10	10/55	17/12	Cwmystwyth, Cards	----	183	Hill farm.
B12	12/55	16/12	Welshpool, Mont	35	----	Hill farm.
B13	12/55	10/12	Melbourn, Cambs	11.2	----	Lowland farm.
B14	12/55	18/12	Clun, Shropshire	15.7	----	Hill farm.
B15	1/56	9/12	Petworth, Sussex	15.4	----	Lowland farm.
B16	2/56	10/12	Croft, Leicester	13.9	----	Lowland farm.
B17	2/56	9/12	Market Harborough, Leics	15.3	16.2	Lowland farm.
B18	2/56	12/12	Petworth, Sussex	15.6	16.6	Lowland farm.
B19	3/56	12/12	Grantham, Lincs	7.8	----	Lowland farm.
B20	4/56	12/12	Haslingden, Lancs	----	65	Hill farm.
B21	4/56	12/12	Talgarth, Brecon	----	24	
U. S	9/55	7/12	Ithaca, N. Y	5.6	----	Intercomparison sample.

MILK

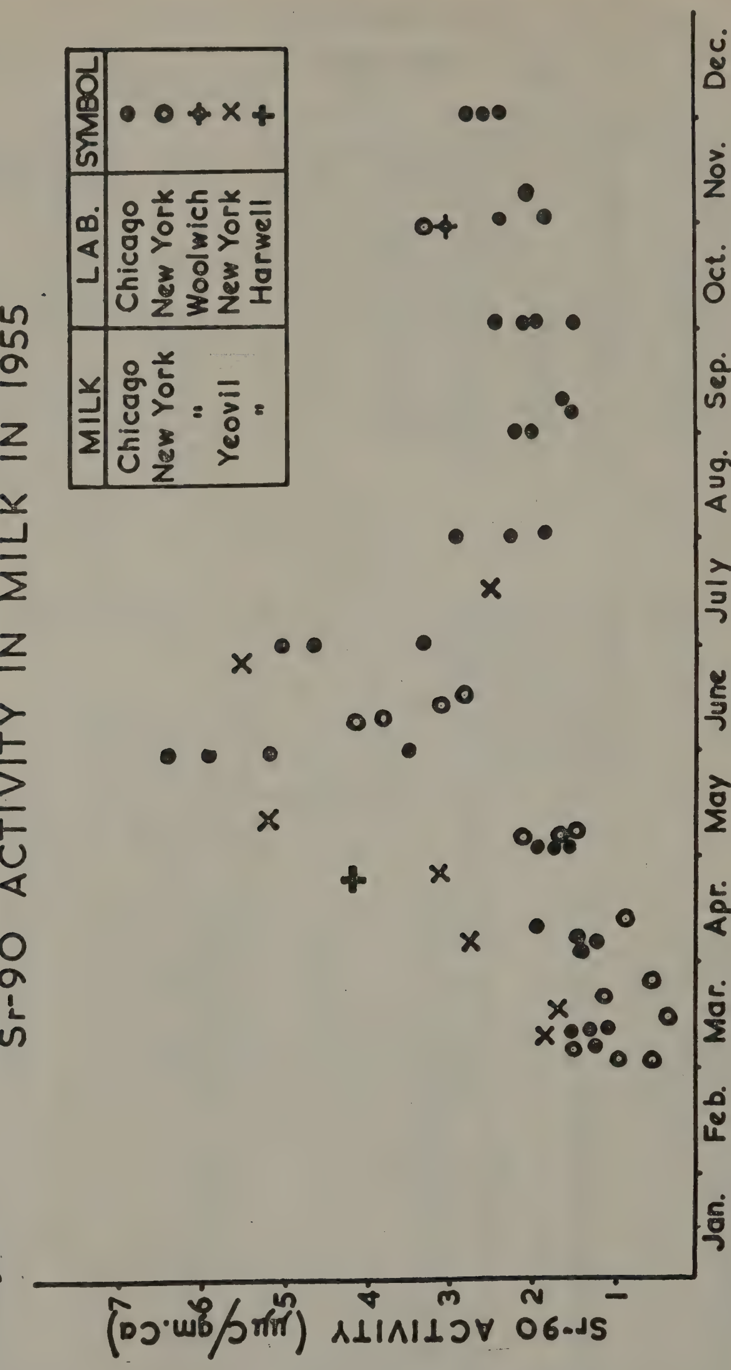
Ref.	Date	District	Sr^{90} Activity S. U.	Lab	Remarks
C1	3/55	Yeovil, Somerset	1.8	N. Y.	Some confusion over references of these samples, but all spring 1955.
C2	3/55	Yeovil, Somerset	1.7, 1.8	N. Y.	
C6	4/55	Yeovil, Somerset	2.6, 2.8	N. Y.	
C7	4/55	Yeovil, Somerset	----	----	
C11	4/55	Yeovil, Somerset	3.3, 2.8	N. Y.	
C12	5/55	Yeovil, Somerset	5.3	N. Y.	
C16	6/55	Yeovil, Somerset	5.3, 5.8	N. Y.	
C19	7/55	Yeovil, Somerset	2.3, 2.8	----	
C9	4/55	Yeovil, Somerset	4.1	H	
	10/55	New York, N. Y	3.1, 3.0	W	Intercomparison sample.
	10/55	New York, N. Y	3.3, 3.1	N. Y.	
	3/56	Yeovil, Somerset	3.8	H	

HUMAN BONES

Ref.	Date	Age (years)	Bone	District	Sr^{90} Activity [1] (S. U.)
HB1	10/55	12	Ribs	Swindon	0.2 ±0.05
HB2	10/55	27	Ribs	Swindon	0.15±0.05
HB3	10/55	40	Ribs	Swindon	0.05±0.05
HB4	10/55	38	Ribs	Reading	0.05±0.05
HB5	10/55	6	Ribs	Swindon	0.15±0.05
HB6	12/55	1	----	Birmingham	1.20±0.07
HB7	11/55	27	----	Reading	0.2 ±0.1
HB8	12/55	31	Ribs	Oxford	0.06±0.01
HB9	11/55	23	Ribs	Reading	0.16±0.08
HB10	12/55	10	Ribs	Birmingham	0.57±0.03
HB11	1/56	19/12	Ribs	Birmingham	0.76±0.05
HB12	1/56	1	Ribs	Birmingham	1.1 ±0.03
HB13	1/56	3½	Ribs	Dudley	1.05±0.07
HB14	1/56	16½	Tibia	Carlisle	0.25±0.02
HB15	1/56	50	Tibia	Carlisle	0.06±0.03
HB16	1/56	65	Tibia	Carlisle	0.13±0.02
HB17	1/56	s/born	Sternum & Femur	Carlisle	0.45±0.06
HB20	2/56	16/12	Femur	Carlisle	0.8 ±0.1
HB21	2/56	3/12	Femur	Carlisle	1.3 ±0.1
HB22	1/56	33	Ribs	Swindon	0.1 ±0.1
HB23	12/55	18	Ribs	Swindon	0.2 ±0.03
HB24	12/55	40	Ribs	Swindon	0.07±0.02
HB25	12/55	34	Ribs	Swindon	Sample lost
HB26	2/56	8/12	Ribs	Birmingham	0.5 ±0.2
HB27	2/56	20	Ribs	Birmingham	0.2 ±0.05
HB28	1/56	16	Ribs	Birmingham	0.2 ±0.03
HB29	2/56	2¼	Femur	Birmingham	0.55±0.03
HB30	2/56	11⅔	Shaft	Birmingham	0.15±0.02
HB31/1	2/56	9½	Femur	Birmingham	0.25±0.02
31/2	2/56	9½	Femur	Birmingham	0.28±0.02
31/3	2/56	9½	----	Birmingham	0.20±0.03

[1] The errors attributed to the S. U. results are equal to twice the standard duration due to counting statis tics, and take no account of other sources of error.

Fig. 1


Sr-90 ACTIVITY IN MILK IN 1955
Sr-90 ACTIVITY (μμC/gm.Ca)
MILK | LAB. | SYMBOL
Chicago | Chicago
New York | New York
" | Woolwich
Yeovil | New York
" | Harwell
Jan. Feb. Mar. Apr. May June July Aug. Sep. Oct. Nov. Dec.
1 2 3 4 5 6 7

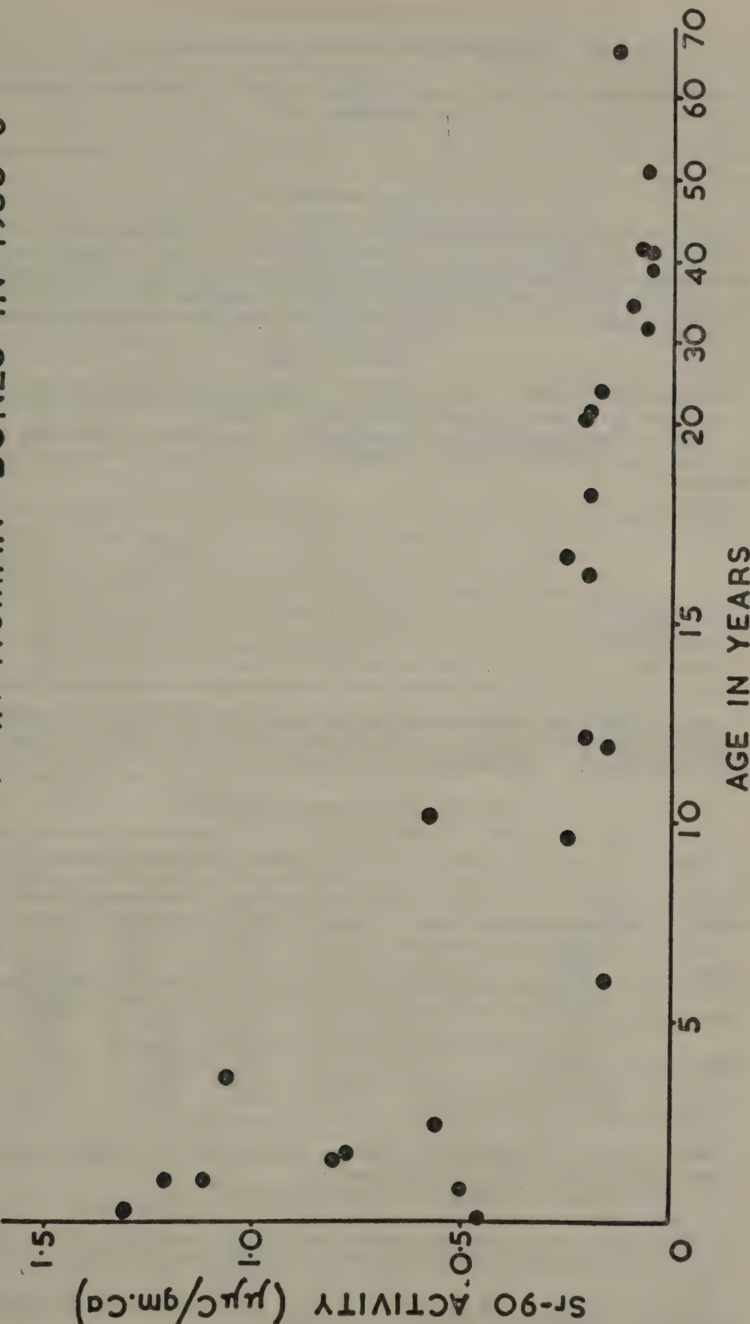


Fig. 2 Sr-90 ACTIVITY IN HUMAN BONES IN 1955-6

A. E. R. E. HP/R 2182

ATOMIC ENERGY RESEARCH ESTABLISHMENT

Radiostrontium and Radiocaesium Measurement in Biological Materials to December 1956

By D. V. Booker, F. J. Bryant, A. C. Chamberlain, A. Morgan, and G. S. Spicer

1. INTRODUCTION

The methods used in the estimation of Sr.90 in biological materials, the inter-comparison of results between different laboratories and the results up to the early part of 1956 were fully described in A. E. R. E. HP/R 2056.[1] The present report gives further results for Sr.90, presents some data on Cs.137 in milk, and examines in greater detail the evidence for the trend of radioactive contamination with time.

2. SR.90 IN SOIL

An effort was made to get a reliable estimate of the total Sr.90 in soil, for comparison with the rain data of Stewart et al (1956).

Three soil horizons (0–4″, 4–8″ and 8–12″) were taken in July, 1956, at each of four sites within 15 miles of Harwell, but on widely differing soil types as follows:

Site	Soil	Annual Rainfall (inches)
Grove, Berks	Clay	27.5
Aldermaston, Berks	Acid sand	27.5
Culham, Oxon	Greensand	25
Chilton, Berks	Chalk	27.5

The soils were extracted with 6M hydrochloric acid at Woolwich. This method has been shown to remove as much Sr. 90 as it got by complete fusion of the soil (HP/R 2056). The results are given in Table I.

TABLE I.—*Sr. 90 in soil (July 1956)*

Ref.	Site	Depth	gms. Ca/kg.	$\mu\mu$c/m²	S. U.
UK36	Aldermaston	0–4″	1.60	2560	18.6
	Aldermaston	4–8″	1.52	<150	<0.8
	Aldermaston	8–12″	1.46	<50	<0.3
UK37	Culham	0–4″	3.00	2530	8.0
	Culham	4–8″	2.68	220	0.6
	Culham	8–12″	3.46	<150	<0.3
UK35	Grove	0–4″	39	1900	0.66
	Grove	4–8″	17	270	0.18
	Grove	8–12″	10	224	0.22
UK38	Chilton	0–4″	156	2180	0.15
	Chilton	4–8″	185	<150	<0.01
	Chilton	8–12″	204	<150	<0.01

Where the sign < is used, the activity was less than the minimum measurable.

The following deductions may be drawn:

(a) at least 80 per cent of the activity is in the top 4″.

(b) There is good agreement in total Sr. 90 between sites. The nature of the soil does not seem to affect the degree of retention of the fallout.

(c) The average level in this area is 2.5 mc/km² (6.5 mc/mi²).

[1] Circulated to the October 1956 meeting of the United Nations Scientific Committee on the Effects of Radiation with reference A/AC82/G/R30.

3. SR. 90 AND CS. 137 IN MILK

The regular supply of skimmed dried milk from a factory at Frome, Somerset[2] has continued. Some of the samples have been analysed at the Health and Safety Laboratory at New York, through the help of Dr. J. H. Harley, and some done at Harwell or Woolwich. In fig. 1a the results are shown in terms of the specific activity of Sr. 90 relative to calcium, using the unit referred to as the strontium unit.

One strontium unit (S. U.) = One micro-micro-curie Sr. 90 per gram of calcium.

Also in fig. 1a is drawn the graph of cumulative Sr. 90 fallout in rain at Milford Haven, Pembrokeshire, given by Stewart et al (1956). The units are millicuries Sr. 90 per square kilometre. The trend of the figures with time is discussed below in para 6.

In fig. 1b is shown the Cs 137 activity of samples from the same series. The Harwell results were obtained by gamma spectrometry of the dried milk, whereas the Woolwich results were by chemical analysis. The details of the gamma spectrometric method are given by Booker (1957).

A series of samples of full cream dried milk from different regions in the U. K. was taken in October, 1956. The results of Sr. 90 and Cs. 137 analysis on these samples are given in Table II.

TABLE II.—*Sr–50 and Cs–137 in milk (October 1956)*

Area	Sr–90 $\mu\mu$c/gm. Ca.	Cs–137 $\mu\mu$c/gm. K
Somerset	4.6	28
Yorkshire	4.3	30
Cumberland	6.5	28
Carmarthen	8.0	65
Antrim	6.9	87
Londonderry	10.3	84

Calcium is about 1.2% and potassium 1.4% of dried milk by weight.

The Sr. 90 activity of 10.3 S. U. in milk from County Londonderry is similar to maxima of 10 or just over found in the U. S. (Harley et al 1956) and in Canada (Canadian submission to U. N. of October, 1956).

4. SR. 90 IN SHEEP BONES

Yearling sheep have been taken from three areas in Wales in each of the years 1954, 5 and 6. The sheep came from the same flocks each year, and the flocks were grazing the same pasture. The areas covered by hill sheep are large, and the exact soil conditions cannot be specified, but analysis of samples showed that the soil was calcium deficient, especially at Cwmystwyth (HP/R 2056, Appendix A1)

The results of Sr. 90 analysis on the long bones of these sheep are given in Table III, and shown graphically in fig. 2.

TABLE III.—*Sr. 90 in bones of Welsh sheep (S. U.)*

	Cwmystwyth Cardigan	Vyrnwy Montgomery	Talgarth Brecon
1954	18.8	7.7	1.5
1955	59, 56	17, 13	5.7, 5.7
1956	170, 151	42, 40	24, 24

The 1954 analyses were done in the U. S. Those of 1955 and 1956 have been done at Woolwich and Harwell independently, and in Table III the results of both analyses are given (Woolwich in the left).

[2] The location was wrongly referred to as Yeovil, Somerset in HP/R 2056, but the milk processed at the factory is in fact drawn from an area of 20 miles radius round it.

In fig. 2 the cumulative fallout of Sr.90 in rain is also given (this time on a log scale). It appears that the rate of increase of Sr.90 activity in these sheep has been approximately exponential, with an average increase of a factor 3 per annum, and has been about as rapid as the rate of increase of cumulative fallout.

Bones fro 10 English sheep killed in 1956 have been analysed for Sr.90. Three hill sheep from Dartmoor, Durham and Lancashire gave 53, 71 and 61 S. U. respectively, showing that the relatively high levels are not confined to limited areas in Wales. Seven lowland sheep from south and east England showed Sr.90 activity ranging from 7.8 to 15.6 S. U. Direct comparison with previous years was not possible, because the Suffolk flocks from which the 1954 and 1955 animals were obtained have been broken up, but the general level in lowland sheep seems little altered in 1956 compared with 1955.

5. SR.90 IN HUMAN BONES

Analyses of 21 bones additional to those reported in HP/R 2056 have been completed and the results are given in Table IV. Work has been concentrated on the bones (femurs) of children, since previously it had been shown that they show higher activity than those of adults. The ages at death ranged from 2 days to 14 years. 12 specimens from infants under one year of age averaged 0.62 S. U., 5 from children of 1 to 4 years averaged 0.76 S. U. and 4 from children of 7 to 14 years 0.27 S. U.

Three specimens, all from 6 month old infants, showed Sr.90 activity of 1 S. U. or greater, but none exceeded the previous maximum of 1.3 S. U. reported in HP/R 2056.

The femur HB38 from a 14 year old boy was divided into four parts transversely before analysis. The epiphyseal bone at either end showed double the specific activity of the centre of the shaft. This was to be expected since recently deposited calcium contains more Sr.90 than that laid down several years ago.

TABLE IV.—*Sr–90 in human femurs (additional to those given in Appendix C4 of HP/R 2056)*

Ref	Date	Age at death	District	Sr–90 (S. U.)	Notes
32	5/56	1 m	Herts	0.5	
33	4/56	2 m	Sussex	.15	
34	6/56	17 m	Surrey	.9	
35/1	5/56	2½ y	London	.8	Proximal end
35/2	5/56	2½ y	London	.8	Distal end
36	4/56	8 y	Middlesex	.27	
37	5/56	12 y	London	.24	
38/1	5/56	14 y	Surrey	.20	Distal subepiphysea
38/2	5/56	14 y	Surrey	.17	Distal plate
38/3	5/56	14 y	Surrey	.11	Centre of shaft
38/4	5/56	14 y	Surrey	.20	Proximal subepiphyseal
39	6/56	2 d	London	.45	
40	7/56	7 d	Kent	.15	
41	7/56	12 d	London	.35	
42	7/56	3 d	London	.8	
43	7/56	1 m	London	.4	
44	7/56	2 m	Sussex	.55	
45	6/56	5 m	London	1.1	
46	6/56	6 m	Bucks	.75	
47	6/56	6 m	London	1.2	
48	7/56	6 m	Lancashire	1.1	
49	7/56	2 y	Middlesex	.75	
50	7/56	2½ y	Essex	.70	
51	7/56	3½ y	Sussex	.64	
52	6/56	7½ y	Essex	.38	

6. TREND OF RESULTS WITH TIME

It would be valuable if the rate of increase of Sr. 90 and Cs. 137 activity in biological materials could be correlated with the physical data on fallout rates. The following indications emerge from the data:

(i) *Milk.*—There was a sudden rise in activity in the Spring of 1955 . This appears in Sr. 90 estimates in British and American data (of. HP/R 2056, fig. 1) and applies to Cs. 137 as well as Sr. 90. This rise was probably due to a combination of two factors.

(a) An increase in the rate of fallout at that time, which is noticeable as a temporary increase in the slope of Stewart's grapt reproduced in fig. 1a.

(b) The sending out to pasture at that time of year of cows which had been given stored feed during the winter.

Since the summer of 1955 the milk levels seem to have been rather steady both in U. K. (fig. 1) and in U. S. (Eisenbud, 1957) ; and the spring increase was not nearly so marked in 1956 as in 1955. The median of 9 Somerset samples in 1956 is 4.2 S. U. as compared with 4.1 S. U. for 13 1955 samples.

(ii) *Sheep bones.*—There is a sharp distinction in the U. K. results between hill sheep and lowland sheep. The Sr. 90 level in the bones of the Welsh sheep seems to be increasing about as rapidly as the cumulative fallout whereas animals from S. E. England show little increase in the period 1955–1956.

(iii) *Human bones.*—Samples are too limited in number and diverse in origin to allow of satisfactory comparison, but there is no evidence of a rapid increase of Sr. 90 activity in the last year. A considerable time lag between changes in the environment and in human bone must be expected.

The tentative conclusion from the results so far is that Sr. 90 activity of tissues deriving their calcium from normal soils is increasing less rapidly than the cumulative fallout, and is probably more nearly proportional to the rate of fallout. On very deficient soils the opposite apears to be true.

These effects may well be correlated with the importance on very low calcium soils of the root as compared with the foliar method of uptake of radiostrontium to vegetation.

7. SUMMARY OF 1956 RESULTS

The range of 1956 Sr. 90 results, and the median values are given in Table V.

TABLE V.—*Sr–90 in biological materials in 1956*

Material	No. of Samples	Sr. 90 activity (S. U.)		
		Max.	Min.	Median
Grass (acid hill soils)	9	2,180	113	370
Grass (normal soils)	27	85	23	40
Sheep bones (hills)	6	170	24	57
Sheep bones (lowlands)	7	15.6	7.8	13.7
Milk (Somerset)	9	5.7	2.9	4.2
Milk (other areas)	5	10.3	4.3	6.9
Human bones (child)	31	1.3	0.15	0.55
Human bones (adult)	8	0.25	0.06	0.20

The details of all the above samples have been given in HP/R 2056 or the present report, except for the grass samples, which will be reported more fully elsewhere.

8. ACKNOWLEDGEMENTS

We are greatly indepted to Mr. K. H. Jones and to the members of the National Agricultural Advisory Service of the Ministry of Agriculture Fisheries & Food for their help in arranging the agricultural samples. We are also greatly indebted to Dr. M. Bodian who obtained the human bone specimens for us.

9. REFERENCES

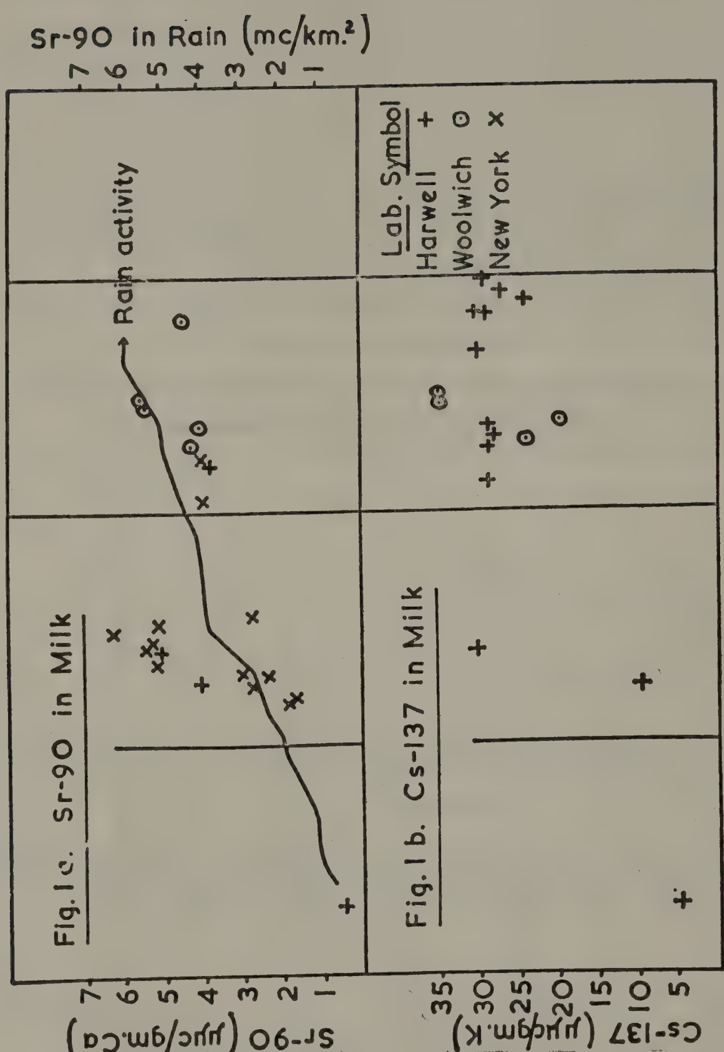
Booker, D. V., A. E. R. E. HP/R 2183 (1957)

Bryant, F. J., Chamberlain, A. C., Morgan, A., and Spicer, G., A. E. R. E. HP/R 2056 (1956)

Eisenbud, M. Paper to Washington Academy of Sciences, 15 Nov. 1956.

Harley, J. H., et al. U. S. A. E. C. Health & Safety Laboratory, NYO 4751 (1956)

Stewart, N G, Crooks, R. N. and Fisher, E. M. R., A. E. R E, HP/R 2017 (1956).


Sr-90 in Rain (mc/km.²)
Rain activity
Lab. Symbol
Harwell +
Woolwich ⊙
New York ×
Fig. 1a. Sr-90 in Milk
Fig. 1b. Cs-137 in Milk
1954
1955
1956
Sr-90 (μμc/gm.Ca)
Cs-137 (μμc/gm.K)

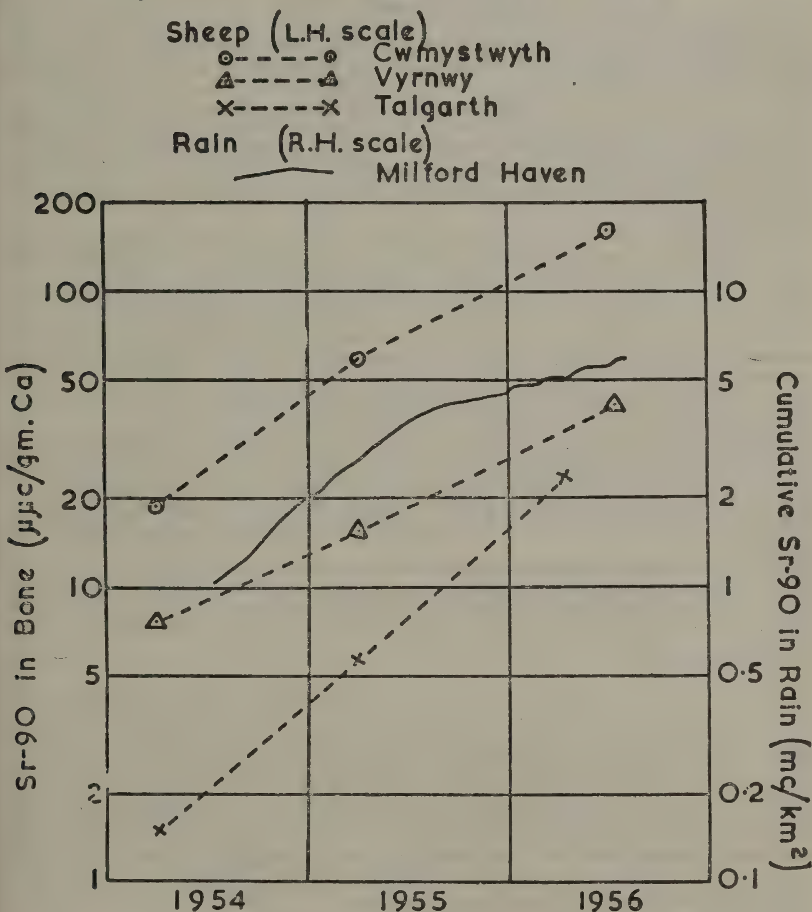


Fig. 2. Sr-90 in Welsh Sheep Bones

Appendix 3

A Report by the World Health Organization on Genetic Effects of Radiation

[World Health Organization, Division of Public Information, Press Release WHO/11, March 13, 1957]

Genetic Effects of Radiation

WHO TO PUBLISH REPORT

Throughout man's existence on earth, he has been continuously bombarded by radiations coming from outer space, from radioactive material in the earth's crust, and from natural radioactive elements within his own bone and flesh.

The intensity of this bombardment is being notably increased, in our days, by man-made sources such as medical X-ray machines, radioactive material used in medicine, and also certain material and apparatus used in science, industry and commerce; artificial radioactive elements distributed by man in nature; and, to a minor extent, shoe-fitting machines, radioactive luminous compounds on watches, etc.

Radiation has been demonstrated to be one of the agents which produces genetic mutation in a wide range of organisms from bacteria to mammals, and a Group of 20 international experts was therefore brought together by the World Health Organization (WHO) in Copenhagen last August to discuss research problems connected with the effect of radiation on human heredity.

This Group has produced a highly technical report which, together with a number of specialized papers prepared by certain of its members, is to be published by the World Health Organization within a few months. Advance copies of the text are at present in the hands of members of the United Nations Scientific Committee on the Effects of Atomic Radiation.

"Man's most unique and precious possession is his hereditary material which must determine the health and orderly development of future generations", states the introduction to the Group's report. It goes on, "This Group is of the opinion that the well-being of the descendants of the present generation is threatened by developments in the use of nuclear energy and of sources of radiation."

The report states categorically that "additional mutation produced in man will be harmful to individuals and their descendants * * *" and that " * * * all man-made radiation must be regarded as harmful to man from the genetic point of view".

Gaps in knowledge

The WHO experts agreed that present developments in the peaceful use of nuclear energy should contribute much to man's social and cultural development, and that therefore some risk must be accepted. They recognize, however, that if the dangers are to be minimized, "every possible step must be taken to reduce the exposure of man to radiations, and to understand the effects of exposure * * * Only in the light of more knowledge can decisions be taken to define more accurately the maximum amount of exposure which may be accepted by individuals and populations without risk of serious harm." The Group therefore examined some of the "larger gaps in knowledge" as they appeared at the present time, and listed some thirteen fields of genetic research in which the need for further investigation is urgent.

Cumulative effect

There are strong grounds for believing that inherited effects of radiation are additive, the experts agreed. A small amount of radiation received by each of a large number of individuals can therefore do an appreciable amount of damage to the population as a whole, although the effects may not appear for a number of generations. It is therefore desirable to limit the accumulated radiation doses received by the sex glands of individual men and women, particularly up to 30 years of age, in order to keep the average dose to the sex glands of the population as whole very low.

The most important sources of radiation to the human sex glands, the experts agreed, are at the present time from the natural radiation (normal level between 2 and 5 roentgens per individual in 30 years) and from the radiation received by patients undergoing medical X-ray examination (probable average in certain countries between 1 and 3 roentgens per individual in 30 years). If exposure during therapeutic X-ray treatment is also considered, the "total" exposure to a population might be greater. It is however difficult, the report states, to get sound data for estimating how much radiation due to therapeutic exposures is received by persons before the age at which procreation may be expected to be ended.

In connexion with the danger of radiation from medical sources, one member of the Group pointed out in a paper that radioscopic apparatus, sometimes not adequately shielded, is to be found more and more frequently in the consulting rooms of general practitioners who do not have the necessary formal training in radiology.

Artificial radioactive elements distributed in nature

In a paper by Professor R. M. Sievert, of the Institute of Radiophysics, Stockholm (Sweden), which accompanies the report, some reference is made to artificial radioactive products distributed in nature as a result of military tests. Professor Sievert recognizes that the WHO Study Group was concerned with the peaceful use of atomic energy and the effects, for instance, of the future disposal of radioactive wastes from such peaceful uses.

He points out, however, that it is essential to take into consideration the evidence obtained following atomic weapon tests, since such information is the best at present available for the study of problems in the field of artificial radioactive elements distributed in nature.

Recent measurements from large samples of foodstuffs in Sweden, Professor Sievert says, have shown that foods such as milk, beef, corn and vegetables eaten today contain artificial radioactive elements with a radiation level in many cases exceeding that due to the naturally-occurring radioactive constituents of animals and plants. It does not yet seem possible, Professor Sievert goes on, to estimate the radiation doses to human tissues, nor their distribution in time, which are necessary data for drawing conclusions of biological significance.

Professor Sievert believes that it is extremely difficult to predict what will in the future be the most important sources of radiation from artificial radioactive elements distributed in nature.

"There is reason to believe that the problems of disposal of radioactive wastes will be satisfactorily solved and that precautions in the handling and use of radioactive material will be adequate", Professor Sievert says, but goes on to warn that "accidents and unforeseen events may gradually spread radioactive substances beyond control; they could then follow unknown paths and be harmful to mankind in ways that would become known to us only after long experience."

Some conclusions

In a section entitled "Some Conclusions" the Group's report lists eight recommendations, including the establishment of more institutions and large university departments concerned with human genetics and improved teaching in this branch; the systematic registration of serious hereditary diseases; and efforts by UN Agencies toward the collection and publication of information on subjects like fertility, consanguineous marriages and parental ages which are so essential as brackground in human biological studies.

The Group was particularly impressed with the genetic hazards of man-made radiation from sources used in medicine, industry, commerce and experimental science. Both as an approach to control and as providing basic background information on radiation exposure and effects on man, it is essential, the experts agreed, that methods be found of recording exposures to individuals and populations, however difficult this may prove.

INFORMATION SUBMITTED BY THE WORLD HEALTH ORGANIZATION TO THE UNITED NATIONS SCIENTIFIC COMMITTEE ON THE EFFECTS OF ATOMIC RADIATION

In the "Conclusions of the First Session" of the United Nations Scientific Committee (A/AC.82R.10 of 27 March 1956) it was stated that "This year the human

geneticists will meet at a congress on human genetics. This opportunity should be used, with the assistance of the World Health Organization to seek advice about the possibility of setting up a standard of recognition for one or more clearly recognizable medical conditions thought to be largely or solely genetic in origin."

A statement describing the United Nations Scientific Committee's request was made at a plenary session of the International Congress of Human Genetics held at Copenhagen in August 1956. There was no formal response to this request from the Congress itself. However, a Study Group on the Effect of Radiation on Human Heredity had been arranged by WHO so as to follow immediately after the International Congress on Human Genetics, and the President and a number of the members of the Conference took part in this Study Group.

A reply to the request of the United ations Scientific Committee on the Effects of Atomic Radiation was formulated during the meetings of this Study Group and the reply is attached as document A. Since this reply is to a considerable extent supplemented by the report and working papers of the Study Group, these are also attached for the consideration of the Committee.

The Study Group convened by WHO on the Effect of Radiation on Human Heredity was designed to have three objectives:

1. To provide a small symposium of papers on the effects of radiation on human heredity.
2. To formulate in simple terms the desirable lines of further research on the effect of radiation on human heredity.
3. To give some recommendations in the particular province of WHO.

Document A

REPLY TO A QUESTION RAISED BY THE UNITED NATIONS SCIENTIFIC COMMITTEE ON THE EFFECTS OF ATOMIC RADIATION

There are at the present time no convenient indicators of recent genetic damage in man, and standards which can be applied in widely separate areas for recognition of hereditary characters are extremely difficult to establish even in the best laboratory conditions. Nevertheless, new methods of recognition are being developed. Techniques are available for accurate identification of serological traits (e. g. blood antigens) and other chemical specificities (e. g. haemoglobins, serum proteins, aminoacids in urine, factors controlling clotting of blood). Ophthalmological tests are also relatively exact but require co-operation of the patient. Many hereditary conditions can only be correctly diagnosed post mortem. Thus it would seem inadvisable to make definite recommendations for standardization on a considerable scale at the present time. If however the urgency of the problem necessitates an immediate attempt to select a group of "indicator traits" then, on the basis of experience to date, the following provisional list is suitable for setting up "standards of recognition":

Retinoblastoma
Neurofibromatosis
Aniridia
Acrocephalosyndactyly
Osteogenesis imperfecta
Chondrodystrophic dwarfs of all kinds
Haemophilia
Sex-linked infantile muscular dystrophy (Duchenne type).

It is emphasized that if "indicator traits" are used in the manner implied by the question, as many traits as possible should be used in the same area at the same time. At the same time variation in the sex ratio should be studied as an index of mutation.

In setting up "standards of recognition", human geneticists and appropriate specialist physicians should consult.

STUDY GROUP ON THE EFFECT OF RADIATION ON HUMAN HEREDITY

Copenhagen, 7–11 August 1956

Members:

Dr. T. C. Carter, Radiobiological Research Unit (Medical Research Council of Great Britain), Atomic Energy Research Establishment, Harwell, Berks, England

Dr. W. M. Court Brown, Group for Research into the General Effects of Radiation (Medical Research Council of Great Britain), Radiotherapy Department, Western General Hospital, Edinburgh, Scotland

Dr. S. Emerson, Biology Branch, Division of Biology and Medicine, US Atomic Energy Commission, Washington, D. C., USA

Dr. N. Freire-Maia, Laboratory of Genetics, University of Parana, Curitiba, Parana, Brazil

Dr. A. R. Gopal-Ayengar, Biology Division, Department of Atomic Energy, Indian Cancer Research Centre, Bombay, India

Dr. A. Hollaender, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., USA (Chairman)

Mr. G. H. Josie, Research and Statistics Division, Department of National Health and Welfare, Ottawa, Canada

Dr. S. Kaae, Finseninstitutet og Radiumstationen, Copenhagen, Denmark

Professor T. Kemp, Universitetets Arvebiologiske Institut, Copenhagen, Denmark

Dr. J. Lejeune, Centre National de la Recherche scientifique, Paris, France

Professor H. J. Muller, Department of Zoology, University of Indiana, Bloomington, Ind., USA

Professor J. V. Neel, Heredity Clinic, Institute of Human Biology, University of Michigan, Ann Arbor, Mich., USA

Dr. H. B. Newcombe, Biology Branch, Atomic Energy of Canada Limited, Chalk River, Ont., Canada

Professor L. S. Penrose, The Galton Laboratory, University College, London, England

Professor R. M. Sievert, Institute of Radiophysics, Karolinska Hospital, Stockholm, Sweden

Dr. C. A. B. Smith, The Galton Laboratory, University College, London, England

Professor A. C. Stevenson, Department of Social and Preventive Medicine, The Queen's University of Belfast, Institute of Clinical Science, Belfast, Northern Ireland (Rapporteur)

Professor O. Freiherr von Verschuer, Institut für Humangenetik der Universität Münster, Münster, Germany

Dr. Bruce Wallace, Biological Laboratory, Long Island Biological Association, Cold Spring Harbor, Long Island, N. Y., USA

Professor M. Westergaard, Universitetets Genetiske Institut, Copenhagen, Denmark

Observers:

Dr. R. K. Appleyard, Acting Secretary, Scientific Committee on the Effects of Atomic Radiation, United Nations, New York, USA

Dr. R. A. Silow, Specialist in Atomic Energy, Agricultural Institutions and Services Branch, Agriculture Division, FAO, Rome, Italy

Dr R. L. Zwemer, Chief, Division of International Co-operation for Scientific Research, Department of Natural Sciences, UNESCO, Paris, France

Secretariat:

Dr. P. Dorolle, Deputy Director-General, WHO

Dr. M. Pizzi, Chief, Epidemiological Information and Morbidity Statistics Section, WHO

Dr. I. S. Eve, Medical Officer in charge of Questions dealing with Atomic Energy and Health, WHO

Two national committees reported in 1953 on the effects of ionizing radiation on man.[1] Although difficult to compare in detail, these reports come to remark-

[1] United States of America, National Academy of Sciences (1956) Biological effects of atomic radiation; Great Britain, Medical Research Council (1956) The hazards to man of nuclear and allied radiations, London.

ably similar conclusions as to the probable effects on the descendants of populations exposed to increased amounts of such radiations.

The emphasis in both these reports was, however, on trying to set some quantitative limits to the risks in the light of existing knowledge.

The purpose of assembling the Study Group whose report is presented here was essentially twofold. The first aim was to obtain the opinions also of authorities on genetics from countries, other than those whose national committees have already stated their views. The second was to have the opinions of a number of experts on an aspect relatively little considered in the national reports—namely, the lines of research which are needed in the light of present knowledge, to increase our understanding of the genetic effects of ionizing radiations on man.

The Group met, by courtesy of the Rector of the University of Copenhagen, in the Council Room of the University, from 7 to 11 August 1956. The agenda adopted was intended to permit exploration of the views of the members on the theoretical and practical difficulties in closing present gaps in knowledge. The procedure adopted was for a number of members to open discussions either by short statements or by submission of invited papers. The opportunity was also taken to discuss a number of subjects not formally introduced.

The papers submitted are reproduced as annexes to this report and whilst the report itself represents the combined opinions of all the members of the Study Group, the annexed papers give the individual views of the authors.

The proceedings were opened by Dr. P. Dorolle, Deputy Director-General of the World Health Organization, and the Group elected Dr. A. Hollaender as Chairman.

1. INTRODUCTION

Man's most unique and precious possession is his heredity material which must determine the health and orderly development of future generations. The Group is of the opinion that the well-being of descendants of the present generation is threatened by developments in the use of nuclear energy and of sources of radiation. Both of these developments are inevitable and they should contribute much to man's social and cultural development. It would seem therefore that some risk must be accepted, but if the dangers are to be minimized every possible step must be taken to reduce the exposure of man and to understand the effects of exposure. Only in the light of more knowledge can decisions be taken to define more accurately the maximum amount of exposure which may be accepted by individuals and populations without risk of serious harm.

Radiation has been demonstrated to be one of the agents which produces mutation in a wide range of organisms from bacteria to mammals. The Group is agreed that additional mutation produced in man will be harmful to individuals and to their descendants. While there may be inherent and environmental mechanisms which modify the impact of these mutations over periods of many generations, the effectiveness of such mechanisms in man is not known. In essence then, all man-made radiation must be regarded as harmful to man from the genetic point of view.

In recent years, considerable quantitative knowledge has been accumulated on the basic mechanisms of genetics. There are strong grounds for believing that most genetic effects are very closely additive so that a small amount of radiation received by each of a large number of individuals can do an appreciable amount of damage to the population as a whole. There are, however, many gaps in knowledge particularly concerning these effects in man. These gaps will only be closed after a great expansion of general and *ad hoc* research in genetics and other fields of biology.

The Group has received the following resolution passed by the First International Congress of Human Genetics in Copenhagen and it notes and agrees (while at the same time recognizing that WHO's work is only concerned with the peaceful use of atomic energy):

"The damage produced by ionizing radiation on the hereditary material is real and should be taken seriously into consideration in both the peaceful and military uses of nuclear energy as well as in all medical, commercial and industrial practices in which X-rays or other ionizing radiation is emitted. It is recommended that the investigation of the amount and type of damage and of related genetic questions, be greatly extended and intensified with a view to safe-guarding the well-being of future generations."

The Group agrees with the memorandum, entitled "Human and Medical Genetics", which was submitted in 1955 by the Government of Denmark to the World Health Organization.[2]

This Group takes note of the report of the National Academy of Sciences of the United States of America and that of the Medical Research Council of Great Britain. It is not intended to reproduce any of the material in these reports but the Group notes the substantial similarity of the findings and recommendations of these reports and is in essential agreement with them.

2. NATURAL AND MAN-MADE SOURCES OF IONIZING RADIATION

The present sources of ionizing radiations of interest for the treatment of problems related to the genetic effects in man include the following:

Natural sources

1. Cosmic radiation.
2. Naturally occurring amounts of radium, thorium and potassium in the earth crust.
3. Content of natural radioactive elements in living tissues.

Man-made sources

4. Radioactive material and technical arrangements producing ionizing radiation (such as X-ray tubes and other particle accelerators, nuclear reactors, etc.) used in education, science, medicine, industry and commerce.
5. Sources used by the population for other purposes than those mentioned in 4 (radioactive luminous compounds on watches and other articles for common use, television sets, etc.), although such sources are much less significant than those mentioned in 4 and 6. It is important, however, that their existence be recognized.
6. Artificial radioactive elements distributed by man in nature.

Information as to the contributions to the doses received by individuals and by large population groups from the various sources listed above is summarized in Professor R. M. Sievert's paper, from which it is obvious that as regards the average dose to the gonads the most important contributions are at present those from the natural radiation (normal level: between 2 and 5 r per individual in 30 years) and from the radiation received by patients undergoing medical X-ray examination (probable average between 1 and 3 r per individual in 30 years). If therapeutic exposures are also considered, the "total" exposure to a population might be greater. It is, however, difficult to get sound data for estimating how much exposure is received in therapeutic exposures to persons before the age at which procreation may be expected to be ended.

It may be noted that at the present time the highest dose to the gonads caused by natural radiation in areas with a large population seems to exist in parts of Travancore, India, on ground containing monazite sand (possibly of the order of between 10 and 20 r per individual in 30 years).

3. IMPORTANCE OF RECORDING RADIATION EXPOSURE IN INDIVIDUALS AND POPULATIONS

From a genetic point of view the total accumulated dose is the important one and for this reason the measurement of exposure to ionizing radiations is an essential preliminary to attempts to relate dosage received to effects in man. For such measurements to be useful, the information must be recorded systematically. Unless the information is available in the form of the dose received by individuals, records of exposure would be unsuitable for many purposes and therefore some system of registration is essential. The effect of recording would almost certainly be to cut down the exposures given in medical diagnosis and treatment, since it would impress radiologists and technicians with the magnitude of such exposures. In one hospital where such recording was started there has been a 30 per cent. reduction in the total exposure of the staff. Doubtless a similar system of recording in diagnostic practice would reduce the exposure to the patients. This in itself would be a sufficient justification for introducing the procedure. It seems likely that the two national reports will already have done much to overcome the hesitation to record the dose on the part of those who would be concerned in making such records but that a recommendation from this Group would also be helpful.

---

[2] Off. Rec. Wld. Hlth. Org., *68*, 147.

The Group is conscious that the adoption of any system of recording dosage will give rise to difficulties because it will increase the burden of work of radiologists and their staffs. Nevertheless, it feels that the importance of these procedures is such, and is so well recognized by radiologists that both those in charge of radiological departments and other physicians who use X-rays will be co-operative.

Whatever system adopted should take into account three desirable requirements:

1. That the individual will not, through lack of information, accumulate excessive exposure.
2. That information becomes available as to how much exposure to the gonads is received at each age in individuals and on an average per head of population.
3. That it should be possible to recognize the amount of exposure received by the parents of a given child. (Eventually, the information would be available for several generations.) This information is particularly valuable for purposes of genetic analysis.

The Group suspects that exposures in some industries and in scientific work are unnecessarily high. Exposures from these sources should be recorded in such a way that the dosage received can be related in individuals and populations to that received from other sources.

It seems unlikely that all countries would favour or indeed would be able to introduce the same standards of registration. Although it is expected that recommendations on mechanisms of recording will shortly be available from the International Commission on Radiological Protection, there should not be any delay in improving the standard of recording of exposures.

Whatever procedures of recording and registration are adopted will entail a large expenditure of money and effort. The need, however, is urgent. Further, the present is the appropriate time to initiate such procedures, since the introduction of atomic energy for industrial use and the extension of the use of radiation tools in biology and medicine make it possible to start with such procedures at an early stage of a period of rapid development.

4. RESEARCH

General

Additions to the understanding of the effects of radiation in man come from a very wide field of research. It is impossible to forecast what work in biology or genetics will contribute information relative to the problems. Accordingly, the Group is strongly of the opinion not only that as much experimental work as possible should be done on radiation effects on suitable organisms and such controlled observation studies as offer in man, but that there should be an intensification of all human and experimental genetic research. The Group feels that there should be the closest possible collaboration between those working in the experimental and human fields: their work is complementary. Each should be stimulating the other's research projects. This need for intensification of research in man and in other organisms raises problems of finance and of shortages of trained research workers. Both these difficulties are likely to be intensified if new areas of work, such as that on tissue cultures, chemical mutagenesis, serology, biochemical genetics and epidemiological problems of genetic disease are to develop as rapidly as is desirable. The problem of manpower shortages, in regard to both biologists and physicians, tends to be perpetuated by lack of career opportunity for those working on genetics. There is also an insufficient number of institutions where an adequate training in genetics, particularly in human genetics, can be given.

It is possible that the results of much effort in these fields will prove disappointing. Nevertheless, research workers and those supporting their work must have the courage to face the possibilities of such disappointments and still go forward.

The developments of nuclear energy would never have been made unless enormous risks of failure had been accepted. These innovations have extremely important implications among which the possible effects on man's genetic composition are outstanding. If there is to be a climate of public opinion favourable to the development of nuclear energy the peoples must be assured that investigations essential for their future health and welfare and that of their children will be undertaken on an adequate scale. This will require recognition by governments that very substantial financial provision must be made for genetic and other biological investigations essential to an under-

standing of the effects of radiation on man. Biological research in the past has suffered severely from lack of funds.

Specific

The Group does not feel that it should attempt to recommend specific research projects. Nevertheless, it seems desirable to recognize the larger gaps in knowledge as they appear at the present time. Among the fields in which the need for further work is urgent, if the genetic hazards of the irradiation of human populations are to be understood, the following appear outstanding. It should be emphasized that the rapid developments in genetics and other sciences must determine that recommendations for lines of research should only be accepted as tentative and should be revised periodically.

(*a*) *Further study of spontaneous and artificially-induced mutation.*—There is need for further study of the number and kinds of mutations produced by various doses and types of irradiation applied at different stages of the life-cycle under a variety of conditions and utilizing different kinds of organisms. The relatively limited opportunities to study irradiated human beings and their offspring should be exploited to the fullest extent possible. The appreciation of radiation-produced mutations is intimately related to a similar extension of knowledge concerning mutations that appear to arise spontaneously or as the result of the action of chemicals and of physical agents other than ionizing radiation.

(*b*) *Mutational component in the somatic changes produced by radiation and other means.*—The role of changes in the hereditary material of somatic cells in the genesis of leukemia, in other forms of neoplasms, and in alterations in the life span, is at present a controversial field whch needs clarification. The effects of low doses of radiation, including those from radioisotopes, require special study. An important method of attack on this problem is opened by recent developments in tissue culture techniques.

(*c*) *Means of protection against mutagenic agents.*—The pioneer studies which indicate the possibility that the production of radiation-induced mutations can be modified by various means have important implications for man and require extension in many directions.

(*d*) *Development of new and improved techniques for the identification of mutants.*—Efforts directed at developing more exact methods for the recognition of mutant individuals, and the distinction between the latter and phenocopies, should be intensified. It is important to prosecute studies of the frequency of a wide range of types of mutations including those with extremely small effects, recognizable only through special statistical or breeding techniques.

(*e*) *Manner of gene action.*—The phenomena of dominance, synergism and other forms of gene interaction, the multiple effects of a single gene and the role of environmental factors in the determination of traits require a great deal of elucidation, since they are highly important in appraising the effects of radiations. They should be studied both in man and in other organisms. In this connexion. the prospects raised by the rapid advances being made on human biochemical specificities are of particular interest.

(*f*) *Selective factors in populations, with particular reference to the special conditions in man.*—Very little is known concerning the detailed effects of natural selection on the frequency of specific genes, constellations of genes, or cytological alterations. Such information is basic to attempts to understand the genetic composition of present and past human communities and to predict future trends consequent upon changes in radiation levels, medical practices, and social and economic conditions. These gaps in knowledge can in part be filled by the collection of relevant demographic and experimental data.

(*g*) *Patterns of mating in human populations and their genetic implications.*—A standard type of information always required in understanding the genetic composition of human populations and the effect on it of various amounts of radiation is the recording and interpretation of data on the consequences of inbreeding, assortative mating, geographical and cultural isolation and random genetic fluctuations.

(*h*) *Twin studies in man.*—These are recognized as being helpful in understanding many problems of human heredity. Such studies have already been extensively used but could be advanced by standardized registration of twins in various countries. They give useful information concerning the relative importance of hereditary and environmental influences.

(*i*) *Determination of the frequency of diseases with a significant genetic component, with particular reference to their epidemiology.*—This is fundamental for investigations on the significance of mutation as a cause of disease in man.

In this connexion central registration of human inbreeding, hereditary disease and variation is of the utmost importance. It is also of importance to know the number of people who on account of hereditary lesions have to be treated in hospitals or institutions or given social aid.

(*j*) *Study of populations of special genetic interest.*—Important information is to be obtained from the study of relatively stable, primitive communities, long isolated by geography or culture. Studies of this type require for their execution teams of persons from a variety of disciplines, such as cultural anthropologists, physicians and geneticists. It should be emphasized that the understanding of the genetic structure of contemporary populations will be greatly aided through these studies, which should be maintained continuously over a considerable period of time. The opportunity for these studies diminishes with each passing year. Among special communities to be studied are those receiving unusually large amounts of radiation, those in which the degree of inbreeding has long been very high or low, and those in which special conditions of selection have prevailed. In some investigations radiation physicists would be essential members of the teams.

(*k*) *Genetic mapping of human chromosomes.*—This is a highly specialized field in which encouraging advances are now being made. Among the possibilities to be exploited is the use of these data to aid in the identification of independently occurring mutant genes and in the study of chromosome rearrangements.

(*l*) *Cytochemistry and human cytology.*—Direct cytological observations should be conducted both on normal individuals and on those with suspected chromosomal abnormalities. Material from the individuals themselves as well as mutant cells of tissue cultures may be used in such work. Basic information concerning the ultramicroscopic structure and chemical composition of the hereditary material, and the manner in which this is altered by irradiation and other mutagens, is essential and should include information on lower organisms as well as man. The new developments in biochemistry, the emerging immunobiochemical investigation of tissue proteins, bone marrow and other tissues, the metabolic investigationes which may elucidate both physical and mental pathology, the new developments in electronmicroscopy which advanced our knowledge of the structure of human sperm all indicate the devolpment of new tools for the study of human genetics.

(*m*) *Development of further statistical methods.*—New mathematical methods have continually to be developed to deal analytically with problems which arise as the result of researches in human and in experimental population genetics. This is particularly so in relation to observations on the genetic structure of and intensity of selection in populations with regard both to traits due to single gene and those due to multiple gene effects. Special techniques requiring electronic computers will also be required for analysing data on genetic linkage in man.

5. SOME CONCLUSIONS

(*a*) The Group is of the opinion that there are too few institutions or large university departments devoted to general genetics and even fewer concerned with human genetics. It recommends the establishment of such institutions and departments and suggests that there could be no one ideal pattern. One of the benefits of such institutions would be to accustom people of different scientific disciplines having implications for genetics to work together. Physicians, general biologists, geneticists, biochemists, cytologists, serologists and statisticians are examples of the find of workers who may be needed. When such institutions are concerned with human genetics their location should have regard to the adequacy of existing medical services, to the kind and size of human populations available for field studies and to the adequacy of background vital statistics and general demographic information of the population concerned. For many purposes a population of about two million is optimal particularly for intensive epidemiological investigations. Such institutions, in addition to their research functions, could eventually serve as centres of elementary and advanced training in genetics.

(*b*) Such research departments and institutions should contribute much to teaching in general and human genetics. Medical undergraduates should all receive training in genetics and the teaching should be co-ordinated with that in radiology and in the use of radioactive substances in medicine, so that the genetic hazards of diagnostic and therapeutic procedures are thoroughly understood. Medical men training as radiologists should have specific, more advanced instruction in genetics. Health physicists, radiological physicists and radio-

logical physicists and radiological technicians should also receive instruction in genetics as part of their technical training.

It seems essential that instruction in genetics should be given to all scientists, particularly those whose work is likely to involve the use of radiation and radioactive materials in research. The principles of human genetics could with advantage be conveyed to those training in the social sciences by means of formal instruction. Finally, the Group is of the opinion that public education in genetics should be more common and adequate than it is at present.

(*c*) In the future it will be necessary from the point of view of preventive medicine and genetic hygiene to register serious hereditary diseases and defects in various populations or countries in the same way as, for instance, epidemic diseases. For that purpose, genetic hygienic ascertainment or registration will be an indispensable and necessary step. The recording of hereditary diseases and defects in various countries and regions is to be highly recommended.

(*d*) In may countries there are very few biologists or physicians properly trained in genetics. This situation will only be solved by producing more career opportunities in genetics, but may be alleviated by granting fellowships or subsidizing training at approved institutions in countries which can offer training facilities. It is possible, also, that advice and technical assistance could be given in connexion with research projects in countries with insufficient rosurces in trained manpower to carry them out.

(*e*) It might be possible for a United Nations Agency to assist on request in administration or supervision of studies of specific populations over a period of years or by strengthening a research team or by giving advice on organization.

(*f*) In the past, United Nations Agencies have done useful service in contributing to the collection and standardization of vital and health statistics. It is recommended that such agencies continue their efforts and stimulate the efforts of others in the collection and publication of specific data such as fertility, consanguineous marriages and parental ages, which are so essential as background information in many studies in human biology.

(*g*) The Group wishes to call attention to the evidence that damage to body tissues produced by radiation after relatively small doses is, at least in part, mediated through effects on genes and chromosomes. There is also some evidence that the life-span may be reduced in mammals even by relatively small doses. *Ad hoc* investigations are urgently needed.

(*h*) The Group is particularly impressed with the genetic hazards of man-made radiation from sources used in medicine, industry, commerce and experimental science, etc. Both as an approach to control and as providing basic background information for relating quantitatively radiation exposure and effects on man, it is essential that methods be found of recording exposures to individuals and populations, however difficult this may prove.

There is reason to believe that radiation exposure can be much reduced, therefore, those in charge of sources of ionizing radiations should always ensure that there is adequate justification for exposing individuals to doses however small. On account of the danger to offspring resulting from irradition of the gonads by X-rays, consideration should be given to determining what efficient means of shielding the gonads could be devised and brought into general use. In addition, in every exposure the X-ray beam ought as far as practicable to be directed so that a minimum of radiation reaches the gonads.

Annex 1

Damage From Point Mutations in Relation to Radiation Dose and Biological Conditions [1]

(Formerly entitled "The conception that mutations accumulate following repeated irradiation")

1. ACCUMULATION

One topic which I have been requested to discuss is that of the accumulation of point mutations following repeated irradiation. An accurately additive

[1] Submitted by Professor H. J. Muller, Department of Zoology, University of Indiana, Bloomington, Indiana, USA. (This paper is a considerably modified version of that presented at the Study Group on the Effect of Radiation on Human Heredity.)

accumulation in the germ cells throughout life has as its necessary and sufficient conditions (*a*) that the induced mutations are stable, i. e. not subject to repair, (*b*) that there is no important amount of intercellular selection to alter the relative frequencies of the mutant and non-mutant cells within a given individual during his lifetime, and (*c*) that radiation given at one time does not by some long-term aftereffect influence the mutagenicity of cells irradiate at a later period. These questions will be considered in turn.

(*a*) Changes of a point-mutational nature induced by radiation have not shown, as a class, unusual instability as compared with those arising spontaneously. Although the possibility is not excluded that there may be a relatively short period, of the order of one or a few cell cycles, before a mutation becomes fully completed and permanent (as in the work of D. Lewis, 1951, on *Oenothera*), this circumstance would not in ordinary cases affect the accumulation process.

(*b*) As for intercellular selection, except for the special case of drastic lethals arising in the X chromosome of a male, which have been shown in a series of experiments with Drosophila (by Kossikov, 1936, Shapiro, 1936, etc.) to be subject to selective elimination in spermatogonia, there is no reason to expect point mutations of the usual "recessive" sort, appearing heterozygously, to influence the multiplication or survival of immature germ cells appreciably. That mature germ cells are not thus influenced was shown long ago by Muller & Settles (1927). The most pertinent evidence on this point as regards immature germ cells, in an organism related to man, is given by experiments carried out by Russell (1951, 1956) in mice to test this very question. The failure of the mutation rate to decline in groups of offspring derived from spermatozoa ejaculated at increasing intervals after spermatogonial irradiation, shows both the absence of germinal selection against the mutant cells (point b) and the essential permanence of the mutant genes (point a).

(*c*) Direct tests of the accuracy of accumulation of lethals induced in Drosophila spermatozoa have been made by comparing their frequency at a given total dose after one treatment concentrated into a short time with that after a divided treatment of the same intensity and after a protracted treatment delivered at a low dose rate. It was found that the frequency depended on the total dose regardless of its distribution in time. When the diverse experiments of this kind carried out by different investigators (see review by Muller, 1954b, page 278, citing work of Patterson, Timoféeff-Ressovsky, Ray-Chaudhuri, Makhijani, Stern and others), are all taken into consideration together, it is found that the time-intensity relation was varied over a range of about 300,000 times, without any influence on the frequency of the mutations produced. Thus, a dose delivered in divided or protracted form over a month's time was as effective as one of the same total amount given in a few minutes. Tests have also been carried out, by Kerkis (1938), by Timoféeff-Ressovsky (1934), and recently by Oster (1955), that showed an additive relation when irradiation was given successively at two widely separated stages, to the immature and mature male germ cells respectively.

The reservation must be made that mutations not of the point variety, that is, those involving gross structural changes of chromosomes, which result from a combination of two or more independently produced chromosome breaks (Muller 1938, 1940) do, as expected, show an increase in frequency when the radiation is delivered in more concentrated form, provided union of the broken ends of the chromosomes can occur to an appreciable extent during the time of the longer treatment. This condition does not hold in mature spermatozoa, the type of cell used for most of the above-mentioned timing experiments, for union of broken ends cannot occur during this stage (Muller, 1940), but it does hold in other germ cells, in which therefore more lethals of the structural type result from concentrated than from very protracted or divided treatments (Herskowitz & Abrahamson, 1956). In the experiments cited in the preceding paragraph in which both immature and mature male germ cells were used there was no test of this matter, since the intervals between irradiations were long enough to avoid interactions between the effects of different exposures.

On the other hand, in gonial cells, which allow union of broken ends during treatment, relatively few of the mutations are of the "structural" type anyway. Moreover, low doses or dose-rates, such as those ordinarily encountered in human occupational exposures, produce relatively few structural changes as compared with point mutations even in the cells (spermatids and spermatozoa) most susceptible to their production, and produce still fewer in gonia. It must further be noted that at these low doses or dose-rates the rare structural changes which

do occur must in most cases have had both or all of their constituent breaks arising as effects of the same fast particle. The frequency of these changes would therefore, in such case, be independent of the time distribution of the irradiation. For these reasons, conditions would seldom be encountered, except in oöcytes, that resulted in overall frequencies of mutations (counting, together, both those of a point and of a grosser nature) differing perceptibly from those expected on an additive relation to the radiation dose. And when point mutations only were considered, the relation would be accurately additive.

2. LINEAR RELATION TO DOSE

Another expression of this additive relation, in the case of point mutations, is shown by the linear dependence of their frequency on radiation dose. That lethals induced in Drosophila spermatozoa do vary in frequency in this way has been abundantly shown for moderate and low doses, at which most of them are point mutations, in a great array of investigations, beginning with those of Hanson & Hayes (1929) and of Oliver (1930) and proceeding through many others to those of Uphoff & Stern (1949), which brought the dose down to 50 and 25 r. In experiments involving a lesser range of dose applied to spermatozoa of Drosophila, visible, non-lethal mutations, which include fewer structural changes than lethals, were found by Timoféeff-Ressovsky to show a linear relation to dose, and a linear relation for them was likewise found by the Indiana group when appreciable structural changes were excluded by cytological examination. Russell has also found a linear relation for visible mutations resulting from the irradiation of the spermatogonia of mice with moderate doses. A linear relation for visible mutations in higher plants was found by Stadler (1928) and in lower plants, for moderate doses, by Hollaender and others (see previously cited review).

It is true that in occasional experiments with very low doses results different from those expected on a strictly linear relation have been obtained. For instance, too few induced lethals seemed to be obtained by Caspari & Stern (1948) and too many induced visibles by Bonnier & Lüning (1949). However, these experiments were carried on at levels of dose so low that small sources of error had a relatively great effect. These sources of error include, in the case of visible mutations, differences in the degree of adverse selection against the mutants as between the control and treated series, caused for instance by differences in degree of crowding. In the case of both lethals and visibles, the numbers of mutations obtained at these doses are so low as to have a relatively large statistical variation. Moreover, the proportion of those obtained which were induced by the radiation is subject to a far higher error still, since it is represented by the difference between the frequency found at the low dose and that found in the control material. Inasmuch as at doses of 25 and 50 r the spontaneous (control) frequency may be a good deal higher than the induced frequency the error of this difference may be relatively enormous. This is especially true because the spontaneous frequency itself is subject to much more variation than that of random sampling. One source of such variation lies in the origination of mutations in clusters of common origin, caused by mutations in early germ cells. Another lies in the great differences between the spontaneous mutation rate existing in different lines, which may be as great as an order of magnitude and give evidence of being caused by genes (Muller, 1928), now called "mutator genes". Finally, both the spontaneous and the induced mutation rates vary considerably according to the history of the germ cells used (e. g. Muller, 1946, Lüning, 1952). Very special techniques are necessary for minimizing these various sources of error.

In view of these difficulties it is not surprising that experiments to test the linear relation have not yet been pushed below 25 r. However, genetic and other techniques have over the course of several years been worked out at Indiana which should now allow significant results to be obtained at doses as low as 10 or even 5 r. Work on the necessary scale would require the co-operation of a group working over some two years and examining several hundred thousand cultures, a project that we estimate might cost some $18,000. We are not especially desirous of carrying it out ourselves since even if the necessary support were provided the work should inevitably entail much digression from our other activities. We should therefore be glad to co-operate in furnishing the stocks and techniques and aiding in the supervision of the work if it were to be carried on elsewhere, although if no other suitable place could be found for the project we would not exclude the possibility of our conducting it.

The fact that the relation is linear, even at 50 r, and even when the irradiation of sperm cells is protracted for several weeks, makes it very probable already that the relation is linear all the way down to zero. For in some of this work it can be shown that there must have been hours between the traversing of a sperm cell by one ionization track and its traversing by another. If, however, the linear relation can be pushed down to doses as low as 5 r (or if at this dose the frequency can merely be shown to be more nearly proportional to the dose itself than to its 1.5 or .5 power), then we would have a justification for concluding with a very high degree of assurance that the relation was indeed linear all the way down to zero. This is because the ionizations are not produced separately but occur in the course of the tracks of the fast ionizing particles (the released electrons). Thus the ionizations come in spurts and a cell either gets a spurt or it does not. With very low doses, such as 5 r or less, an individual spermatozoon would hardly ever be traversed by more than one track, that is, it would not have received more than one spurt. Hence lowering the dose would not have the effect of lessening the number of ionizations in cells that received a spurt but only of lessening the number of cells that received any spurt at all. For these very low doses, then, the mutation frequency would be proportional only to the number of cells "hit," which is necessarily proportional to dose. Therefore we could justifiably extrapolate the results from 5 r linearly all the way down to zero. We need only make the one proviso here that the mutations produced in a cell by ionizing radiation result from ionizations or activations arising in that cell itself, not from those in the medium, but there is evidence from other work (see Muller 1954b) that this is true for mutations produced by ionizing radiation in Drosophila.

3. INFLUENCE OF LOCAL CONCENTRATION OF ACTIVATIONS

Even if we assumed the linear relation to hold all the way down to zero for X-rays and gamma-rays, this still would not mean that a given mutation necessarily results from just one ionization or excitation. For, many of the ionizations and excitations are grouped together in small clusters in the course of the tracks of the fast particles and it is possible that a cluster rather than a single quantum change is usually required to cause a gene mutation or chromosome break. At first sight it might be thought that this view is contradicted by the lack of influence of intensity changes on the dose-mutation rate relation, inasmuch as this result indicates that a given number of nearby ionizations when crowded together in time are no more mutagenic than when scattered in their time distribution. However, this inference is inapplicable to the question at issue, because the crowding attained in this way is much less than that within the minute clusters formed in the course of the track of a fast particle. That a cluster of such density is in fact more effective mutagenically than the same number of scattered activiations is shown by recent work (e. g. Ives, Mickey, Muller, all 1954) proving the higher effectiveness of neutrons than of X-rays in producing both point mutations and chromosome breaks. Other evidence to the same effect lies in the lower mutagenic effectivenes apparently shown by betatron radiation having an energy of about 15 Mev, as compared with ordinary Xrays, inasmuch as the radiation of higher energy is thought to result in a somewhat lesser amount of clustering than do ordinary X-rays (Herskowitz, Muller & Laughlin, 1956). I am inclined to view the greater effectiveness of more densely crowded activations in terms of the Watson-Crick model of chromosome structure, by supposing that a hit on both complementary strands at corresponding, or nearly corresponding, points is more likely to result in a permanent alteration in the chromosome than when just one of the strands is changed.

4. COMPLICATIONS AT HIGH DOSES

The breakage of chromosomes by radiation complicates in more than one way, at high doses, the relation between radiation dose and the observed frequency of visible or lethal mutations. For one thing, the ensuing chromosome abnormalities often kill the affected cells or their descendant cells by causing chromosome bridges at a subsequent mitosis, and, short of such an effect, can lower the multiplication rate of the descendant cells or even kill them by means of the resulting aneuploidy (the abnormal proportions existing between different chromosome parts). This circumstance would not in itself affect the observed frequency of point mutations were it not for the fact that germ cells in different

stages of the reproductive and mitotic cycles differ from one another in their susceptibility to having their chromosomes broken, and that they differ in a parallel manner in their susceptibility to having point mutations induced within them. At higher doses there is necessarily more killing off of the more susceptible cells, relatively to the less susceptible ones, by means of chromosome changes, than at lower doses (as well as more reduction of the multiplication rate of those not actually killed). Now, since the cells of the groups more injured in this way are also the ones that have had more point mutations produced in them it follows that at high doses there is more selective elimination (or reduction in relative numbers) of the germ cells containing point mutations as compared with the unmutated ones than there is at low doses. Hence, at higher and higher doses the frequency of point mutations observed among the offspring will fall further and further short (in a relative sense) of the frequency with which the point mutations had actually been produced, and the graph of the observed results will bend down ever further from the straight line extrapolated from the data obtained at low and moderate doses.

It is evident that the more heterogeneous in the susceptibilities is the lot of irradiated germ cells from which the given offspring are derived, the more pronounced will this falling off from linearity be. A very marked illustration of this effect, involving only a one-and-a-half-fold increase in observed lethal mutation frequency with a fourfold increase in dose (from 1000 to 4000 r) was obtained (Muller, Abrahamson, Herskowitz & Oster, 1954) by taking offspring from copulations of Drosophila males that had occurred seven to 10 days after their irradiation as just hatched imagos. The reason the effect was here so marked was because, as Lüning's already mentioned work had shown, the germ cells released during this period were at the time of irradiation in a number of different stages, having widely different susceptibilities. Although the irradiation of a quite homogeneous lot of germ cells would, theoretically, fail to give rise to any effect of this find, this has so far remained, in Drosophila, an ideal situation, that has probably not been obtained in practice.

Even gonial cells are of differing mutagenic susceptibilities, depending, for one thing, upon whether or not they happen to be in mitosis at irradiation. As Oster (1954) has shown, gonial cells containing the condensed chromosomes of mitotic stages (produced in this case by colchicine or acenaphthene treatment) are, like other cells with condensed chromosomes, more susceptible to radiation mutagenesis. This fits in with Russell's finding that the mutation frequencies found on examination of mice derived from irradiated spermatogonia, although linear for the dose range 300 r to 600 r, fell markedly below the expectation for linearity when a dose of 1000 r was used.

In organisms such as Drosophila and, probably, moulds, in which mutations of visible or lethal expression can arise in connexion with gross structural changes of chromosomes, either as position effects or as deficiencies, the complication exists that the frequency of these structural changes rises more rapidly than the dose (approximately as its 3/2 power, Muller, 1938, 1940). The observed mutants, unless analysed for gross structural changes, will represent a mixture of these and point mutations (the latter in turn consisting of gene mutations and minute structural changes, both of which vary linearly with the dose). Thus at lower doses, where the great majority of the mutations are in the point category, the frequency will be linearly related to dose, but at high doses, where the gross structural changes become numerically important, it might be expected that the overall frequency of lethal and of visible mutations would gradually rise, to approach the 3/2 power relation. Just this is seen in the results for visible mutations observed by Stapleton, Hollaender & Martin (1952) after irradiation of spores of the mould *Aspergillus*, whereas the offspring obtained after irradiation of mature Drosophila males have in most experiments seemed to show a linear relation for lethal and for visible mutations even at high doses. The interpretation of this at first sight paradoxical result is doubtless to be sought in the fact that in these experiments with Drosophila the germ cells used had been heterogeneous enough when irradiated to result in a tendency of the frequency to fall from linearity, in consequence of selectice elimination of the products of the more susceptible germ cells, and that this tendency largely compensated for the rise above linearity that would otherwise have been produced by the ever greater relative numbers of structural change mutants arising at the higher doses.

Because of these complications results with high doses are apt to be erratic and difficult of anlysis. Thus observations with moderate doses are better suited for arriving at an understanding of the fundamental frequency-dose relationship.

5. INFLUENCE OF CELL TYPE ON INDUCED MUTATION RATE

It has long been known (e. g. Stadler, 1928, Muller, 1930) that cells of different types or tages differ considerably in their susceptibility to mutagenesis by ionizing radiation. Although gross structural changes of chromosomes show the most variation in frequency with cell type, point mutations (including what are probably changes within a gene as well as minute deficiencies and rearrangements of one to a few genes) probably have a frequency range of at least fourfold when a given dose is applied to different types of germ cells. This is to be concluded both from results on lethals arising at moderate doses (at which relatively few of the changes are in gross chromosolne structure) and from visible mutations found by cytological observation to be free of discernible changes in the chromosomes.

Putting togther the results of earlier and later studies (see review previously cited and also recent papers by Bonnier & Lüning, 1953, Telfer, 1954, Abrahamson, 1956, and Oster, 1956), the early germ cells and gonia have the lowest frequency of induced point mutations yet the highest ratio of point mutations to changes of any kind that can be demonstrated to be structural (i. e. in these cells the structural changes fall to a minimum which is relatively much lower still). At these stages, the mutation frequency and distribution of types is much the same in male and female. In the later male germ cells, the overall mutation frequency, including that of recessive lethals, rises to a sharp maximum during the period of spermatid formation and transformation (although we must omit the preceding meiotic stages from consideration here as not being well enough known in this respect). Lüning has given reasons for inferring that much or all of the exceptionally high frequency of recessive lethals induced in the spermatid period involves those connected with gross and minute structural changes of chromosomes rather than true gene mutations. The overall mutation frequency, including that of recessive lethals, then falls sharply from the spermatid period to a second minimum to the immature spermatozoa (a minimum not nearly as low, however, as the preceding one in the gonia), only to rise again within the next few days until the time of ejaculation. After insemination, within the reproductive tract of the female, the male germ cells attain and maintain at a relatively constant level their highest known frequency of recessive lethals as well as of demonstrable structural changes, except for that found in the spermatids.

In rodents, the fact has long been known that ionizing radiation has a far more damaging effect on the genetic material when applied to mature or nearly mature male germ cells than when applied to immature ones (gonia), as judged by the killing of the resulting embryos. It remained for Snell (1935) to provide evidence that these effects, and the inherited "semi-sterility" which he found also to be induced in mice, were caused by gross structural changes of chromosomes, a class of effects with which we are not primarily concerned in this paper. Later, however, evidence was obtained by P. Hertwig (1941) that at those same stages there is also a relatively high frequency of production of point mutations by ionizing radiation, just as was known to be true in Drosophila. Fortunately, in man, the period during which the germ cells of the male remain in the gonial stage exceeds by over a hundred times that of the spermatid and spermatozoon stages, so that the high susceptibility of the latter stages presents a relatively minor practical problem. Thus it is the less mutable gonia of mammals, studied mainly by Russell, which are of greater interest in assessing the genetic damage produced by radiation in human populations. As noted in section 3, however, gonia themselves do not constitute one homogeneous class so far as susceptibility to mutagenesis is concerned, but may differ considerably, according to their developmental and mitotic stage and perhaps also their physiological condition.

As for the female germ cells, the point mutation frequency in the late oöcytes of Drosophila, during the last three or four days before ovulation, attains a level almost as high as that in the nearly mature unejaculated spermatozoa, when high doses of radiation are used (Muller, H. J., Valencia, R. M. & Valencia, J. I., 1950). However, in the previously mentioned work of Herskowitz & Abrahamson it was found that lethals induced at this stage show dependence on a higher power of the dose than 1, and on the timing of the dose, as well as other peculiarities, all indicating that a high proportion of them consists of small structural changes involving two independently produced chromosome breaks. These mutations (like many of those induced in spermatids and spermatozoa),

although not strictly point mutations, must usually be classed with them operationally since the making of the distinction is commonly impracticable or even impossible.

In mammals the germ cells of females may, according to one view, remain for a long time in a stage corresponding to the late oöcytes of Drosophila. It will therefore be important to determine to what extent mamalian female germ cells follow similar principles to those of Drosophila late oöcytes in regard to induced mutations. If they are long in such a stage, we should have to admit a notable departure from linearity for female germ cells. Whatever the answer may be, however, it is to be expected that for low doses, as for most occupational and diagnostic exposures, the frequency would be linearly proportional to dose even in late oöcytes (because of any given mutagenically sensitive region being so seldom traversed by more than one track), and that the frequency for a given low dose would not be lower in them than in gonia.

That somatic cells, like germ cells, can have point mutations induced in them by ionizing radiation was first shown by Patterson (1928), using Drosophila embryos and larvae. Calculations which I made on the basis of the early results, confirmed by studies by Timoféeff-Ressovsky (1929), and more recently by Lefevre (1950), show that for given genes the frequency of point mutations is similar to that obtained for gonia, or perhaps somewhat higher. This point is of importance in considerations of those effects of radiation on the exposed individual himself, such as leukaemia and other malignancies, which might have their basis in point mutations of his somatic cells.

With the development by Puck and his co-workers of methods of culturing and subculturing human somatic cells like micro-organisms, finding and breeding lines of mutant cells (1956b), and determining the effects of different doses of ionizing radiation (1956a), the way has now been paved for carrying forward to man the exact study of the induction of point mutations and other genetic changes in somatic cells. As an early result from this study, some evidence has already been adduced (Puck et al., 1956a) that the killing effect of the radiation on the cells is, as was to have been expected, caused by chromosome structural change rather than point mutation. It is probable on a number of grounds that this genetic killing of individual cells and genetic impairment of others, caused by gross chromosome changes, lies at the basis of much of the damaging effect of radiation on the body of the exposed individual, such as epilation, leucocytopenia, destruction of the intestinal lining and other manifestations of radiation sickness, production of cataracts, retardation and distortion of growth, reduction of regenerative capacity, and (probably the most important effect) reduction of the life span (see discussions by Muller, 1950b, 1956b, Quastler, 1956, Sacher, 1956).

6. ESTIMATION OF TOTAL DAMAGE FROM POINT MUTATIONS

The prime questions regarding the damage done to posterity by a given amount of radiation are, what will the total amount of that damage be, and how will it be distributed? In our previous treatment we have discussed how the frequency of lethal or visible mutations varies with dose and with type of cell, but we have not considered the absolute frequency of such mutations for any given dose, still less the total frequency of mutations of all kinds. It is this total frequency that counts. For, as long ago shown by Haldane (1937) and as later developed by Muller (1950a), in a population at mutational equilibrium (i. e. a population in which about as many mutant genes are dying out in each generation through death or failure to reproduce of the individuals containing them as are arising anew through mutation) the average reduction in fitness of an individual lies between the total frequency of all detrimental mutations, counting equally those with large and those with small effects, and twice that frequency. If all the mutant genes were strictly recessive the lower figure (the mutation rate, μ, itself) would apply, whereas if they were all dominant enough to be eliminated as heterozygates the figure would be wice this (2μ). As Muller (*ibid.*) pointed out there is good reason for inferring the higher figure, 2μ, to be nearly correct both in Drosophila and in man. This same figure for reduction of fitness would on the whole express the proportion of individuals in the population who would have to suffer "genetic death" (selective elimination by dying before maturity or failure to reproduce) to maintain the genetic equilibrium. Some reduction of the figure for elimination rate (probably by not more than a factor of 2) might, however, have to be made to allow for some synergistic operation by

detrimental genes: a mode of action giving individuals with multiple defects a lower survival rate than the product of the survival rates of those with the separate defects.

In estimating this total mutation rate for practical purposes only point mutations need usually be considered, since the great majority both of spontaneous mutations and of those that would be likely to be produced by radiation in a human population are of this nature. The first approach toward determining the total mutation rate in any organism was made independently and simultaneously in 1934–35 by Kerkis working in collaboration with myself and by Timoféeff-Ressovsky (see his and my review papers already referred to), using descendants of irradiated Drosophila males. Special techniques were used for the detection of mutations having neither a visible nor fully lethal effect, but only reducing the expectation of survival to maturity: the so-called detrimental mutations. Both pieces of work agreed that these mutations arise some three to four times as frequently as the fully lethal mutations. Essentially similar results have recently been reported by Käfer (1952), working under the guidance of Hadorn, and Falk (1955), working under the guidance of Bonnier.

It is admitted by all these investigators, however, that there had been little chance, by their techniques, of detecting mutations that reduced survival up to maturity by less than some 5–10 per cent. Moreover, there must be many mutations, undetectable by these techniques, the detrimental effect of which occurs mainly after maturity is reached or which affect reproductive capacity rather than individual survival. Thus the estimate that in Drosophila there are some five times as many harmful mutations altogether as the number of lethals, and some 30 times the number of sex-linked lethals, is a bare minimum, possibly only half the true value. It now becomes of great importance to extend the range of detected mutations to those with still less effect, and with other types of effect, so as to throw light on the extent to which the present estimate should be raised. As in the case of the proposed investigation of low dosage, we have for some years been developing techniques for such an attack in Drosophila, but again the work would necessarily be on so large a scale that group work and considerable expenditure (comparable in magnitude with that for the low dosage project) would be required.

In absolute numbers the above estimate becomes for a dose of, say 100 r applied to spermatozoa of young Drosophila males a day or two before their mating, or applied to late oöcytes, about one induced mutation in every 12 germ cells or one in six offspring. Thus a continuation of this exposure, applied to both sexes through many successive generations, would reduce the average fitness of the individual in the equilibrium population by about a sixth (some 17 per cent.) and would cause nearly one individual in six to meet "genetic death" in consequence of the irradiation. It can further be estimated (see below) that the total effect of spontaneous mutations in Drosophila is about half as much as this; that is, the given amount of radiation, applied at the stages specified, would constitute about twice the "doubling dose". But it should be borne in mind that these present estimates are in both cases minimal ones.

7. MANNER OF DISTRIBUTION AND EXPRESSION OF THE TOTAL DAMAGE

How does this mutational damage become distributed and expressed among the descendants? The amount of damage done by any given mutant gene in a heterozygous descendant may be represented as the amount of detrimental effect it would exert when homozygous multiplied by its amount of dominance (the ratio of its effect when heterozygous to that when homozygous). Now the dominance of lethals in Drosphila has been found both in work of Stern and his co-workers (see Stern et al., 1952) and of the present author and Campbell (see Morton, Crow & Muller, 1956) to average about 0.04 to 0.05, so that even these mutant genes with extreme effects would individually reduce viability in the heterozygote by only some 5 per cent. The merely detrimental genes are suspected on theoretical grounds (Muller, 1950a) to have somewhat more dominance than the lethals, and there has recently been some direct evidence for this (Falk, 1955), but even when considerable allowance is made for this possibility the effect exerted in a heterozygote by a detrimental is expected, on the average, to be less, absolutely, than that exerted by a lethal. Thus, taking individual mutant genes of all degrees, they should average well below five per cent. in individually lowering the fitness of the heterozygote. Since at the same time the visible effects of these genes in the heterozygote, *taken individually*, usually

escape notice, it follows that the effects of mutations induced by radiation in any one generation at a frequency comparable with that above considered would not ordinarily be observed among the next or any subsequent generation. Nevertheless the total loss of fitness in the next generation, being about one in six (the minimum frequency of offspring with newly induced mutations) times, say, one per cent. (to take a bare minimum for their average expression in heterozygotes) would in a population of 1 000 000 entail the "genetic death" of at least 1700 individuals of that generation. Moreover, a comparable amount of damage would continue to be exerted for scores of generations.

The number of generations that a mutant gene persists before causing genetic death is on the average about the reciprocal of the amount of damage it does to the heterozygote, so that the average Drosophila lethal in an autosome might be expected to persist for some 22 generations. However, the average persistence of a group of mutant genes is the *harmonic*, not the arithmetic, mean of the persistence of the individual mutant genes, and this value for the Drosophila lethals investigated turns out to be about 50 generations, though with a high error (see Morton, Crow & Muller, 1956). The persistence of detrimentals must be even greater. This is the so-called accumulation figure representing not only the average persistence of the mutant genes arising in a given generation but also the average amount of overlapping, within the individuals of any given generation, of mutant genes that arose in different generations, provided that the same mutation rate has existed in successive generations for a long period and mutational equilibrium has therefore been established. Hence if the 100 r exposure above postulated has been applied to Drosophila for many generations it is to be expected that each generation would be damaged at least 50 times as much as above calculated for the first generation of offspring (in fact, by an amount equal to 2μ or in this case 17 per cent.) Moreover, instead of one individual in six carrying a mutant gene induced by the radiation each individual would contain at least 50 X 1/6, or at least eight of them, on the average. Thus, although the effects of the mutant genes would seldom be individually noticed their collective effect would in the great majority of individuals be quite appreciable. It would of course tend to give a different pattern of impairment from one individual to another.

8. THE INDUCED IN RELATION TO THE SPONTANEOUS MUTATIONAL DAMAGE

The damage caused by the induced mutations is of course intermingled with that caused by spontaneous mutations. Although the amount of the radiation-induced mutational damage is largely independent of that caused by the spontaneous mutations it is helpful, in grasping its meaning, to compare it with that of the naturally existing mutational impairment since a species is in a sense adjusted to the latter and since, in man, we have a rough pragmatic familiarity with it. For this purpose it is desirable to be able to express spontaneous mutations in the same terms as those used above for induced mutations, namely, in terms of total mutation rate and loss of fitness. This is easily done, once estimates of these total values have been made for the *induced* mutations occurring at some given dose, provided only that the frequency of some particular group of mutations, e. g. sex-linked lethals, or visibles of a given collection or category (but preferably not those confined to just one allele series) has been determined under comparable circumstances both in unirradiated and in irradiated material. For there is good reason to believe that, for point mutations, the following relation will approximately hold: spontaneous total mutations/spontaneous mutations of given category-induced total mutations/induced mutations of same category. Thus, if figures are obtainable for the last three terms, the first one (the spontaneous total) can be solved for. The particular category best determined and most used for this purpose in Drosophila work has been that of sex-linked lethals.

Any *one* particular allele series (or "locus") cannot be relied upon by itself for the above purpose because the frequencies of mutation to different series may not bear the same relation to one another for spontaneous as for radiation-induced or otherwise induced mutations (see e. g. Giles, 1952). However, there is no reason to suspect that any broad phenotypic category or section of chromatin, or a whole group of allele-series chosen for their technical convenience, will show any consistent preference as between spontaneous and radiation-induced mutability. Experimental evidence that there is no such differential susceptability in Drosophila was obtained in the observation, by Timoféeff-Ressovsky (1937),

myself (see Patterson & Muller, 1930), and others, of the similar ratio of sex-linked lethals to sex-linked visibles both in unirradiated and irradiated material (especially when allowance is made for the relatively higher frequency of deficiencies and other structural changes after irradiation).

As noted above, the ratio of "total" mutations to sex-linked lethals in Drosophila when radiation is used has been estimated to be at least 30, and we may therefore (in accordance with the preceding formula) multiply the spontaneous sex-linked lethal frequency by 30 to obtain the spontaneous total. The problem arises, however, of what observed value of the spontaneous sex-linked lethal frequency to choose. For this value has been found to vary by at least an order of magnitude from one experiment to another according to the stocks used (Muller, 1928, confirmed by later workers) and to vary by more than half an order of magnitude according to the developmental history of the germ cells (Muller, 1946 and unpublished), not to speak of variations caused by temperature and other environmental differences within the natural range. However, the upshot of a large number of studies of the spontaneous sex-linked frequency in Drosophila, by different investigators, has shown that the great majority of individuals bred at 25° C. under reasonably favourable conditions, in such manner that the germ cells used to produce the offspring do not give undue representation to those with extreme developmental histories, have a sex-linked lethal frequency averaging about 0.1 to 0.2 per cent. This is true in both sexes, but the female value appears to vary less with germ cell history and commonly to approximate 0.17 per cent. whereas the male value, which is higher (0.2 per cent.) for the sperm released very early, is a good deal lower (e. g. 0.06 per cent.) for those released in what might be called the prime of life. Taking 0.14 per cent. as a reasonable average and multiplying it by 30, our minimum figure for the total spontaneous mutation rate per gamete is 4.2 per cent. and that for the zygote is 8.4 per cent., a figure which also represents the average reduction in fitness or risk of genetic death as a result of spontaneous mutations. It was on the basis of this estimate that an irradiation of 100 r given to Drosophila in the manner specified in section 6 was there stated to constitute about twice the doubling dose, inasmuch as it had been calcuated to give an induced rate of 17 per cent. per zygote.

From the above it will be seen that, in Drosophila at least, there is much more uncertainty about the amount of spontaneous mutational damage, because of the high variability of the spontaneous mutation rate, than about that caused by any given amont of radiation applied to a known stage or group of stages. Because of this uncertainty, determinations of the spontaneous mutation rate of any particular category of mutants in Drosophila, such as a given group of "visibles", should always, in order to have significance in relation to other work, be accompanied by a yardstick indicating the general mutability characteristic of the material studied. At present the most convenient such yardstick is to be found in the sex-linked lethal rate, which must be ascertained under precisely the same conditions. Only when such a yardstick is provided can we, for example, use data on the frequency of spontaneous mutations of given types to estimate the ratio they bear to the total mutation frequency, or to the frequency of some other particular category, inasmuch as these other quantities themselves are properly expressed in relation to a corresponding yardstick.

It is true that the radiation-induced rate also varies to some extent according to the stocks used (see below), the environmental conditions, and the germ cell stages involved. These differences, however, are not of a type which would usually throw our reckoning off nearly so much as in the case of spontaneous mutations since there is more knowledge of how they may be allowed for. But they must be taken into account.

9. SPECIES DIFFERENCES AND THE PROBLEM OF EXTRAPOLATION

In view of the evidence already referred to of the variation of the radiation-induced frequency of point mutations in Drosophila according to the type of cell irradiated, and the abundant evidence that has been obtained in recent years of the influence of conditions associated with the irradiation, such as oxygen concentration, enzyme-inhibitors, etc., on the frequency (see author's review, 1954b), it would be strange if genetic differences failed to affect the result. Indeed, Dubovsky (1935) reported that some stocks of *D. melanogaster* from widely separated localities differed by a factor of about 2 in the frequency of lethals produced by irradiation of the male. It is true that such differences can

be produced in the same stock by slight differences in the timing of the germ cells used, a matter not then realized, and that stocks may also differ genetically in their natural timing, yet genetic differences of many kinds would be expected to be capable of influencing the result. In the light of these considerations, however, it is rather noteworthy that, contrariwise, even the specific difference between *D. simulans* and *melanogaster* was found by Kossikov (1935) not to be associated with a significant difference between the induced frequencies of lethals of flies of these two kinds. This similarity may indicate that the induced frequency, like the spontaneous one (see below), even though readily altered, tends to be maintained at a certain level by some active selective processes operating on features that, perhaps as a by-product, tend to maintain susceptibility to these mutagenic factors at the level found.

However that may be, it is not to be expected that widely different species, such as those of different phyla, would have similar induced or spontaneous mutation frequencies, either total or of any given overall phenotypic class and/or chromosomal type (such as sterility mutations or sex-linked lethals), nor that they would have a similar ratio of total mutation rate to that in such a category. One reason for this disparity is that the amount and distribution of the genetic material must differ enormously as between such organisms; another is that the processes whereby the genes reach expression must be so different that a superficial resemblance in effect would provide little or no indication of a homologous genetic basis. Thus even if the frequency of production of, for example, sex-linked lethals were known in a mammal, one certainly would not be justified in applying to this figure the Drosophila factor of 30 times, to estimate the total frequency of induced mutations in the mammal.

The case is, however, different when we use as our index of relative mutation rates in two widely different species a category consisting of the average frequency of origination, in each species, of members of a single allele (or pseudoallele) series, often called the "specific-locus rate", provided that this average has been determined through observations of a number of different series ("loci") in each species and that most of the values found for the different series of the same species show (as they have done) a tendency to be clustered within about one order of magnitude. The reasonable agreement between the results for some 12 different allele-series involving visible point mutations (including those that are at the same time lethal) after irradiation of spermatozoa of Drosophila Muller, 1954a and unpublished) and also for some seven series after irradiation of spermatogonia of mice (Russell, 1952, 1956, Kimball, 1956), justifies us in speaking of an average or modal induced mutability for such an allele-series in each species. We may then infer that differences in the detectability of the mutations of the different series, in the complexity of the genetic regions concerned, and in their actual mutability, are usually insufficient to cause inordinate discrepancies between the values for the different series.

In Drosophila the ratio between the "total" and the average single allele-series rate is at least 10 000 (e. g. Muller, 1955b) and is probably a good deal higher. This value has been obtained by multiplying the ratio of "all" detrimentals and lethals to sex-linked lethals by the ratio of the latter to the average single allele-series frequency. (These two constituent ratios have of course been obtained in different experiments, under different conditions.) Are we now justified in assuming that a mammal would have at least as high a ratio as a fly of the "total" to the average single allele-series rate, and may we therefore multiply the latter rate, as determined in Russell's irradiation experiments, by 10 000, to obtain the minimum value for the total induced mutation rate in mice?

The justification for this procedure lies almost entirely in general considerations. The main consideration is that a mammal, by no matter what criterion, stands at least as high in the scale of biological organization as a fly, and probably a good deal higher as judged by its complexity of gross and histological structure, physiology, and behaviour. It would therefore be surprising if the genetic basis of the mammal were not as least as complicated and, accordingly, compounded of as many parts (such as nucleotides) as that of the fly. This would imply also that it had at least as many, and probably more, different ways of mutating, and that any one allele-series, on the average, represented no larger, but probably a smaller, fraction of all the mutational potentialities in the case of the mammal than in the case of the fly. The several times greater DNA content of the mammalian than of the Drosophila chromosome-set tends to support this inference.

It is to be noted that this method of obtaining a minimum estimate of the total induced rate in the mouse avoids any assumptions regarding the means of defining the limits of a gene or locus, and the number of such entities. It is true that in the past the argument has usually been stated in terms of genes or loci (but see Muller, 1955, 1956a, 1957) but this has, for the present writer at least, been only a short-cut mode of expression. For, what was meant by the "specific locus" frequency was really the frequency with which mutations arose that were on operational grounds to be classed as being probably members of the same allele series, without assumptions being made as to what proportion of mutations actually occurring in the chromosome region in question would fall into the given allele category. Moreover, although 10 000 was sometimes stated to be a minimum value of the number of genes or loci, as estimated by several very different methods, the justification for using it also as the ratio of total mutations to mutations in one average allele series ("specific locus") was that, empirically, the experiments on detrimental mutations, lethals, and allele-series mutations had shown this ratio to hold, no matter what the number of genes may be, or how we define them. It is quite possible, for instance, that some of the same chromosome regions that gave rise by mutation to members of a given visible allele series also gave rise to lethals and/or detrimentals (which may or may not have been included in the count of the allele-series frequency, according to whether or not they also produced the visible effect that served as the criterion), but this was irrelevant to the determination of the ratio since all the lethals and detrimentals of sufficient detectability to be recorded as such were included in the measurement of the frequency of these classes and therefore in the "total" rate. Thus the only relevant questions concerning the validity of the extrapolation process for obtaining a minimum estimate are whether or not a sufficiently representative sample of allele series has been obtained, and whether we are willing to admit the probability of the proposition that the average allele series, as operationally defined, would constitute at least as small a fraction of the total mutation rate in a mammal as in a fly.

If we grant these points and apply our factor of 10 000 to Russell's observed allele-series rate of 25×10^{-8} mutations per r in the spermatogonia of mice, we find as our minimum estimate of the total induced frequency in this material 25×10^{-4}, which may also be expressed by saying that there is at least one mutation per germ cell for every 400 r. As for the human induced mutation rate, we can at present only say that this is what it would be if it were like that of mice, that there are no data from man as yet that are inconsistent with this, and that this rate is about an order of magnitude higher than the induced rate in Drosophila.

On the other hand, we do have for man, as well as for the mouse, some data that allow us to estimate the spontaneous mutation frequency for allele series. As this matter has recently been discussed elsewhere (Mueller, 1957), an appraisal of the validity of this evidence will not be attempted here, except to point out, first, that the determination for man has the advantage of being based on large-scale data that give, as it were, a cross section of results from different genetic lines and from different ages and conditions of reproduction, and, second, that the results of the different allele series agree reasonably well with each other and, what is more surprising, that their consensus agrees well with the average based on mice.

Here again, then, is evidence of the operation of selective processes that tend to stabilize the mutation rate, as was noted in section 8 in connexion with the radiation-induced rate. Even more striking evidence of this, in the case of spontaneous mutation is the unexpected similarity between both these human and mouse values for the spontaneous allele-series rate and that (in the neighbourhood of 0.5×10^{-5}) deduced to be characteristic of Drosphila. It is true that thus far there has only been one series of experiments (Muller, H. J., Valencia J. I. & Valencia, R. M. 1950) in which a considerable group of spontaneous allele-series rates in Drosophila has been directly determined and in which at the same time a yardstick (sex-linked lethals) was used so that the obtained rates could be converted (as proved necessary) into more typical ones. However, approximately the same figure had been reached earlier by taking the typical spontaneous sex-linked lethal rate and dividing it by the ratio found to hold between the induced sex-linked lethal rate and the induced allele-series rate. Moreover, confirmation of the order of magnitude of this value (although probably involving some reduction of the value itself) is now being obtained in another series of direct observations, checked by lethals, conducted by Schalet at the Indiana

Laboratory. In any case, such a correspondence between such different species tends to impart confidence in the estimated orders of magnitude.

When, now, the factor of 10 000 is applied to the estimated value for an allele series in man, taking for the latter the rather conservative figure of 10^{-5}, we find that the minimum estimate of the "total" spontaneous mutation rate turns out to be 0.1 per gamete or 0.2 per individual, a value higher than has commonly been suspected to apply to our own species.

10. LIGHT FROM ANOTHER SOURCE

Extrapolation of the type above discussed is not the only means of arriving at estimates of the spontaneous mutation rate in man on the basis of existing data. As explained by Morton, Crow and Muller in a parallel paper (1956, see also Crow, 1956 and Muller, 1957) several different studies of the mortality found among the offspring of consanguineous as compared with non-consanguineous matings in man agree reasonably well in giving evidence from which it can be deduced that the average human gamete carries a mutational load accumulated from past generations which if it became homozygous would be twice as much as needed to kill the individual containing it at some time between a late foetal and early adult stage. Much of this load is probably scattered among diverse mutant genes any one of which would, if homozygous, entail a relatively small risk of death. There must in addition be a considerable load of detrimental genes in the gamete that tend to cause death before or after the period studied, or that interfere with reproduction rather than survival. Moreover, in a population living under more primitive conditions than those studied, more genes would find such expression than did so in the given populations. Finally, the individual himself carries twice as many such genes as the zygote. All in all, then, the load carried, mainly heterozygously, by the zygote is probably (if expressed in terms of the damage it would do homozygously) as much as about eight "lethal equivalents."

Now this rather directly measured load does not in itself tell us anything of the mutation rate per generation. However, if there are means of obtaining a reasonable estimate, by extrapolation or otherwise, of the relative amount of expression which this load actually attains in the average individual (a matter dependent upon the degree of dominance of the mutant genes and of the frequency with which occasional homozygosity occurs), we should then have a value for the average reduction of fitness. As noted previously, this would be almost equal to μ (the total spontaneous mutation rate) if the eliminations in the given population are brought about mainly through the homozygous effects and almost 2μ if the dominance is enough for elimination usually to be caused by the heterozygous effects. Now although the data from man are insufficient to allow us to set a value for the average dominance of mutant genes there are considerations (pointed out in some of the above papers) that allow us to set some fairly reasonable limits to such a value. Moreover, the value found for Drosophila lethals lies well between these limits. It is also possible to arrive at reasonable limits for the frequency of homozygosity caused by inbreeding. If then we extrapolate by taking the value for dominance found in Drosophila, and at the same time use in our reckoning the human inbreeding factor, we reach a value for reduction of fitness of approximately 0.1 per gamete or 0.2 per individual. This in turn gives us, as the value for the "total" spontaneous mutation rate, μ=0.1 per gamete, as was estimated by the other method, explained in section 9.

It must be pointed out that the present method involves data and methods of calculation both of which are entirely separate, as well as different in character, from those used in the other mode of attack. Although extrapolation is employed at one point in the present attack—namely, for estimating the degree of dominance—this item did not enter at all into the earlier calculation. Moreover, there seems little doubt, in consideration of observations concerned with man himself (see e. g. Levit, 1935), that the dominance factor in man would at least be within the same order of magnitude as that here assumed on the basis of extrapolation. If this is true, then the estimate for mutation rate here arrived at is likewise of the right order of magnitude, at least as a minimum value. A further circumstance to be taken into consideration in evaluation of the present result is that it was not realized until the calculations were carried through that they would give a value even distantly in agreement with what had been obtained by the other method, and that no attempt was made to manipu-

late them to obtain a satisfactory fit to expectation. For these reasons, it would seem that the present result, although itself involving interpolation, lends material support, from an independent direction, to that arrived at previously.

Although the present mode of attack is concerned only with spontaneous mutations, the estimate of the total spontaneous rate, as well as of the total load, thereby arrived at, affords an important independent possibility for gauging the total mutational damage which would be produced in a human population by radiation. Before this could be accomplished, however, there would have to be some means of determining, for some limited genetic category capable of being used as an index, the relation between the spontaneous rate and the rate induced by a given dose of radiation. Possibly somatic or tissue-culture mutations, if there were good reason to infer them to be of the point type, would be useful to provide such an index. At any rate, if it were once furnished, it would then be relatively easy to combine this information with that on the total load, derived from the results of inbreeding, so as to obtain a realistic view of the all-around and long-term meaning of a given dose of radiation.

Of course we are far from the final or exact answers concerning the total frequency of either induced or spontaneous mutations, or concerning the persistence factor, for any lower organism, and much further yet from these answers for man. But the ways are opening up, and there seems good reason to believe that our present estimates for man, although involving extrapolation, may with assurance be regarded as minimal ones, and of the right order of magnitude. Before this point could be arrived at it was necessary to carry out a vast amount of work in the genetics of lower organisms, and also to collect very considerable data from man, and to consider these in connexion with one another. An increasing attack along both lines will be necessary if we are to attain the knowledge we need for the adequate protection and the fostering of our most precious trust, our genetic heritage.

REFERENCES

1. Abrahamson, S. & Telfer, J. D. (1956) The relative constancy of the X-ray induced mutation frequency of *Drosophila melanogaster* sperm in inseminated females. *Genetics*, *41*, 677–684.
2. Bonnier, G. & Lünning, K. G. (1949) Studies on X-ray mutations in the white and forked loci of *Drosophila melanogaster*. I. A statistical analysis of mutation frequencies. *Heriditas* (*Lund*), *35*, 163–189.
3. Bonnier, G. & Lünning, K. G. (1953) Sex-lined lethals in *Drosophila melanogaster* from different modes of X-ray irradiation. *Heriditas* (*Lund*), *39*, 193–200.
4. Caspari, E. & Stern, C. (1948) The influence of chronic irradiation with Gamma-rays at low dosages on the mutation rate in *Drosophila Melanogaster*. *Genetics*, *33*, 75–95.
5. Crow, J. F. (1956) The estimation of spontaneous and radiation-induced mutation rates in man. *Eugen, Quart. 3*, 201–208.
6. Dubovsky, N. V. (1935) On the question of the comparative mutability of stocks of *Drosophila Melanogaster* of different origins. *C. R. Acad. Sci. U. R. S. S. 4*, 95–97.
7. Falk, R. (1955) Studies on X-ray induced viability mutations in the third chromosome of *Drosophila Melanogaster*. *Heriditis* (*Lund*), *41*, 259–278.
8. Giles, N. H. Jr., (1952) Studies on the mechanism of reversion in biochemical mutants of *Neurospora crassa*. *Cold Spr. Harb. Symp. quant. Biol. 16*, 283–313.
9. Haldane, J. B. S. (1937) The effect of variation on fitness. *Amer. Nat. 71*, 337–349.
10. Hanson, F. B. & Heys, F. (1929) An analysis of the effects of the different rays of radium in producing lethal mutations in Drosophila. *Amer. Nat. 63*, 511–516.
11. Herskowitz, I. H. & Abrahamson, S. (1956) Dependence of X-ray-induced crossover-like exchanges in Drosophila oocytes and oogonia upon radiation intensity. *Genetics*, *41*, 646.
12. Herskowitz, I. H., Muller, H. J. & Laughlin, J. S. (1956) The higher efficiency of ordinary X-rays than 18 Mev electrons in inducing chromosome changes when applied to Drosophila spermatozoa. *Genetics*, *41*, 646–647.
13. Hertwig, P. (1941) Erbänderungen bei Mäusen nach Röntgenbestrahlung. *Proc. 7th int. gent. Congr.* (*J. Genet.* Suppl.), pp. 145–146.

14. Ives, P. T., Levine, R. P. & Yost, H. T. Jr. (1954) The production of mutations in *Drosophila melanogaster* by the fast neutron radiation of an atomic explosion. *Proc. nat. Acad. Sci. (Wash.)*, *40*, 165–171
15. Käfer, E. (1952) Vitalitätsmutationen, ausgelöst durch Röntgenstrahlen bei *Drosophila melanogaster. Z indukt. Abstamm.-u-VererbLehre*, *84*, 508–535
16. Kerkis, J. J. (1938) On the possibility of increasing the gene mutability by X-ray treatment of the organism at the early stage of development. *Izv. Akad. Nauk SSSR, Otd. mat.-est., Ser. biol.* 67–74.
17. Kimball, A. W. (1956) Approximate confidence intervals for specific locus mutation rates. *Amer. Nat. 90*, 369–376
18. Kossikov, K. V. (1935) X-ray mutations in *Drosophila simulans. Bull. Inst. Genetics Acad. Sci. USSR*, *10*, 189–197
19. Kossikov, K. V. (1936) The influence of the age and sex of the germ cells on the frequency of mutations in *Drosophila simulans. C. R. (Dokl.) Acad. Sci. URSS*, N. S. *2* (*11*), 115–118
20. Lefevre, G. (1950) X-ray-induced genetic changes in germinal and somatic tissue of *Drosophila melanogaster. Amer. Nat. 84*, 341–366
21. Levit, S. G. (1936) The problem of dominance in man. *J. Genet. 33*, 411–434
22. Lewis, D. (1951) Structure of the incompatibility gene. III. Types of spontaneous and induced mutation. *Heredity*, *5*, 399–414
23. Lüning, K. (1952) Studies on X-ray induced mutations in various stages of spermatogenesis in *Drosophila melanogaster*. Doctoral Dissertation, University of Stockholm. A. Bonnier's Press, Stockholm
24. Mickey, G. H. (1954) Visible and lethal mutations in Drosophila. *Amer. Nat. 88*, 241–255
25. Morton, N. E., Crow, J. F. & Muller, H. J. (1956) An estimate of the mutational damage in man from data on consanguineous marriages. *Proc. nat. Acad. Sci. (Wash.)*, *42*, 855–863
26. Muller, H. J. (1928) The measurement of gene mutation rate in Drosophila, its high variability, and its dependence upon temperature. *Genetics*, *13*, 279–357
27. Muller, H. J. (1930) Radiation and genetics. *Amer. Nat. 64*, 220–251
28. Muller, H. J. (1938) The remaking of chromosomes. *Collect. Net* (Woods Hole) *13*, 181, 183–195, 198
29. Muller, H. J. (1940) An analysis of the process of structural change in chromosomes of Drosophila. *J. Genet. 40*, 1–66
30. Muller, H. J. (1946) Age in relation to the frequency of spontaneous mutations in Drosophila. *Yearb. Amer. philos. Soc. 1945*, 150–153
31. Muller, H. J. (1950a) Our load of mutations. *Amer. J. hum. Genet. 2*, 111–176
32. Muller, H. J. (1950b) Radiation damage to the genetic material. II. Effects manifested mainly in the exposed individual. *Amer. Sci. 38*, 399–425
33. Muller, H. J. (1954a) The nature of the genetic effects produced by radiation. *Radiation Biology*, **1**, Chapter **7**, pp. 351–473. Ed. A. Hollaender, New York: McGraw-Hill
34. Muller, H. J. (1954b) The manner of production of mutations by radiation. Chapter 8, pp. 475–626. *Radiation Biology*, 1., Ed. A. Hollaender, New York
35. Muller, H. J. (1954c) Characteristics of the far stronger but "spottier" mutagenicity of fast neutrons as compared with X-rays in Drosophila spermatozoa. *Genetics*, *39*, 985
36. Muller, H. J. (1955) Comments on the genetic effects of radiation on human populations. *J. Hered. 46*, 199–200
37. Muller, H. J. (1956a) How radiation changes the genetic constitution. Proceedings of the International Conference on the Peaceful Uses of Atomic Energy, Geneva, 1955, *11*, 387–399
38. Muller, H. J. (1956b) After-effects of nuclear radiation. *J. Amer. Soc. Safety Engineers*, *1*, 42–48
39. Muller, H. J. (1957) Further studies bearing on the load of mutations in man. Proceedings of the First International Congress on Human Genetics, Copenhagen, 1956, *Acta Genet. et Statistica Medica, Suppl.* (in press)
40. Muller, H. J., Herskowitz, I. H., Abrahamson, S. & Oster, I. I. (1954) A non-linear relation between X-ray dose and recovered lethal mutations in Drosophila. *Genetics*, *39*, 741–749
41. Muller, H. J. & Settles, F. (1927) The non-functioning of the genes in spermatozoa. *Anat. Rec. 31*, 347

42. Muller, H. J., Valencia, J. I. & Valencia, R. M. (1950) The frequency of spontaneous mutations at individual loci in Drosophila. *Genetics, 35,* 125–126

43. Muller, H. J., Valencia, R. M. & Valencia, J. I. (1950) The production of mutations at individual loci in oöcytes and oögonia. *Genetics. 35,* 126

44. Oliver, C. P. (1930) The effect of varying the duration of X-ray treatment upon the frequency of mutation, *Science, 71,* 44–46

45. Oster, I. I. (1954) The effect of mitotic poisons on the sensitivity of Drosophila germ cells to ionizing radiation, *Excerpta med. (Amst.), Sect. XIV, 8,* 406

46. Oster, I. I. (1955) Modification of X-ray mutagenesis in Drosophila. I. Reunion of chromosomes irradiated during spermiogenesis, *Genetics, 40,* 692–696

47. Oster, I. I. (1956?) Modification of X-ray mutagenesis in Drosophila. II. Relative sensitivity of spermatids and mature spermatozoa, *Proceedings of the Fifth International Symposium on Radiobiology,* Stockholm, 1956 (in press)

48. Patterson, J. T. (1928) The effects of X-rays in producing mutations in the somatic cells of *Drosophila melanogaster, Science, 68,* 41–43

49. Patterson, J. T. & Muller, H. J. (1930) Are "progressive" mutations produced by X-rays? *Genetics, 15,* 495–578

50. Puck, T. T. & Marcus, P. I. (1956) Action of X-rays on mammalian cells. *J. exp. Med. 103,* 653–666

51. Puck, T. T., Marcus, P. I. & Cieciura, S. J. (1956) Clonal growth of mammalian cells in vitro, *J. exp. Med. 103,* 273–284

52. Quastler, H. (1956) Modes of acute radiation death. *Proceedings of the International Conference on the Peaceful Uses of Atomic Energy,* Geneva, 1955, *11,* 121–124

53. Russell, W. L. (1951) X-ray-induced mutations in mice, *Cold Spr. Harb. Symp. quant, Biol. 16,* 327–336

54. Russell, W. L. (1956) Genetic effects of radiation in mice and their bearing on the estimation of human hazards, *Proceedings of the International Conference on the Peaceful Uses of Atomic Energy,* Geneva, 1955, *11,* 382–383

55. Sacher, G. A. (1956) On the statistical nature of mortality, with especial reference to chronic radiation mortality. *Radiology,* 67, 250–257

56. Serebrovskaya, R. I. & Shapiro, N. I. (1935) The frequency of mutations induced by X-rays in the autosomes of mature and immature germ-cells of *Drosophila melanogaster* males, *C. R. (Dokl.) Acad. Sci. URSS,* N. S. *2 (11),* 421–428

57. Shapiro, N. I. (1936) Is there germ cell selection in *Drosophila melanogaster? C. R. (Dokl.) Acad. Sci. URSS,* N. S. *2(11),* 119–122

58. Snell, G. D. (1935) The induction by X-rays of hereditary changes in mice, *Genetics, 20,* 545–567

59. Stadler, L. J. (1928) The rate of induced mutation in relation to dormancy, temperature, and dosage, *Anat. Rec. 41, 97*

60. Stapleton, G. E., Hollaender, A. & Martin, F. L. (1952) Mechanism of lethal and mutagenic action of ionizing radiation on *Aspergillus terreus.* I. Relationship of relative biological efficiency to ion density. *J. cell. Comp. Physiol. 39* (Suppl. 1), 87–100

61. Stern, C., Carson, G., Kinst, M., Novitski, E. & Uphoff, D. (1952) The viability of heterozygotes for lethals, *Genetics, 37,* 413–449

62. Telfer, J. D. & Abrahamson, S. (1954) The production of sex-chromosome loss and dominant lethals in Drosophila sperm by fast neutrons. *Dros. Info. Serv. 28,* 161–163

63. Timoféeff-Ressovsky, N. W. (1929) The effect of X-rays in producing somatic genovariations of a definite locus in different directions in *Drosophila melanogaster, Amer. Nat. 63,* 118–124

64. Timoféeff-Ressovsky, N. W. (1934) Einige Versuche an *Drosophila melanogaster* über die Beziehungen zwischen Dosis und Art der Röntgenbestrahlung under der dadurch ausgelösten Mutationsrate, *Strahlentherapie, 49,* 463–478

65. Timoféeff-Ressovsky, N. W. (1937) Experimentelle Mutationsforschung in der Vererbungslehre. Beeinflussung der Erbanlagen durch Strahlung und andere Faktoren, *Wissensch. Forschungsber. Naturwiss. Reihe, 42,* x + 184 pp. Dresden and Leipzig: Theodor Steinkopff

66. Uphoff, D. E. & Stern, C. (1949) The genetic effects of low intensity radiation, *Science, 109,* 609–610

ANNEX 2

THE TYPES OF MUTATION PRODUCED AT KNOWN GENE LOCI AND THE POSSIBILITY OF HITHERTO UNRECOGNIZED MUTATIONS BEING INDUCED ANIMAL POPULATION IRRADIATION RESULTS AND WORK NEEDED [1]

It is commonly acceptable as a working hypothesis that ionizing radiations do not induce new types of mutation, but only raise the mutation rates of existing alleles. The basis of this assumption is partly theoretical and partly experimental. The theoretical argument rests on the fact that all living matter is continuously exposed to natural background radiation, and always has been so exposed; therefore, it is argued, any mutation which could be induced by ionizing radiation must already have been induced by natural background radiation at some time in the past; therefore no new type of mutation could be induced by man-made radiation. The experimental data, which are now extensive, do not disprove this; but in so far as it is essentially a negative hypothesis, and fails to specify the extent of either spontaneous or induced mutation, it is by its very nature not amenable to experimental test. Thus if in some experiment radiation exposure induces mutations of a type previously unknown, this can always be explained as nothing more than a manifestation of the limited nature of prior knowledge of spontaneous mutation; conversely, if exposure fails to induce mutations of a type previously known, that can always be explained as a manifestation of the finite nature of the experimental set-up. The hypothesis that ionizing radiations do not induce new types of mutation is therefore, like so many others in biology, unprovable and undisprovable. As such, it can only be of heuristic value; the extent of its value depends on our assessment of the extent to which it may be true, and the extent to which we are willing to use it as a guide in planning future action.

In point of fact, geneticists are willing to place so much faith in its validity that this hypothesis forms the basis of all present day estimates of the genetic hazard of ionizing radiation to man. It is therefore worth while to ask if circumstances can be visualized in which it might break down. The supposition underlying it is that man-made radiations do not differ in any essential respect from natural background radiations. In respect of dose-rate they extend far beyond the natural range, but we have no clear evidence of dose-rate thresholds for the induction of genetic effects. So far as present knowledge goes, it seems that linear energy transfer is the biologically most important characteristic of a radiation; and in this respect natural background radiation covers the whole known range, from the sparse ionization of naturally occurring gamma rays to the dense ionization produced by alpha particles and heavy cosmic nuclei. Thus there does not at present appear to be any obvious theoretical reason for expecting man-made radiations to induce alleles that were previously unknown. On the other hand, there is no theoretical basis for the converse supposition, namely that ionizing radiation can induce all known alleles; in fact, there is a certain amount of experimental evidence that it tends to induce especially the more extreme alleles at a locus.

It is worth noting that though this hypothesis has a theoretical basis which is probably valid for mutagenesis by ionizing radiations, the analogous hypothesis for chemical mutagenesis has none. There is no ground for postulating the natural occurrence in biological material of all chemical mutagens which might be synthesized in the laboratory. Furthermore, some chemical mutagens might be expected to have a relatively mild action, and induce subtle genetic changes, compared with the generally destructive action of ionizing radiation. Recent experimental work in this field, notably that of Fahmy & Fahmy (1956), supports an interpretation of this type. Furthermore, if (as seems probable) ionizing radiation is responsible for only about one-tenth of human spontaneous mutation, leaving nine-tenths to be accounted for, we should be unwise to ignore the possibility that chemical substances may be much more important than ionizing radiation as a cause of human mutation.

Thus far I have considered the gene only as the unit of mutation, its constancy between mutational events being implicit. But a gene is also a unit of action, its presence being recognizable only by its effect on the phenotype of an individual. Furthermore, the final effect of a gene, unlike the gene itself, may be extremely variable, depending upon the other allele at the same locus, the alleles at other loci and the mass of non-genetic factors, grouped together under the term

[1] **Submitted by Dr. T. C. Carter, Geneticist, Radiobiological Research Unit (Medical Research Council of Great Britain) Atomic Energy Research Establishment, Harwell, Berkshire, England.**

"environment". In no two individuals are the total genotype and the total environment identical, and therefore in no two individuals can the same allele be expected *a priori* to produce identical end effects. Variability of gene expression may be very great where the end effect is putatively remote from the primary gene product, with many morphological mutants; conversely it may be relatively slight where the effect observed is believed to be close to the primary gene product, as with the blood group antigens. In so far as the practices of civilization have wrought great changes in the macro and microenvironment of man, we must suppose that they have changed and are changing the expression of many human alleles.

Many systems can be invented for the classification of human genes, and which particular system is used will depend on the interests of the user. The population geneticist is interested primarily in the biological value of a genotype. He will therefore classify alleles according to their average effect on the fitness of their carriers, that is to say on the number of zygotes that will be contributed to the next generation by a zygote of the present generation. It will be a twofold classification, according as the allele is in the homozygous or heterozygous state. Mutant alleles are probably almost always disadvantageous to some extent when homozygous, but their action in heterozygotes may vary from severe detriment through neutrality to advantage. This fact divides them into two broad classes: those which are unconditionally disadvantageous, and those which are disadvantageous in some individuals but advantageous in others. The distinction is fundamental, for it determines the nature of the forces which will maintain the allele in the population and the frequency at which it will be maintained. An unconditionally detrimental allele will be maintained at a low frequency under the opposed action of mutation to the allele and natural selection against it. On the other hand, an allele which is advantageous in some individuals and disadvantageous in others will be maintained at a high frequency, depending on the degree of advantage or disadvantage in the various individuals. Mutation will play only a minor role, or even none at all, in determining the structure of the population in respect to alleles of this type. It is therefore of importance to any assessment of the genetical hazards of radiations to man to know whether alleles of this type are of common occurrence. Unfortunately it is a problem of exceptional inherent difficulty, because we may expect that the more easily recognized genes will largely be among those with notably detrimental effects; and, conversely, that the conditionally advantageous genes will be mainly among those with minor effects and that they may, for just this reason, be difficult to recognize.

This brings me to a point where I find it necessary to voice some misgivings which I have felt for a long time about one aspect of what might be called "genetical public relations". Soon after H. J. Muller's demonstration that X-rays have a mutagenic action it was realized that they present a genetic hazard to man. At that time genes were thought of as consisting mainly, if not entirely, of common, advantageous, wild-type alleles and rare, deleterious, mutant alleles. They were unconditionally good or bad. Mutation was viewed as a necessary evil; it was something which happened, without which the species would lack the heritable variation on which future evolution depends, but it introduced into the population a load of mutant alleles which had to be eliminated by processes of natural selection. Each mutational event implied the occurrence of another mutant allele to be eliminated sooner or later through the "genetic" death of some individual if equilibrium were to be maintained.

I do not think anyone seriously doubts that this is a reasonably accurate representation of the state of affairs in respect of grossly deleterious autosomal dominant or sex-lined genes such as retinoblastoma or haemophilia. On the other hand, I think may geneticists would now doubt whether this concept is valid for more than a relatively small proportion of all human genes. Clear-cut unconditionally deleterious oligogenes may be relative rarities. They may represent only one tail of a distribution; numerically they may come far behind the polygenes, each with an effect so small as to be virtually undetectable by the methods of classical genetics, yet together of major importance because they regulate the quantitatively variable characteristics of each species through which evolution must largely operate. Now the outstanding feature of almost any quantitative character is that it has a central optimum; the extremes in either direction appear to be at a disadvantage, in respect of biological fitness, compared with some intermediate phenotype. The theoretical interpretation is that heterozygotes for genes affecting a quantitative character have a greater

biological fitness than the corresponding homozygotes; and this implies that the mutation rate may be relatively unimportant in determining the gene frequency.

The above argument has been based mainly on theoretical considerations; but there is now a great mass of observational and experimental evidence that heterozygosity is the rule rather than the exception in wild populations. If anyone doubts it, he should re-read the writings of Dobzhansky and his co-workers on wild *Drosophila* populations, of Bruce Wallace on irradiated *Drosophila* populations and of Dunn on mouse populations; or he should try inbreeding any species that is normally crossbreeding.

In the face of all this I find it disconcerting that geneticists, when writing for the public, still often base their argument on an assertion that all mutation (or very nearly all) is harmful. I say it is disconcerting in the full knowledge that I use exactly the same argument when, as happens all too often nowadays, I have to give a talk on radiation hazards to an intelligent but gentically uninstructed audience. Perhaps its attraction is that it is a relatively easy argument to put over; or perhaps we use it because one can draw quantitative inferences about the genetic load due to some unconditionally deleterious human alleles, whereas at present it is almost impossible to speak quantitatively about human polygenic characters. But, whatever the reason, it is extremely important that we geneticists should not bind ourselves to the fact that unconditionally deleterious oligogenes may constitute only a small fraction of the human genome. Furthermore, I am not entirely happy that any science can be really healthy when it has one story for home consumption and another for the rest of mankind.

The necessity for using an argument such as this stems essentially from one fact we know something about mutation in man and experimental animals, but we know very little about the effect on a population in which mutation is induced. We know enough to be reasonably certain that the current theory of mendelian populations is over-simplified and unable to accommodate some essential features of real populations; but we have not yet got a satisfactory theory to put in its place. For the present there can be only one corollary; we must have more research on the genetic structure of populations ,in the hope that the nature of the facts will become clearer and will stimulate the development of a more complete theory. This theory would have to cover the origin and loss of variation in populatons its origin by spontaneous or artificially enhanced mutation and by environmental action, and its loss by natural or artificial selection.

There have been many genetic studies of wild populations. Though in most of them the object was to study the effects of natural selection, it is only rarely that direct evidence has been obtained that the effect observed really was due to this cause. Thus the spread of melanic forms of various species of moths in industrial areas has been observed for over a century; and it has been assumed throughout that the spread was due to a selective advantage of the melanic form, following an environmental change from the relatively clean agricultural to the sooty industrial economy; but it was only last year that Kettlewell (1955) was able to confirm the validity of this assumption, by direct observation of the numbers of moths of the various phenotypes taken by bird predators. It has also been a characteristic of studies of wild populations that, with few exceptions, the material studied has been polymorphic. This must have been due largely to subjective selection by the investigator, since a polymorphic population holds an obvious interest which a monomorphic population lacks. Nevertheless, where an apparently monomorphic population has been sufficiently closely observed, it has often proved to be polymorphic, even though the polymorphism may have been cryptic. Obvious examples are the populations of various *Drosophila* species studied by Dobzhansky and his school, (vide Wallace, 1954), and the mouse populations studied by L. C. Dunn (1953). Dunn's work is of especial interest, because it shows that mechanisms whereby coadapted blocks of genes could come into existence are not peculiar to *Drosophila*. The mechanism in the mouse differs from that in *Drosophila*, but the effects are the same: suppression of crossing over and selective advantage of the heterozygous genotype in which it has been suppressed, even at the cost of a high proportion of inviable homozygotes. His findings gain significance in the light of the recent demonstration by my colleague Dr. Mary Lyon, using an induced translocation, that the region of suppressed crossing over is at least five times as long as the short segment marked in Dunn's experiments.

The study of mutation and artificial selection in the laboratory and of natural selection in wild populations are three approaches to a much more difficult study, namely that of populations with mutation rates that have been enhanced by ioniz-

ing radiations or other mutagens. Nor must it be forgotten that ionizing radiations have other genetic effects besides mutagenesis; they increase crossing over, a fact which was known before their mutagenic action was discovered, and which may be of great importance in the study of polygenic systems.

Thus far few have attempted to work with irradiated animal populations. History dictated that one of the first studies in this field should be of a human population; but the genetical work of the Atomic Bomb Casualty Commission was almost foredoomed to failure, in the sense that it was very unlikely that statistically significant observations could have been made, even on the basis of the most extreme assumption, namely that all human "spontaneous" mutation is really induced by background radiation and that the doubling dose for men is consequently as low as 3 or 4r. In the event the results were, with one possible exception, negative; but all who are concerned with planning human radiation genetic studies in the future will owe a debt to Neel and his colleagues for doing the pioneer work in this field and exposing some of the problems (Neel *et al*,. 1953). The only other genetic studies of irradiated human populations of which I am aware are those of Crow (1955) and of Macht & Lawrence (1955); in each case the irradiated population consisted of radiologists. Here also the results were, in the main, negative; and the work suffered from the further limitation that it was impossible to estimate, even roughly, the radiation dose received.

There remain the experimental studies of irradiated animal populations. Of these there have been exceptionally few; and in almost all the experimental material has been *Drosophila melanogaster.* There are two reasons for this: first, a population to be maintained under known irradiation conditions must almost of necessity be kept in the laboratory; second, to guard against the possible effects of genetic drift, the effective breeding population should be at least of several hundred individuals. These requirements of laboratory culture and population size can be reconciled only by limiting the size of the individual animal. Subject to this limitation, *Drosophila melanogaster* is the obvious choice, being exceptionally well known genetically. We hope to develop techniques at Harwell for maintaining mouse populations in the laboratory, but I am doubtful whether it would be feasible to keep free-living populations of larger animals in an irradiated space. A possible solution might be to find an isolated wild colony and irradiate its habitat; this procedure would have the inherent defect, however, that one cannot be sure of obtaining a truly comparable control population; and in this work controls are a *sine qua non.*

If anything were needed to show how wide is the gap between observational fact and existing population genetic theory, the few published studies of irradiated *Drosophila* populations would do it. The various writers have given up any attempt to interpret their observations in terms of gene frequencies, contending themselves with observing what happens in their populations and attempting to interpret their observations in terms appropriate to the polygenic systems studied. Various combinations of selection type and mutational status have been used, and various types of foundation population. Bruce Wallace (1952) has observed the effects of natural selection on biological fiitness in populations originally derived from an inbred strain but now heterogenous, which were exposed to various levels of acute and chronic irradiation. Buzzati-Traverso (1954) likewise observed the effects of natural selection in irradiated populations; but here the foundation populations were inbred and the effects observed were egg-production and the incidence of the *non-spineless* phenotype due to modification of the genetic milieu in a homozygous *spineless* population. Clayton and Alan Robertson (1955) likewise used inbred foundation populations; they observed the variance of the number of abdominal bristles and the response to artificial selection for this character. Scossiroli (1954) selected for sternopleural hairs in an irradiated population which was genetically heterogenous but which had previously been selected by Mather without irradiation and had reached a plateau.

It is too early to attempt to draw general conclusions from these experiments, but some things are clear. Buzzati-Traverso's work shows that irradiation of an inbred population can release genetic variability in a character such as egg-production, which is one of the components of biological fitness, and can thereby enable natural selection to increase fitness. Scossiroli's work shows that irradiation can release genetic variability in a heterogeneous population which has reached a selection limit, and can thereby enable the limit to be surpassed. Bruce Wallace's work shows that populations can live success-

fully under conditions of irradiation in which a large proportion of their chromosomes carry gene combinations which are lethal when homozygous, but that some of these combinations may be advantageous when heterozygous. The work of Clayton and Robertson shows that the amount of genetic variability arising spontaneously through new mutation in each generation is only a minute fraction, perhaps a thousandth, of that normally present in a *Drosophila* population; and that a part only of the additional genetic variability released by irradiation may be available for selection.

Just what the full implications are for human genetics it is impossible at present to assess; but two conclusions seem inescapable. First, it is essential to extend work of this type and to cover other species, including mammals, with a much lower reproductive potential than *Drosophila;* the results might be very different in species where the female produced only ten young instead of hundreds and selection differentials were consequently lower. Second, we have no mandate from experimental fact to extend to the whole human genome the theoretical treatment of the genetic hazard of radiations that we now apply with a fair measure of confidence to grossly deleterious gene mutations. It follows that for the present we must limit quantitative assessment to this part of the hazard alone; and this implies that the first task of human genetics must be to identify as completely as possible that part of the social load which is due to genes in this class.

REFERENCES

Buzzati-Traverso, A. (1954). On the role of mutation rate in evolution: *Caryologia, Suppl. 1,* 450–462

Clayton, G. & Robertson, A. (1955). Mutation and quantitative variation. *Amer. Nat. 89,* 151–158

Crow, J. F. (1955). A comparison of fetal and infant death rates in the progeny of radiologists and pathologists. *Amer. J. Roentgenol. 73,* 467–471

Dunn, L. C. (1953). Variations in the segregation ratio as causes of variations in gene frequency. *Acta genet. 4,* 139–151

Fahmy, O. G. & Fahmy, M. J. (1956). Cytogenetic analysis of the action of carcinogens and tumor inhibitors in *Drosophila melanogaster.* V. Differential genetic response to the alkylating mutagens and X-radiation. *J. Genet. 54,* 146–164

Kettlewell, H. B. D. (1955). Selection experiments on industrial melanism in the Lepidoptera. *Heredity, 9,* 323–342

Macht, S. H. & Lawrence, P. S. (1955). National survey of congenital malformations resulting from exposure to roentgen radiation. *Amer J. Roentgenol. 73,* 442–466

Neel, J. V., Schull, W. J., McDonald, D. J., Morton, N. E., Kodani, M., Takeshima, K., Anderson, R. C., Wood, J., Brewer, R., Wright, S., Yaizajaki, J., Suzuki, M. & Kitamura, S. (1953). The effect of exposure to the atomic bombs on pregnancy termination in Hiroshima and Nagasaki: preliminary report. *Science, 118,* 537–541

Scossiroli, R. E. (1954). Effectiveness of artificial selection under irradiation of plateaued populations of *Drosophila melanogaster. I. U. B. S. Publications, Series B, 15,* 42–66

Wallace, B. (1952). The estimation of adaptive values of experimental populations. *Evolution, 6,* 333–341

Wallace, B. (1954). Coadaptation and the gene arrangement of *Drosophila pseudoobscura. I. U. B. S. Publications, Series B, 15,* 67–94.

ANNEX 3

A DISCUSSION OF SOME OF THE PROBLEMS ACCOMPANYING AN INCREASE OF MUTATION RATES IN MENDELIAN POPULATIONS[1]

Problems arising from the exposure of man to irradiation are extremely numerous. They bear on many aspects of his health and his children's health. To the extent that the original exposure—medical or industrial—aims at improving man's welfare, he benefits, to the extent, however, that the exposure does him bodily harm or induces gene mutations that will harm his offspring, he suffers.

[1] Submitted by Dr. Bruce Wallace, Biological Laboratory, Long Island Biological Association, Cold Spring Harbor, Long Island, N. Y., USA.

The mutagenic effects of radiation pose problems of immediate concern to the geneticist. These problems are of three major types: the development of a theory of population genetics adequate for the formulation of predictions, the design of experiments capable of testing the theory and of supplying empirical values for various parameters, and the extrapolation of theory and experimental results to human populations.

The postulated role of mutations in Mendelian populations depends largely upon the basic concept one entertains regarding the genetic structure of populations. In the main, there exist two contrasting but not mutually exclusive concepts: the first is based upon the superiority of homozygous individuals; the second, upon the superiority of heterozygotes.

The first concept postulates that individuals of the highest possible fitness can be completely homozygous. Natural selection acting within a constant environment would favour these individuals and would tend toward the establishment of a population composed entirely of homozygous individuals. In such a population the individuals of each generation should, ideally, be identical and the individuals of one generation should be identical to those of the next. Mutations in a population such as this operate to frustrate the aims of natural selection. By definition, the new mutations are deleterious and, consequently, their constant formation prevents the population from reaching the level of fitness theoretically possible. Furthermore, under equilibrium conditions the deleterious effect of mutations on the population is a function of mutation rates and is independent of the harm done to any one individual by any one mutation. Theoretical treatments of this problem have been given by Haldane (1937), Crow (1952), and, in great detail by Muller (1950). Although no one actually believes that environmental conditions are constant or that the ideal population described above actually exists, the model is reasonable nevertheless if one assumes that near-equilibrium conditions exist at any moment and that genetic changes within populations occur slowly (see, for instance, Haldane, 1954).

The second concept assumes that even under constant environmental conditions the individual with the highest fitness is genetically heterozygous rather than homozygous. Furthermore, there need not be one ideal genotype but many. An ideal population of this sort would consist of individuals as phenotypically uniform as possible consistent with the demands of natural selection but these individuals would be genetically diverse. Similarly, individuals of one generation would not be genetically identical, even under ideal conditions, with those of the next. The selective coefficient of any gene in this type of population would be a function of the genetic situation prevailing within that population. Since the population consists of individuals of diverse genotypes, selection would be constantly shuffling gene frequencies and selective values simply because of the uncertainties associated with the formation of chance gene combinations. The details of this model have not been developed in a way comparable with the first; one can say, however, that gene frequencies under this model are primarily a function of selection and only secondarily a function of mutation rates.

These two concepts are not mutually exclusive. It may develop that one or the other is substantially correct. It may be that for some loci one is correct while for others the second applies. It is quite probable that different species differ in their genetic structure. Finally, at different times and in different places the genetic structure of a population may shift from one model to the other.

A few remarks may be made regarding the logic underlying these two concepts of population structure. Genes and chromosomes are the means by which information is passed from one generation to the next; in some cases they are the only means, in others this hereditary information is supplemented by the "spoken" word which allows individuals of one generation to communicate with those of the next. The first concept, that based on the superiority of homozygous individuals, stresses the accuracy of the transmitted information. In the absence of mutations and of environmental change, every individual of a generation would be supplied with precisely that information which has proven valuable in the past. There is no wastage through the formation of ill-adapted individuals. Furthermore, it is a moral system in that, under ideal conditions, every individual is his neighbour's equal. The second concept entails wastage; certain individuals must obtain hereditary information that is not perfectly accurate. In so far as this wastage can be equated with suffering (and it certainly can be considered in this way for human beings), the second concept is morally deficient.

What arguments, then, can be mustered to support the second concept, and to justify giving it serious consideration? First, to the extent that genes are semi-dominant, their frequencies are changed much more rapidly by the action of selection on heterozygous individuals than by that on rare homozygotes. Second, a gene that is beneficial through some semi-dominant effect need not be beneficial when hemozygous; the nature of these homozygous individuals is unimportant to the population at the time selection favours the heterozygotes. Third, the replacement of superior aa′ individuals by equally good a″a″ individuals requires that the allele a″ also be advantageous when heterozygous. Fourth, there are physiological reasons for doubting in some instances whether a single allele in homozygous individuals can actually duplicate the action of two contrasting alleles in heterozygotes.

One difficulty confronting the second concept is more apparent than real. It arises from the geneticist's inability to distinguish which of two alleles is a favourable dominant and which is a deleterious recessive (see Crow, 1952, footnote p. 285). A geneticist can detect gene effects by substitution only. Genetic changes within a population are determined by the sequence in which mutations occur. By completely ignoring the sequence of genetic change and by regarding the favourable dominant as "normal", one is forced to the absurd conclusion that the origin of each favourable dominant (or semi-dominant) lowers the fitness of the population and that the population regains its normal fitness only if the new dominant attains fixation in the population.

Finally, in reference to the first concept, I have mental reservations that stem from the assumed independence of the effect of the gene mutation on the population and the effect of the gene on individuals of the population. In other words, no matter how slight the deviation from the "normal" allele, the effect of a given class of mutant alleles is said to be proportional to mutation rate alone. In fact Muller (1948) mentions the possibility that small harmful mutations may be even worse for the population than fully lethal ones. I do not question the calculations that demonstrate this fact; I question the assumptions upon which the calculations are based and which result in a curve with such an abrupt break regardless of how infinitesimal the effect of the mutation might be.

The problems mentioned so far lie in the realm of theoretical speculations. They are problems one meets when attempting to visualize techniques employed by Mendelian populations in meeting the demands of existence, techniques compatible with the known facts of genetics. The second large class of problems arises in connection with the design of experiments aimed at testing the validity of theoretical models. Regardless of one's concept of the genetic structure of a population, obtaining experimental data to verify the concept or to furnish evidence regarding certain parameters is an overwhelming chore.

Information required for the manipulation of equations under the model that stresses homozygosity includes estimations of numbers of loci, total mutation rates, distributions of mutations in terms of their effects on various components of fitness (viability and fertility in particular), the distribution of deleterious mutations among individuals of a population, and dominance-recessive relationships. Information along these lines is being gathered, among other laboratories, at Oak Ridge and at the University of Indiana under the direction of Dr. Russell and of Professor Muller, respectively. I believe we all recognize the tremendous effort required to obtain this information.

In our laboratory we have taken what appears superficially to be a somewhat simpler approach: the simultaneous analysis of the genetic content of experimental populations of *D. melanogaster* in terms of genes affecting fitness and measures of fitness itself. The latter measure will be required for the final verification of one's concept of population structure regardless of which of the two one entertains. The chief difficulties in this approach lie in the estimation of fitness and in determining the amount of selection required to maintain this fitness. These difficulties are compounded by the necessity to limit one's studies to components of fitness and to carry out the analyses outside the population, outside even an experimental population. In studies of components of fitness one generally assumes that these components are to some degree correlated with one another and with their sum. Robertson (1955) has pointed out, though, that in a population at genetic equilibrium the components of fitness must be negatively correlated. This indicates that a technique for measuring total fitness must eventually be found if the role of mutations in populations is to be evaluated experimentally.

Difficulties associated with the determination of selection pressures within populations do not seem insurmountable at the moment. Specific genetic changes within populations offer one source of information—for instance, the increase in frequency of one particular mutation, the establishment of equilibrium frequencies, or the loss of mutations following the cessation of irradiation. Estimations of population size shed light on the extent of inter-progeny selection; that is, one can judge whether a population exists because a few parents leave many offspring or because many parents leave a few each. Furthermore, larval mortality rates can be altered substantially without changing the adult population size to any appreciable extent; manipulations of this sort will offer an approach to the study of intra-progeny selection.

The final group of problems deals with the extrapolation of theory and experimental findings to man. The first problem that comes to mind is the shift in emphasis demanded by the importance of man's intellect. In experimental material "fitness" is equated with the ability to live and to reproduce; the emphasis in eugenic studies on the differential fertility existing in relation to I. Q. and racial origins shows that the experimental concept of fitness is not completely acceptable for human populations. Furthermore, although a long life and a full life is highly desirable, length *per se* is not all-important. Although the pertinent facts lie outside the realm of genetics, I suspect that the change from a 60-hour to a 40-hour work week has added more pleasurable, livable years to the average working man's life than he has lost by way of industrial and automobile accidents. These and similar problems are concerned with values; although there may be a consensus of opinion regarding these matters, there are bound to be sharp disagreements between societies and persons and even sharp changes in the views held by the same individual at different times.

Additional problems arise, too, because man is a social animal. The two concepts of populations described earlier dealt with ideal individuals with the highest possible fitness; these concepts are applicable to populations in which, with the exception of mating, there is no interaction between individuals in determining the fitness of the population. Such concepts are inadequate for dealing with populations of social organisms in which the fitness of the population is a function not only of the fitness of the individual members but also of the interaction between individuals. It would seem that before one can approach the problem of the ideal genetic architecture of populations of social organisms, including man, one would have to solve the simpler problem of the ideal constellation of phenotypes. I do not recall having seen such an analysis for human populations.

The next problems to be discussed concern what may be described as experimental human ecology. The central problem concerns the extent by which the visible human population, or, better, the reproducing human population, differs from the initial population of fertilized eggs from which it came. How strong are the selective forces operating within human populations? Mortality figures are available for the post-natal and late pre-natal periods. Figures are undoubtedly available, too, for the proportion of individuals who remain childless throughout life. Good data concerning the mortality of individuals in the early post-fertilization periods are not available at the moment. Lacking, too, are indications of the extent to which this mortality and sterility (effective, if not actual, sterility) are selective; random elimination, of course, is ineffective in bringing about genetic changes within populations. Haldane (1954) has developed a method for estimating the intensity of selection that utilizes phenotypic measurements only; this method may prove valuable in the analysis of human populations. Other data that would shed light on the selective potentialities of human populations are those dealing with the rapidity with which resistance to certain diseases has spread within memory of man and the effectiveness of this newly acquired resistance; this information would need to include the price in terms of mortality that the affected populations paid while selection operated. Along these same lines, it would be of particular interest to determine the factors responsible for limiting the number of children per couple in many human societies. When the average number of offspring per pair falls irrevocably below two for any species, that species can no longer replace itself numerically from one generation to the next and extinction is inevitable. In some human communities the present average is but slightly above two. Since this average is determined by a combination of sociological and biological factors, some effort should be expended to determine

the actual biological limit for the number of offspring human couples can have.

If it should develop that selection is more effective in man than we have suspected, we must nevertheless be wary of those who claim that radiation will do no harm to the human species. The rate at which mutant genes enter the gene pool of a population must equal the rate at which they leave. Mutant genes leave the gene pool by the effective elimination of individuals either through death, sterility, failure to reproduce, or a tendency to reproduce at a reduced rate. Effective elimination of individuals means, for human beings, that one individual is placed at a disadvantage relative to another; in many instances the "elimination" is accompanied by mental or physical suffering. Therefore, regardless of the ability or inability of "natural" selection within human populations to forestall extinction or to maintain the "fitness" of the population as a whole, we are still forced to the conclusion that every exposure of individuals to irradiation must be justifiable in terms of the beneficial effects that exposure confers either to the exposed individual or to the population as a whole. In the light of known effects of radiation it is impossible to defend unnecessary or unnecessarily high exposures.

REFERENCES

Crow, James F. (1952) Dominance and overdominance. *Heterosis*, Ed. J. S. Gowen, Iowa State College Press, Ames, Iowa

Haldane, J. B. S. (1937) The effect of variation on fitness. *Amer. Nat. 71*, 337–349

Haldane, J. B. S. (1954) The measurement of natural selection. *Proc. IX Int. Cong. Genet., Caryologia, Suppl. 6*, 480–487

Haldane, J. B. S. (1954) The statics of evolution. *Evolution as a process*. Eds. J. Huxley, A. C. Hardy, E. B. Ford. George Allen & Unwin, London, England

Muller, H. J. (1948) Mutational prophylaxis. *Bull. N. Y. Acad. Med. 24*, 447–469

Muller, H. J. (1950) Our load of mutations. *Amer. J. hum. Genet. 2*, 111–176

Robertson, Alan (1955) Selection in animals (Synthesis). *Cold Spring Harbor Symp. Quant. Biol. 20* (in press)

ANNEX 4

THE EXPOSURE OF MAN TO IONIZING RADIATIONS, WITH SPECIAL REFERENCE TO POSSIBLE GENETIC EFFECTS [1]

The purpose of this review is to show which generally occurring sources of ionizing radiation may at present be relevant and which may be irrelevant in discussing the effects of ionizing radiations on man.

We have to consider the direct effects on human tissues, as well as the indirect effects due to mutations of somatic cells, causing harmful effects to the individual himself, or of germ cells. The latter may either involve risks for the offspring already in the next generation, consequently being of interest to the individual himself, or may—with irradiation of a large number of inhabitants—constitute a long-term problem in the entire population.

The present sources of ionizing radiations which are of interest in these connexions include the following:

Natural sources of radiation

1. Sources of cosmic radiation.
2. The natural radioactive elements, particularly radium, thorium and potassium in the earth crust.
3. The natural content of radioactive elements in man.

Man-made sources of radiation

4. Radioactive material and technical arrangements producing radiation (X-ray tubes, other particle accelerators and nuclear reactors) used under such circumstances that the user generally is aware of the presence of the radiation (e. g. in education, science, medicine, and industry).
5. Sources of radiation used for purposes in which, as a rule, only the specialist is aware of the presence of ionizing radiation (e. g. radioactive luminous compounds on watches and other articles for common use, television sets, etc.).
6. Radioactive elements artificially distributed in nature.

[1] **Submitted by Professor R. M. Sievert, Institute of Radiophysics, Karolinska Hospital, Stockholm, Sweden.**

THE MAXIMUM PERMISSIBLE LEVELS OF IONIZING RADIATION FOR INDIVIDUALS AND LARGE POPULATIONS

Before treating the different sources of radiation which contribute to a larger or smaller extent in producing the present level of ionizing radiation in man, a brief account of the maximum permissible doses recommended may be given.

The International Commission on Radiological Protection (ICRP) at its session in Geneva, April 1956, decided to make the following additions to their earlier recommendations:

* * * * * * *

> A *controlled* area is one in which the occupational exposure of personnel to radiation or radioactive material is under the supervision of a radiation safety officer.
>
> For such personnel the maximum permissible levels of exposure are those specified for occupational exposure. In the case of prolonged exposure to radiation from external sources the maximum permissible levels for occupational exposure are represented by weekly doses of 600 mrem in the skin and 300 mrem in the blood-forming organs, the gonads and the lenses of the eyes.

* * * * * * *

> For any person in any place outside of controlled areas the maximum permissible levels of exposure of 10% of the occupational exposure levels.

* * * * * * *

> When genetic aspects of the effects of radiation are considered, the dose received by the whole population is of importance. Scientific data derived from human as distinct from experimental animal populations are so scanty that no precise permissible dose for a population can, at present, be set. The available information is being assessed by the Commission and other groups including geneticists. Until general agreement is reached, it is prudent to limit the dose of radiation received by gametes from all sources additional to the natural background to an amount of the order of the natural background in presently inhabited regions of the earth.

* * * * * * *

The recommended maximum permissible weekly doses and the modified values for special circumstances, permit a desirable degree of flexibility for their application. In practice it has been found that in order not to exceed these maximum limits and also to comply with the general recommendations of the Commission "that exposure to radiation be kept at the lowest practicable level in all cases" a considerable factor of safety must be allowed in the design of protective devices and operating procedures. Therefore, under present conditions, it is expected that the average yearly occupational dose actually received by an occupationally exposed person would be about 5 rems and the accumulated dose in the employment period up to 30 years of age would be about 50 rems. Accordingly, the Committee recommends continuation of the present conservative practice as regards doses actually received by occupationally exposed personnel, to keep the accumulated dose as low as practicable especially up to age 30.

In the report of the Medical Research Council (MRC)[2] "The Hazards to Man of Nuclear and Allied Radiations," issued in June 1956, the following conclusions are drawn:

* * * * * * *

> (2) Dose levels to the individual: (a) In conditions involving persistent exposure to ionizing radiations, the present standard, recommended by the International Commission on Radiological Protection, that the dose received shall not exceed 0.3 r weekly averaged over any period of 13 consecutive weeks, should, for the present, continue to be accepted.
>
> (b) During his whole lifetime, an individual should not be allowed to accumulate more than 200 r of "whole-body" radiation, in addition to that received from the natural background, and this allowance should be spread over tens of years; but every endeavour should be made to keep the level of exposure as low as possible.
>
> (c) An individual should not be allowed to accumulate more than 50 r of radiation to the gonads, in addition to that received from the natural

[2] Medical Research Council, "The Hazards to Man of Nuclear and Allied Radiations", London, 1956.

> background, from conception to the age of 30 years; and this allowance should not apply to more than one-fiftieth of the total population of this country.
>
> (3) Dose level to the population: Those responsible for authorizing the development and use of sources of ionizing radiation should be advised that the upper limit which future knowledge may set to the total dose of extra radiation which may be received by the population as a whole, is not likely to be more than twice the dose which is already received from the natural background; the recommended figure may indeed be appreciably lower than this.
>
> * * * * * * *

In the report of the National Academy of Sciences in Washington (NAS)[8] "The Biological Effects of Atomic Radiation" the following recommendations are made.

Thus we recommend:

> (C) That for the present it be accepted as a uniform national standard that X-ray installations (medical and non-medical), power installations, disposal of radioactive wastes, experimental installations, testing of weapons, and all other humanly controllable sources of radiations be so restricted that members of our general population shall not receive from such sources an average of more than 10 roentgens, in addition to background, of ionizing radiation as a total accumulated dose to the reproductive cells from conception to age 30.
>
> * * * * * * *
>
> (E) That individual persons not receive more than a total accumulated dose to the reproductive cells of 50 roentgens up to age 30 years (by which age, on the average, over half of the children will have been born), and not more than 50 roentgens additional up to age 40 (by which time about nine tenths of their children will have been born).

Obviously it is generally agreed that at present it is desirable to limit the doses received by the gonads of individuals to less than 5 roentgens per year and 50 roentgens before 30 years of age and that the average dose to the gonads of the population as a whole should be kept very low; of the order of the natural background (ICRP) twice this level (MRC) or to 10 r before 30 years of age (NAS). The difference in these figures is not very important as their order of magnitude will in practice be about the same.

According to our present knowledge it seems likely that a dose of 30—80 r will (according to MRC) double the natural mutation rate in man, which is probably only to a minor fraction (perhaps about 10%) caused by ionizing radiations. The rest of the natural mutations will, to an unknown extent, be due to chemicals and to the thermal movements of the molecules. It seems to be highly desirable for mutations induced by chemicals in particular to be investigated, in order to elucidate the relative role of radiation-induced mutations.

The recommendations made by the organizations quoted above are as regards the whole population based mainly on the natural level of ionizing radiation. It is not the place here to discuss whether this is a corerct starting point, nor whether or not the maximum permissible dose levels recommended are reasonable. They are fixed after careful consideration based on our present, unfortunately very incomplete knowledge of the biological effects of small radiation doses, but are agreed to by specialists in biology, genetics, haematology, physics, and radiology having long experience in radiation protection both on the research and on the practical side.

With respect to the risks of injurious effects on man one matter may, however, be stressed. There must always be a reasonable ratio between what can be gained by the use of ionizing radiation and the risks of its injurious effects. The use in medicine of ionizing radiation for examination and treatment of patients therefore occupies an exceptional position, that has not always been taken into account in recent years, in the discussion of the problems of the general irradiation of mankind. There must certainly be a sound balance between the benefits of a good health service and the risks to the patients with respect to onset of malignant disease or genetic damage of which, however, we at present do not know very much.

---

[8] National Academy of Sciences, "The Biological Effects of Atomic Radiation", Washington, 1956.

NATURAL SOURCES OF IONIZING RADIATION

Cosmic radiation

The cosmic radiations produce the doses shown in Table I.

The values for O—4000 m have been calculated from the work of Compton and co-workers (Fig. 1) taking into consideration that some reduction due to absorption may be justified indoors, and the values for 6000—18,000 m from Millican and co-workers (Fig. 2). The values are fairly approximate, as there are many factors which are difficult to allow for at the higher altitudes, especially with regard to the unknown relative biological effectiveness of heavy nuclei rays.

Variations with time.—Major variations in cosmic radiation occur during short periods only, and the few occassions associated with an appreciable increase are so rare and of such short duration that the doses caused by cosmic radiation for a certain altitude and geomagnetic latitude may at the earth surface be regarded from the practical point of view as constant. A record of the variation in the cosmic radiation on February 23, 1956, is shown in Fig. 3. This is one of the occasions on which an extraordinary increase was observed. The dose due to this temporary increase was, at sea level, less than 0.03 millirem.

As to the long-term variations, it seems highly unlikely that any major variations in cosmic radiation have taken place during the last 2,000 years.

Variations with site.—The maximum variation between different places on the earth surface, excluding mountains of more than 4000 m high, is about 2 r per 30 years.

Doses to individuals.—The doses to individuals may be of importance for very high altitudes. The present development of communication by air makes it necessary to take into account the fact that, at very high altitudes, the maximum permissible dose of 50 rem may especially at high geomagnetic latitude already be exceeded if on the average some 10 hours per week are spent at this altitude during 10 years, which might well be possible for future personnel in aircraft. The increase in cosmic radiation on February 23 may perhaps at altitudes of 20,000 metres correspond to a dose of less than some tenths of 1 rem obtained during a few hours, therefore probably being of limited biological significance.

Doses to large populations.—The contribution to the irradiation of large population groups (>100,000) varies between 0.7 and 2.7 r or approximately between 1 and 3 rem per 30 years.

The fact that an appreciable part of the radiation can be screened off by reasonable quantities of material may be of certain value for judging the risk for stratosphere and intersteller traffic. Investigations of the biological effects of cosmic radiation at very high altitudes are however desirable, because of the lack of knowledge as to the RBE values for heavy nuclei radiations.

Natural external γ radiation

The external γ radiation in nature varies with the radium, thorium, and potassium content of the ground and of the building material in houses. The γ dose in free air produced above level ground can be calculated according to the simple formulae:[1]

Radium: dose in r per 30 years	$0.57 \cdot 10^{12}.s(Ra)$
Uranium: dose in r per 30 years	$0.20 \cdot 10^{6}.s(U)$
Thorium: dose in r per 30 years	$0.094 \cdot 10^{6}.s(Th)$
Potassium: dose in r per 30 years	$41 .s(K39)$

in which s(Ra), s(U), s(Th) and s(K39) are the contents of radium, uranium, thorium, and potassium in g element per g substance of the ground.

To obtain an estimate of the dose to the gonads in rad,[4] the doses in free air have to be multiplied by a factor of 0.5 for women and 0.7 for men (1) or on the average 0.6, to account for the absorption in the shielding part of the body. The same factor may be approximately applicable to most of the other organs For the skeleton, the factor might be considered on the average, to be about 0.8.

The doses due to natural γ radiation over ground containing various minerals are given in Table II, and the dose in dwellings in Sweden (1) are seen from Table III and Fig. 4. These are in good agreement with the few observations in other countries.

[4] 1 rad corresponds to a dose of about 1.07 r in soft tissue.

The γ radiation from the ground is absorbed by snow, as seen from Fig. 5. A snow cover of 40 cm depth and of medium volume weight absorbs about 50% of the γ radiation from the ground.

A factor of considerable importance is the relation between the time spent indoors and out of doors. Here, it is assumed that on an average in large population groups ¼ of the life is spent out of doors.

As an additional contribution to the irradiation of man from natural radioactive elements in the earth crust, the radon and thoron of the air may play an important role in special cases. In general, the content of these elements in the air is too small to contribute to the dose received by the human body by more than a few percent. In some places and during some periods, this content can be fairly high, for instance in rooms where water of high radon concentration is used or the ventilation is insufficient, (1) in cellars where radon and thoron comes up from the earth, and in large cities during calm weather. (2) Such cases seem only occasionally to have been investigated. It would probably be worth while to make more systematic studies in this field. At present, these sources of natural radiation are too little known to be treated in this survey and will therefore be disregarded, although it is possible that they are of significance for the irradiation of the pulmonary system of a comparatively great number of individuals living in certain areas.

Variation with time.—The average annual dose to human beings due to natural sources has probably been of roughly the same magnitude during the present geological period. A slight decrease in the radiation occurred when man learned to use wood for building houses, and stopped living in earthen huts or in rocks where the amount of radon in the air was sometimes probably quite high. An increase subsequently took place again with the use of bricks and concrete as building materials, and when people moved to cities where material containing minerals more frequently comprise the surrounding material.

Another factor which may have caused a reduction in the environmental γ radiation for some populations may have occurred during the ice periods followed by certain areas covered by ice and snow during a greater part of the year than today. As already shown the snow causes an absorption of the γ radiation from the ground which appreciably reduces the irradiation out of doors, and produces a seasonal variation (see Fig. 6) in the irradiation of large population groups, especially those living outside the cities.

Variation with site.—As a rule, the difference in the level of natural γ radiation in different parts of the world is probably not very large. Even over areas containing rich uranium or thorium ores, the γ doses to the inhabitants only in rare cases exceed a few times the normal level. This is because the ores are generally very unevenly distributed both in rocks and sands, and are often covered or surrounded by material of normal radioactivity. The inhabitants moving over the area in question might thus, on an average, be exposed to doses which are much lower than could be conceived. This experience based on observations in Sweden needs further verification, but will probably be found to apply to most population groups throughout the world.

The doses of γ radiation to persons living in places more or less permanently covered by deep ice or snow, and those spending most of their time at sea, are generally very small. Here, the amount of γ radiation from the earth is often so minute that it can be entirely disregarded in comparison with the radiation from other natural sources. Recent investigations of the radiation level on wooden and iron vessels of different size have shown that the γ radiation already at a few metres distance from a granite quai is entirely negligible.

In Table IV some observations of natural γ radiation in Sweden are compared with the results of similar investigations in the UK and USA.

In view of the statements in the aforegoing with respect to the average doses received by individuals, it would be of interest to carry out long term measurements by means of personal monitoring, in order to arrive at reliable data regarding the doses actually received.

The natural content of radioactive elements in man

In areas where the radium content of drinking water and food is not exceptionally high, the potassium content of human tissue is the main source of internal irradiation (see Fig. 7). The doses in rad due to the amount of potassium 40 (0.012% in natural potassium) in some human organs is shown in Table V. With respect to some tissues, particularly bone, the data of different authors vary considerably.

The content of carbon 14 and radon contributes about 5 and 10%, respectively, of the average potassium radiation.

According to measurements by Hursh & Gates (3) and recently by Sievert & Hultqvist (4) (Fig. 8), the radium content of the ekeleton is probably less than $0.3 \cdot 10^{-9}$ g, in areas with a radium content in the water of less than 0.2 $\mu\mu$ C per litre. According to Spiers (MRC), the mean dose to the osteocytes is about 6 rem per 30 years for $0.5 \cdot 10^{-9}$ g total radium body burden. The radium amount is, however, very unevenly distributed in the skeleton, and the dose significant for the production of osteosarcoma therefore seems to be extremely difficult to assess.

The *variation with time and site* in the natural internal irradiation is mainly a question of the variations in the radium content of water and food and of the radon in the air. Referring to what has already been said, it may be stated that there are not at present sufficient data available to give any reliable figures for different areas in the world. This also applies to the problem of the natural radioactive elements taken up in the pulmonary system. With respect to these matters, references can be made to a recent publication by Hultqvist (loc. cit.) in which an extensive bibliography is given.

The common limits of the doses to large population groups (>100,000) and to individuals from natural radiations are collected in Table VI.

MAN-MADE SOURCES OF IONIZING RADIATIONS

Radioactive material and technical arrangements producing ionizing radiation used under such circumstances that the user is generally aware of the presence of the radiation

Here, *occupational exposure* and *exposure of patients* undergoing treatment or investigation in radiology are the two matters to be considered.

The doses received by those carrying out work with ionizing radiation in education, science, medicine, technics, and industry are in most cases small, as the personnel can generally be adequately well protected. Furthermore, in all work where patients are not involved, there is no reason to permit irradiation which can in any way cause ill-effects. Here, the maximum permissible levels for individuals and large population groups are exceeded in rare cases only.

In radiology, especially some procedures in γ-ray therapy and in examinations using X rays, circumstances do not always permit entirely satisfactory protection of doctors and personnel. Here, the individual dose will sometimes be close to the maximum permissible levels, or even occasionally exceed them.

The occupational doses contribute to the radiation per capita of whole populations with an amount which in the UK (MRC) has been estimated at about 2.5 r per year as an average for about 14,000 people in research, medical and industrial work, and at about 0.4 r per year for about 7000 people in atomic energy work. Altogether, the average gonad dose per capita due to occupational exposure is estimated at 0.0016 r per year or, if 10 years is supposed to be the average period of work before reproduction, the relevant average gonad dose for the whole population may be less than 0.02 r before 30 years of age. An estimate of the corresponding figure for Sweden has given a considerably lower figure.

The occupational dose is apparently over the whole world attributed mainly to medical radiology, but is presumably very uncertain. It seems, however, that occupational irradiation does not at present contribute to the gonad dose of whole populations with any appreciable amount.

The doses received by patients undergoing treatment and examination by means of ionizing radiations, on the other hand, are of decisive importance, since they contribute by far the largest exposure of the population to man-made sources of radiation. In Germany, France, Sweden, the UK, and the USA, investigations have been carried out in order to ascertain the doses to the patients during various types of radiologic procedures. Numerous publications are available but up to now estimations to find a correct figure for the present average dose to the whole population due to the irradiation of patients have been made only in Sweden, the UK, and the USA. The results are that the average gonad dose per capita to the patients examined seems to be of the magnitude of 1–3 r in 30 years. The reliability of these figures has been much discussed, and it seems advisable to await further investigations based on radiation measurements and some sort of sampling method, before accepting any definite figures. It is nevertheless highly probable that the order of magnitude of the figures quoted is correct, since the estimations were made independently in three different countries.

Sources of radiation used for purposes in which, as a rule, only the specialist is aware of the present of ionizing radiation

At present, we are faced in this field with only a few matters of minor significance. The average gonad dose from luminous compounds in watches is found in the UK (MRC) to contribute to the average gonad dose by 0.001 r per year, and the radiation from television sets to a still more insignificant dose.

In the future development of atomic energy it seems highly probable, however, that the use of radioisotopes for various purposes will change the situation, and constitute a new problem by the distribution in the community of a large number of small radiation sources, each being completely harmless individually, but collectively raising the level of irradiation of the population.

Artificial radioactive elements distributed in nature

WHO and the study group which it has convened are concerned with the peaceful use of atomic energy and the effects of, for instance, radioactive waste disposal from such peaceful uses. However, it is essential to take into consideration the evidence available from atomic weapon tests since the problems in the field of artificial radioactive elements distributed in nature are at the present time mainly related to fallout from these tests. The dose due to the external γ radiation from fallout may at present (December 1956) be disregarded in comparison with the internal dose.

If the fallout in the vicinity of the test area and the effects of radiation during the first few days after the explosion are disregarded, two different effects may be of interest. One is caused by mixed fission products of medium half-life (a few days to less than one year), the other by the fission products of long half life, particularly Sr 90 (28 years) and Cs 137 (33 years).

Fission products of medium half-life are very unevenly distributed over the world after an atomic explosion. Here, meteorological circumstances play a most important role, since a jet stream, a cold or warm front causing turbulence in the atmosphere, and rain or snowfall can lead to a concentration of the radioactive material in some areas even at a great distance (several thousand kilometres) from the explosion.

A typical example of such an effect is given in Fig. 9, showing the γ radiation recorded during about one month in the four northernmost places indicated in Fig. 3b. The increase in the γ radiation occurred about five days after an atomic bomb test. It is obvious from these observations that a comparatively narrow set of stations is required to give an adequate picture of the distribution.

It has been shown by recent measurements of the γ radiation from large samples of foodstuffs in Sweden that most of our food today (milk, beef, corn and vegetables) now contains artificial radioactive elements, in many cases greatly exceeding the K 40 radiation level of animals and plants. As an example, a decay curve obtained from powdered milk is shown in Fig. 10.

After some bomb tests J 131 is easily detectable in the thyroid of growing cattle. The content of this element in Swedish cattle during September–October 1956 is shown in Fig. 11. The maximum dose per week was here 0.04 rad, or about 20 times the dose due to the average natural radiation, which can be considered to be about 0.002 rad per week. It is to be noted that the effects demonstrated in Figs. 10 and 11 are due mainly to atomic bomb tests in August and September 1956, but that even before that time the foodstuffs were contaminated to an easily detectable extent, partly owing to medium half-life elements.

It is impossible to estimate today what doses have been received by populations in different parts of the world from mixed fission products. In comparison with the doses from the fallout of Sr 90 and Cs 137, the mixed fission products may in many cases give smaller doses calculated over a long period. It must, however, be borne in mind, that many biological effects are dependent—perhaps more than we know at present—on the intensity of the radiation. Often there is a threshold to be exceeded before a biological effect is obtained. Our knowledge of the effects of small doses over long periods is very scanty and we cannot as yet be sure that the time-intensity factor can be disregarded, even with respect to genetic effects.

The fallout of Sr 90 and Cc 37 has been carefully studied during the past years. These elements are probably comparatively evenly distributed over the whole world (with the possible exception of the polar regions). Large amounts of these elements remain in the upper atmosphere and will successively contribute to an increase in their present abundance on the earth surface by a factor of 3–5, even if the firing of atom bombs is stopped. The incorporation of Sr 90 into the skeleton may, in places where the calcium content of the soil is small, be regarded as important.

It does not yet seem possible to estimate the doses to human tissue due to fall-out, nor their distribution in time, which are necessary data for judging its possible biologic significance. Experience during the past year is, however, likely to raise doubts as to the lack of biologic importance to the tests of nuclear weapons, at any rate if they are continued on the present scale.

It is extremely difficult to predict what will in the future be the most important sources of radiation caused by artificial radioactive elements distributed in nature. There is reason to believe that the problems of disposal of radioactive wastes will be satisfactorily solved, and that precautions in the handling and use of radioactive material will be adequate, but accidents and unforeseen events may gradually spread radioactive substances of medium and long half-life beyond control. These radioactive materials will follow unknown paths, and may be harmful to mankind in ways that will become known to us only after long experience.

REFERENCES

1. Hultqvist, B. (1956) Studies on naturally occurring ionizing radiations, *Kungl. Svenska Vetenskapsakademiens Handlingar*, Ser. 4, Vol. 6, No. 3
2. Anderson, W., Mayneord, W. V. & Turner, R. C. (1954) *Nature, 174*, 424
3. Hursh, J. B. & Gates, A. (1950) *Nucleonics*, 7, 46
4. Sievert, R. & Hultqvist, B. (1956) *Brit. J. Radiol.* Suppl. 7, 1

TABLE I.—*Roughly estimated doses in soft tissue by cosmic radiation in rad per 30 years (figures in brackets are estimated values in rem considering a rbe of 10)*

Geomagnetic latitude	Dose in rad at an altitude above sea level in metros at						Hours per week to accumulate a dose of 50 rem during 10 years at 18 000 m.
	0 [1]	2000 [1]	4000 [1]	6000 [2]	12 000 [2]	18 000 [2]	
0°	0.7	1.1	2.0	8	35	40 (400)	63 h
40°	0.8	1.3	2.5	12	70	110 (1100)	25 h
>60°	0.8	1.4	2.7	14	85	150 (1500)	17 h

[1] Calculated from measurements of A. H. Compton and co-workers. (See D. Halliday, 1950: Introductory nuclear physics. New York, p. 461.)

[2] Calculated from measurements of R. A. Millikan and co-workers. (See H. J. Schaefer, Oct. 1950, Aviation Medicine, p. 383.)

TABLE II.—*Calculated gonad doses above various minerals*

Mineral	Ionization (ion pairs/cm³/sec) due to content of			Gonad dose (excluding cosmic radiation) r per 30 years
	Ra	Th	K	
Igneous rocks:				
Average	1.6	2.5	2.4	1.9
Granites	------	------	3.2	------
North America, Greenland	2.0	1.7	------	2.0
Finland	5.9	5.9	------	4.1
Alps	5.5	6.9	------	4.3
Basalts	------	------	1.2	------
North America, Greenland	1.2	2.1	------	1.2
England, Germany, France, and Hungary	1.6	1.9	------	1.2
Sedimentary rocks:				
Sandstone	0.4	1.0	0.9	0.8
Limestone	1.3	0.2	0.3	0.6
Alum shales in Sweden	75	0.3	3.2	21.0
Ore containing: [1]				
1% U	------	------	------	1,000
1% Th	------	------	------	500
0.01–0.001% Th [2]	------	------	------	0.5–5.0

[1] The uranium and thorium are in most cases very unevenly distributed and therefore the figures given here may be of limited practical value. According to a personal communication from Professor Z. M. Bacq, University of Liége, the background radiation in Kataanga, Belgian Congo, will reach 100–150 times the normal background.

[2] Travancore sand, containing monazite, according to a personal communication by J. Eklund, Geological Survey of Sweden.

TABLE III.—*Summary of the results of gamma radiation measurements in Swedish dwellings (calculated from Hultqvist's[1] figures; cosmic radiation excluded)*

Building material in outer walls	Mean gonad dose in r per 30 years		
	Middle of room	Highest value recorded	Lowest value recorded
Wood	1.0	1.1	0.95
Brick	2.0	2.2	1.9
Light-weight concrete containing alum shale	3.2	3.8	3.0

[1] Hultqvist, B. (1956) *Kungl. Svenska Vetenskapsakademiens Handlingar*, Ser. 4, Vol. 6, No. 3.

TABLE IV.—*Average values for the irradiation of large population groups due to natural sources*

Sweden	2–5 rem/30 years.
UK	2–3 rem/30 years.
USA	4.3 rem/30 years.

TABLE V.—*Potassium content in adult human subjects according to Shol[1] (A) and Forbes & Lewis[2] (B & C) and the dose due to K 40*

Organ	Weight in percent of whole body			Percent K 39			Dose in organ r in 30 years mean (B and C)
	A	B	C	A	B	C	
	kg	*kg*	*kg*				
Skin	7.3	6.4	6.5	0.09	0.15	0.16	0.30
Skeleton	17.5	17.5	14.7	0.055	0.10	0.11	0.20
Tibia	--------	1.4	--------	--------	--------	0.05	--------
Muscle	43.0	39.5	39.6	0.42	0.33	0.30	0.62
Nerve	--------	3.0	2.1	--------	0.28	0.29	0.56
Liver	2.7	2.3	2.3	0.17	0.27	0.22	0.49
Heart	0.5	0.5	0.6	0.13	0.22	0.19	0.40
Lungs	1.5	3.3	2.2	0.15	0.24	0.26	0.50
Kidneys	0.5	0.5	0.4	0.17	0.16	0.22	0.38
Gl. tract	--------	1.8	1.5	--------	0.13	0.13	0.26
Adipose	--------	11.3	21.4	--------	0.08	0.06	0.14
Remainder	--------	11.3	6.4	--------	0.18	0.17	0.34
Weight loss on dissection	--------	2.6	2.2	--------	--------	--------	
	70	53.8	73.5	0.205	0.212	0.190	0.40

[1] Shol, A. T. (1939) *Mineral Metabolism*, New York, Reinhold Publishing Corp., 19.
[2] Forbes, G. B. & Lewis, A. M. (1956) *J. clin. Invest.* 35, 596.

TABLE VI.—*Estimated values for the irradiation of the gonads of the population due to natural sources in rem per 30 years*

	For large population groups			For individuals	
	Minimum	Maximum	Average	Minimum	Maximum
Cosmic radiation	0.7, including screening in dwellings.	3?, about 4,000 m above sea level.	1?	0.5? (for some miners).	5? (50?) 3% of 30 years 18 000 m above sea level.
Natural radiation:					
¼ of 30 years out of doors.	<0.1 above water (in boats), snow and ice.	1 above igneous rocks.	0.5	0	15 (20?).
¾ of 30 years indoors.	0.9, in wooden houses.	3, in some types of brick and concrete houses.	2		
Radon in air	0.03, out of doors and in wooden houses with good ventilation. (3×10^{-13} c/1)	0.8, in cellar and in stone houses with poor ventilation. (50×10^{-13} c/1)	0.2	<0.01 (<10^{-13} c/1).	2.0 (10^{-11} c/1).
K 40 in body (+0.03 for C 14):					
Fat	0.2	0.2	0.2	0.2	0.2.
Muscles	0.7	0.7	0.7	0.7	0.7.
Gonads	0.5	0.5	0.5	0.5	0.5.
Approximate sum for gonads.	2	6 (8?)	4	1	20 (>50?).

INDEX TO FIGURES

FIG. 1

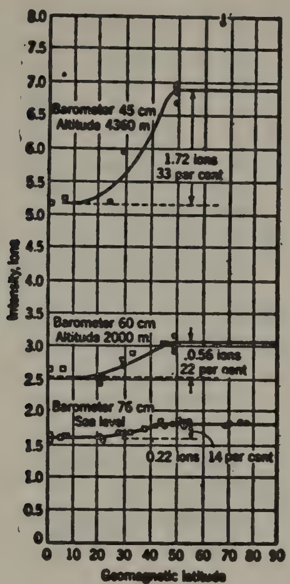

Barometer 45 cm
Altitude 4360 m
1.72 ions
33 per cent
Barometer 60 cm
Altitude 2000 m
.0.56 ions
22 per cent
Barometer 76 cm
Sea level
0.22 ions
14 per cent
Intensity, ions
Geomagnetic latitude

FIG. 2

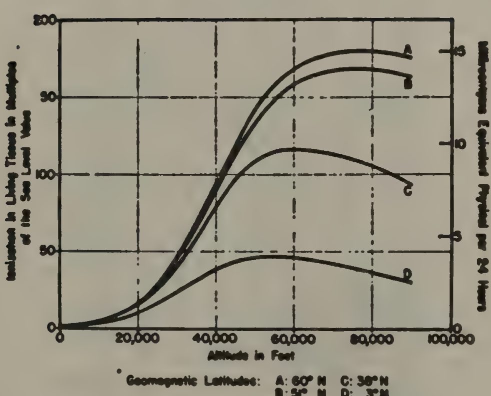

A
B
C
D
Altitude in Feet
Geomagnetic Latitudes: A: 60° N C: 38° N
B: 51° N D: 3° N

FIG. 3a

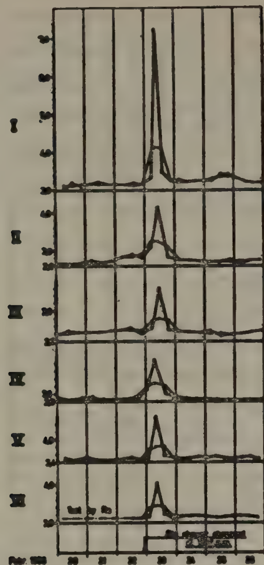
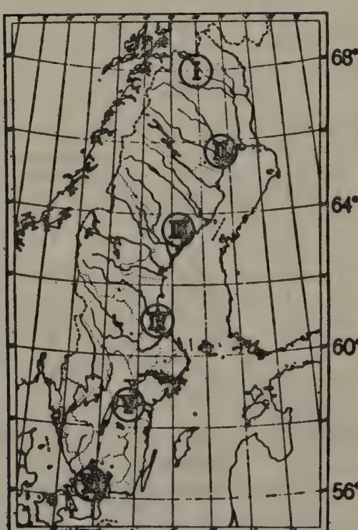


FIG. 3b



Scale 1:16 mill.

FIG. 4

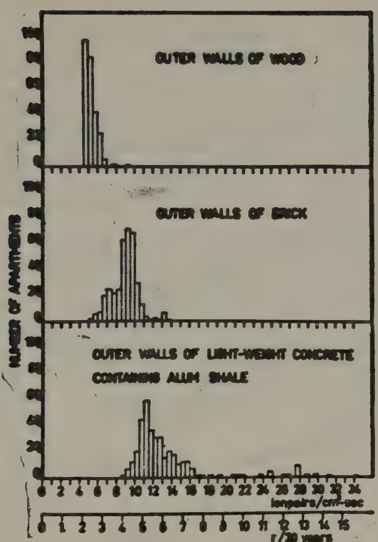

OUTER WALLS OF WOOD
OUTER WALLS OF BRICK
OUTER WALLS OF LIGHT-WEIGHT CONCRETE
CONTAINING ALUM SHALE
NUMBER OF APARTMENTS
ionpairs/cm³-sec
r/30 years

FIG. 5

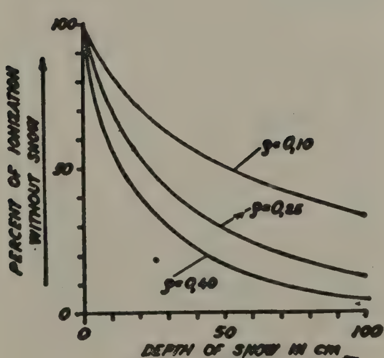

PERCENT OF IONIZATION WITHOUT SNOW
DEPTH OF SNOW IN CM
g=0,10
g=0,25
g=0,40
100
50
0

FIG. 6

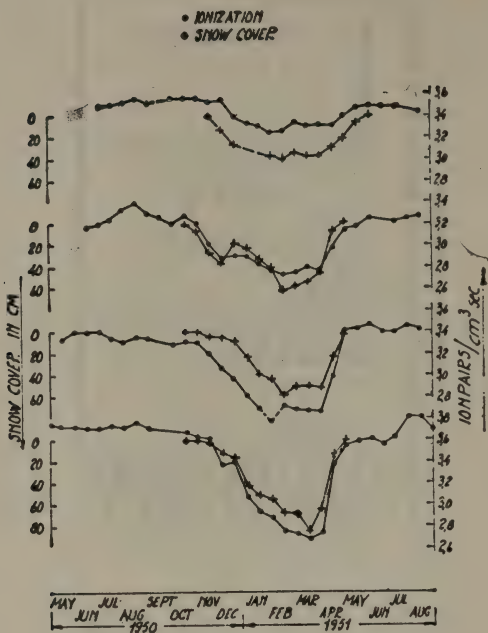

• IONIZATION
• SNOW COVER
SNOW COVER IN CM
ION PAIRS/cm³ SEC
MAY JUN JUL AUG SEPT OCT NOV DEC JAN FEB MAR APR MAY JUN JUL AUG
1950
1951

FIG. 7

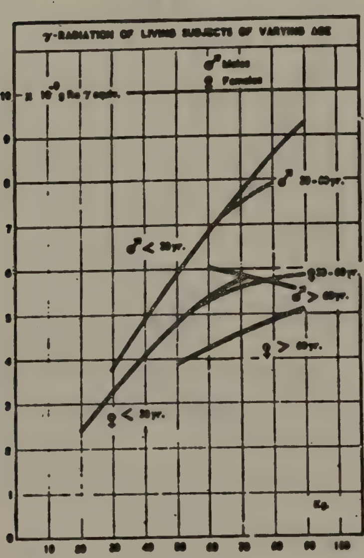

γ-RADIATION OF LIVING SUBJECTS OF VARYING AGE
♂ Males
♀ Females
0 1 2 3 4 5 6 7 8 9 10
10 20 30 40 50 60 70 80 90 100

FIG. 8

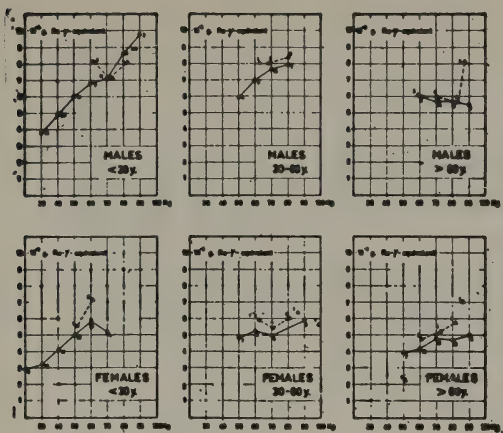

MALES
<20y.
MALES
20-60y.
MALES
>60y.
FEMALES
<20y.
FEMALES
20-60y.
FEMALES
>60y.

FIG. 9

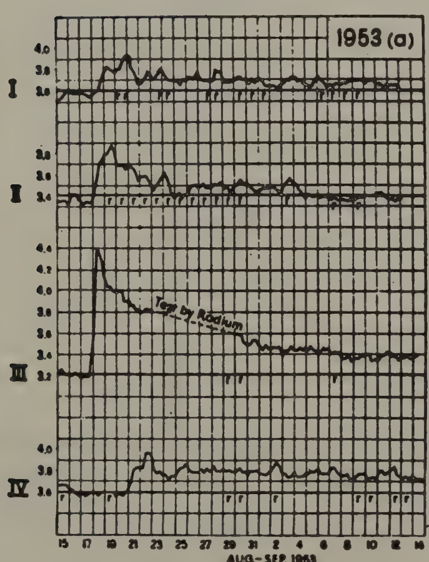
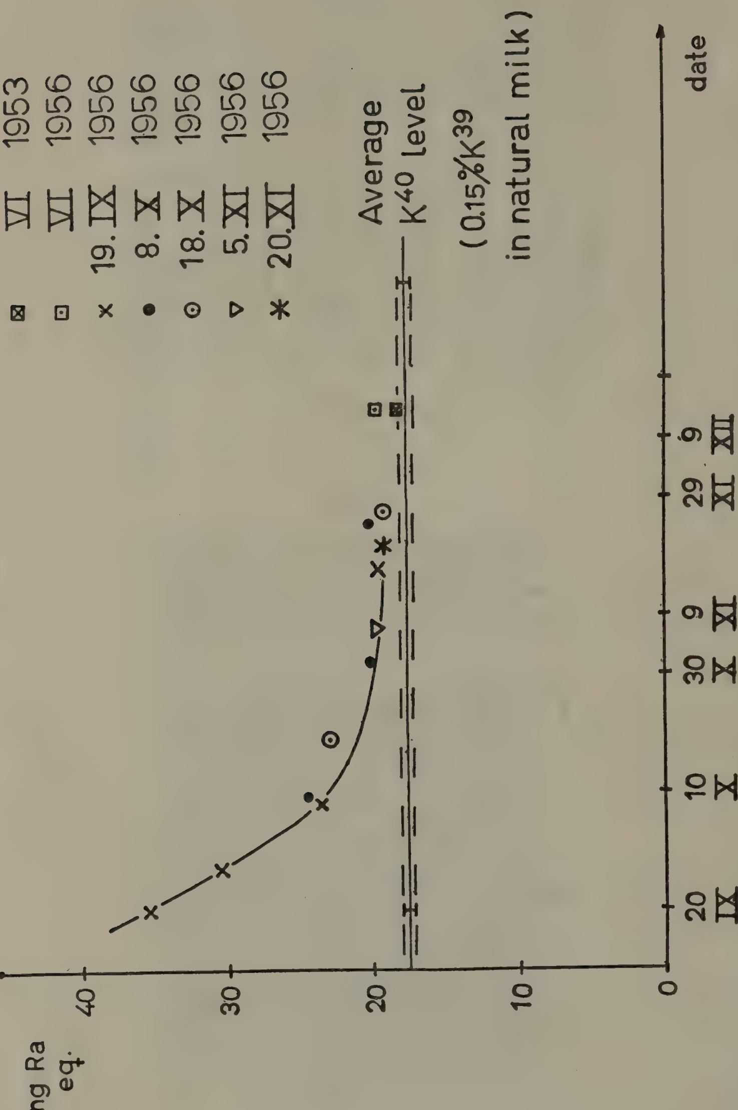

1953 (a)
I
II
III
IV
Test by Radium
15 17 19 21 23 25 27 29 31 2 4 6 8 10 12 14
AUG-SEP. 1953

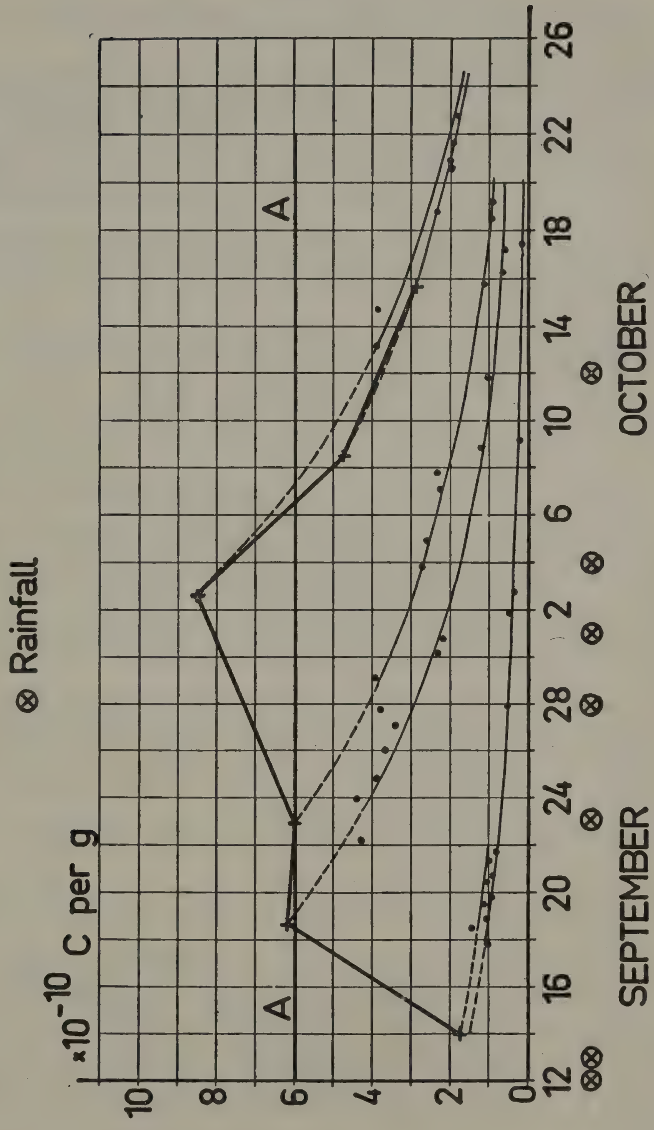
FIG. 10



ng Ra eq.
40
30
20
10
0
⊠ VII 1953
⊡ VII 1956
× 19. IX 1956
● 8. X 1956
⊙ 18. X 1956
▷ 5. XI 1956
✱ 20. XI 1956
Average K^{40} level
(0.15% K^{39}
in natural milk)
20 IX
10 X
30 X
9 XI
29 XI
9 XII
date

FIG. 11

^{131}J, PER G OF THYROID IN GRAZING CATTLE



Annex 5

Detection of Induced Mutations in Offspring of Irradiated Parents [1]

THE PROBLEM OF THE GONAD DOSE

Among the various sources of ionizing radiation, apparatus for radiodiagnosis and radiotherapy today represents the main contribution of man to the increase in background radiation.

Radiodiagnosis, which is being carried out more and more frequently every year in all the developed countries, at present plays a dominant role in this connexion. Ranging as it does from the radiographs and radioscopies called for by some pathological condition, to the periodic radiographs of the whole skeleton used in studying the growth of normal children, the field of exploratory radiology now covers an extremely large proportion of the population; for example, in France, all children of school age are submitted yearly to systematic radiographic examination.

While there can be no doubt that most radiodiagnostic examinations affect the gonads only very slightly, all examinations of this type which involve the pelvic region may have genetic effects (for example, gynaecological and obstetrical examinations).

One has only to consult the appropriate tables, such as the one drawn up by Plough,[2] to see that radioscopy may involve very high doses; for example, 10 to 20 r per minute for a gastro-intestinal radioscopy. While techniques such as radiocinematography, which, happily, are not used very extensively, are much more harmful, since they involve even higher doses. Several authors have attempted to calculate the mean gonad dose, on the basis of isodose curves, for whole populations. The estimates vary according to the author, but, in general, the gonad dose may be taken as being of the order of one roentgen; in other words, it represents a very considerable fraction of the quantity of natural radiation to which we are inevitably exposed.

Be this as it may, these estimates are based on the assumption that the radiation emitted by such apparatus is of known quality and that the methods employed in its application are standardized, which is very far from being the case. In fact, considering that radioscopic apparatus, often poorly shielded, is to be found more and more frequently in the consulting-rooms of general practitioners, one is justified in concluding that a very high proportion of radioscopic examinations are being carried out by physicians with no formal training in radiology.

In the opinion of the writer and his colleague, Professor R. Turpin, it would be desirable to obtain, by means of surveys among general practitioners as well as in hospitals, an experimental estimate of the gonad dose involved in any given radiological examination, under the actual conditions in which the said examination is carried out. Such surveys would make it possible to check the validity of the extrapolations at present in use.

A simple method would be to place micro-counters—or, perhaps even better, small films of the type used in atomic plants—in contact with the genital organs of all persons examined.

Even if such surveys were to result only in an increase in the precautions taken by those using X-ray apparatus, they would have partially fulfilled their purpose.

Radiotherapy certainly represents a much smaller risk from the genetic point of view, on the one hand because it is carried out by specialists, and on the other because in most cases the people concerned are elderly. Nevertheless, on examining the statistics of the radiotherapeutic services of the Paris hospitals, Turpin, Lejeune & Rethore[3] found that among 238,800 case-histories there were 4.428 cases of pelvic irradiation of adults under 35 years of age. The proportion of the total—about 2%—is, of course, low, but it does represent cases in which the radiation has impinged directly on the gonads of subjects who are still young enough to reproduce.

Although the calculation of the gonad dose is much more reliable in radiotherapy than in radiodiagnosis, direct determination during all forms of treat-

---

[1] Submitted by Dr. J. Lejeune, Chargé de Recherche au Centre National de la Recherche scientifique, Paris, France.

[2] Plough (1952) Nucleonics, 10, 17.

[3] Turpin, R., Lejeune, J. & Rethore, M. O. (1956), Etude de la descendance de sujets traités par radiothérapie pelvienne. Note préliminaire (paper presented at the First International Congress of Human Genetics, Copenhagen; unpublished).

ment (even extra-pelvic), by means of films or micro-counters placed in contact with the scrotum or in the vaginal fornix, would also be very desirable.

It follows from the foregoing considerations that the accuracy of an estimate of the mean gonad dose received in 30 years by nubile subjects should be checked by random sampling to determine the dose actually received under various examination conditions. It should be noted, however, that such work will be useless if it is not accompanied by systematic recording of the doses received by every individual in the population.

One method of obtaining systematic records, though perhaps a little startling, has already proved its worth. It entails treating the administration of X-rays in the same way as that of morphine for example, by providing all owners of X-ray apparatus with a counterfoil book (of the type used for prescribing narcotics), in which every radiological operation should be recorded and the following particulars given: name, age, and address of patients; reason for the examination, area examined; and details required for calculation of the dose (e. g., kilovolts, millicuries, type of filter, size of beam, etc.).

In practice, this form of registration would probably involve very considerable difficulties, but the increasing socialization of medicine would probably make it feasible for a known fraction of all radiological operations and would certainly draw the attention of the medical profession to a danger of which it is but little aware at the present time.

METHODS FOR DETECTION OF MUTATIONS INDUCED IN OFFSPRING OF IRRADIATED SUBJECTS

Since human geneticists cannot employ methods such as those used for *Drosophila,* they have to resort to statistical comparison of two populations of children supposedly identical in all respects save the dose of roentgens received by the gonads of their parents.

(a) *The study of abnormalities and malformations* may reveal an increase in frequency in the progeny of the irradiated group. This increase could be considered to be linked with the appearance of unfavourable dominant mutations. Although probably reliable in the case of definite genetic syndromes such as achondroplasia, this method is much less precise in respect of congenital abnormalities as a whole, since the latter are affected by extremely varied factors (age of mother, parity, etc.). However, if sufficient data are to be obtained to make it possible to draw conclusions, all abnormalities must be considered.

(b) *The study of the frequency of sex-linked recessive diseases* in the sons of irradiated mothers, although theoretically possible, necessitates such a large number of observations that it has not been undertaken.

(c) *The production of lethal genes* can be more easily detected. The most serious effect, sterility, is the one most generally known, but measurement of sterility or even of subfertility in man is extremely difficult. As has been remarked elsewhere (Turpin & Lejeune),[4] the actual fertility of civilized populations is hardly a third of the potential fertility of non-malthusian societies, which greatly diminishes the possibility of demonstrating the effects of sterility.

On the other hand, it is logical to expect the production of dominant lethals to bring about an increase in the frequency of miscarriages, which is difficult to establish, and of stillbirths, which can be obtained with much greater accuracy.

It is, however, essentially in the X-chromosome that the lethal genes can be detected, through a study of the sex ratio. Owing to the chromosomal structure of sex, the X-linked lethal mutations appear in different forms according to the sex of the irradiated parent. Thus, in the offspring of a woman exposed to radiation, dominant lethal mutations linked to the sex chromosome have no effect on the sex ratio, whereas sex-linked recessives bring about a deficit of boys. The contrary is true of men, in whose progeny only the dominant lethals manifest themselves by bringing about a deficit of girls.

If we call "*n*" the average number of dominant lethals linked to the X-chromosome in the offspring of men who have received a given dose of roentgens, then the following simple equation should apply:

$$\text{frequency of surviving daughters} = \frac{}{\text{number expected}} \times e^{-n},$$

[4] Turpin, R. & Lejeune, J. (1955), Bull. Acad. nat. Méd. (Paris), No. 5/6, p. 104.

since the number of mutable loci in the X-chromosome should be large enough for the distribution to be of the Poisson type. Moreover, since it may be taken as a first approximation that this average number, n, should be identical for all the chromosomes, the autosomally viable zygotes represent a fraction of the total fertilized ova roughly equal to $(e^{-n})^{22}$, and it is in this fraction alone that it will be possible to observe a disturbance of the sex ratio.

Similar reasoning can be applied to the case of the offspring of irradiated women, bearing in mind the fact of that there is a relationship between the frequency of the dominant and the recessive lethals.

Since, in theory, the parameter n bears a linear relationship to the roentgen dose received, and since our estimates of the gonad dose are very approximate, there is probably a fairly strong correlation between the actual fertility of the parents after irradiation and the deviation of the sex ratio observed in their progeny. In other words, the most pronounced variations in the sex ratio would be shown by the offspring of parents who are almost sterile owing to the irradiation of one of them (for example, couples who have only one child).

It follows from the above that, in the absence of an accurate estimate of the gonad dose, an overall study of the sex ratio of all children born of an irradiated father or mother may, if it does not take into account the number of siblings born after treatment, result in the masking of the phenomenon by a "dilution effect", caused by the very numerous siblings who are the issue of a parent relatively slightly affected.

Further, the problem of control samples can only be correctly solved by comparing children born before and after treatment of the same irradiated parent and thus eliminating any genetic factor due to the couple itself, as well as the possible influence of siblings of one sex only.

Finally, and bearing in mind the above limitations, it would seem that the sex ratio is the most sensitive touchstone for detecting the production of lethal mutations in the first generation of children born of irradiated parents.

INFORMATION AVAILABLE AT PRESENT

Relatively few direct investigations of the influence of irradiation have been carried out and the writer will mention them in succession here, under the headings of the three main characteristics referred to earlier: frequency of abnormalities, frequency of miscarriages and stillbirths, and variations in the sex ratio.

(a) Frequency of abnormalities

Murphy & Goldstein [5] and Maurer [6] have published statistics on the offspring of women treated with X-rays or radium in the pelvic region. Unfortunately, neither of these papers can be considered very satisfactory, owing to the lack of detail concerning the families, on the one hand, and the absence of any controls, on the other.

Two recent papers cast more light on this question. In 1953, Neel et al.,[7] on studying the offspring of the survivors of the atomic bombing of Nagasaki and Hiroshima, did not find any increase in the frequency of serious abnormalities. While Macht & Lawrence [8] on comparing the children of fathers who were radiologists with those whose fathers were medical specialists not exposed to ionization risks, found a significant overall increase in abnormalities among the progeny of the radiologists. Unfortunately, the latter authors on the one hand included as abnormalities syndromes of a very varied and sometimes quite unsuitable nature (foetal erythroblastosis, for example) and on the other accepted the diagnosis made by the parents themselves, who, though admittedly physicians, clearly lacked the necessary objectivity. These considerations greatly limit the significance of the conclusions drawn by Macht & Lawrence, but it is only fair to stress that if, from the authors' data, those relating only to congenital forms of heart disease are selected, the increase pointed out among the progeny of the radiologists remains statistically significant.

(b) Frequency of miscarriages and stillbirths

Macht & Lawrence [8] mention a non-significant increase in the overall frequency of stillbirths plus miscarriages, while Neel et al.[7] report a non-significant

---

[5] Murphy, D. P., and Goldstein, L. (1929), Amer. J. Roentgenol. 22, 207.
[6] Maurer (1933), Zbl. Gynäk. p. 819.
[7] Neel, J. V. et al. (1953), Science, 118, 537.
[8] Macht, S. H., and Lawrence, P. S. (1955), Amer. J. Roentgenol. 73, 442.

increase in the frequency of stillbirths. Finally, Crow,[9] who studied the offspring of American radiologists through a survey, by questionnaire, which was carried out along lines similar to those followed by Macht & Lawrence, has also reported a slight and non-significant increase in foetal mortality among the progeny of irradiated fathers.

All in all, although the published data agree fairly well on this point, they cannot strictly be regarded as conclusive.

(*c*) *Variations in the sex ratio*

Of the publications already mentioned, only that of Neel et al.[7] supplies any usable material. For example, neither Murphy & Goldstein,[5] nor Maurer[6] nor Crow[9] indicate the sex of the children; and although Macht & Lawrence[8] give some figures, they do not specify the sex of about 10% of the children, so that one can hardly rely on their statistics.

In their preliminary report, Neel et al.[7] showed that among the offspring of the survivors in Nagasaki there was a statistically significant deviation of the sex ratio, an increase being observed among the children of irradiated fathers and a decrease among those of irradiated mothers. On the other hand, such variations were small or non-existent in the more numerous offspring of the Hiroshima survivors.

At the First International Congress of Human Genetics, held in Copenhagen in August 1956, Dr. J. V. Neel presented some further statistics on the subjects mentioned above, including all the births which had occurred in these families since 1953. In this larger sample, the deviations observed in 1953 are no longer discernible.

In Paris, a survey has been carried out on the offspring of subjects given pelvic radiotherapy in all the hospitals in the city and the surrounding districts (Turpin, Lejeune & Rethore).[3] The initial findings, which are concerned exclusively with the sex ratio, were presented by Professor Turpin at the First International Congress of Human Genetics; they are briefly summarized in the table below.

Offspring of various subjects before and after pelvic radiotherapy

Subjects and reason for treatment	Number of children		Sex ratio
	♂	♀	
Before treatment:			
Men (138); various reasons	116	115	0.502±0.034
Men (284); sciatica	242	223	0.520±0.024
Women (154)	131	106	0.553±0.034
After treatment:			
Men (95); various reasons ($\bar{r}$*=1461 r)	68	62	0.523±0.048
Men (194); sciatica ($\bar{r}$=1295 r)	157	118	0.571±0.030
Women (97); ($\bar{r}$=1360 r)	63	73	0.463±0.044

*$\bar{r}$=average skin dose, *not* gonad dose.

The figures given in the table show that, before treatment, the sex ratio of the children was statistically comparable in the two groups, i. e., the male and female subjects. After treatment of one of the parents, however, the sex ratio increased in the offspring of the treated fathers and decreased in those of the treated mothers, this heterogeneity being statistically significant.

Conclusions

In concluding this very rapid review of the few usable data at present available, the writer would like to stress the following two points:

1. The gonad dose per 30 years, in the form in which it has already been established.[10 11] probably gives an acceptable approximation of the risk resulting from artificial ionizing radiation. Nevertheless, an accurate evaluation can be arrived at only by systematic recording of all individual exposures. Moreover,

[9] Crow, J. F. (1955), Amer. J. Roentgenol. 73, 467.

[10] Great Britain, Medical Research Council (1956), The hazards to man of nuclear and allied radiations, London.

[11] United States of America, National Academy of Sciences (1956), Biological effects of atomic radiation.

it is essential that the gonad dose actually received during irradiation under the conditions obtaining in practice should be checked experimentally. The first—and perhaps the most valuable—result of such investigations would probably be a substantial decrease in the degree of exposure of the gonads.

2. From an analysis of the observations already made, it appears that with the doses used in radiotherapy, it would probably be possible to detect some effect in the first generation. The urgency of research in this connection need hardly be stressed. It is only when a list of the mutations which are at present detectable has been drawn up that it will be possible to extrapolate and obtain an estimate of the over-all genetic damage.

Annex 6

Gonad Doses From Diagnostic and Therapeutic Radiology [1]

DIAGNOSTIC RADIOLOGY

Several estimates have been made recently of the gonad dose to the population in excess of that from natural background radiation, resulting from diagnostic radiology. These range in value from approximately 10% of the background radiation (Martin [2]) to approximately 58% (Clark [3]). Intermediate between these two is the estimate of Osborn & Smith [4] of at least 22%. There appears to be some agreement, on present knowledge, that the "doubling dose" for many may lie between 30 and 80 r in a period of 30 years. The concept of the "doubling dose" is, however, an over-simplification of the situation, as there most likely exists a spectrum of gene sensitivity. It may well be that already we are in the position that the mutation rate for some genes is significantly raised.

The most comprehensive analysis yet made of the gonad dose from diagnostic radiology is that of Osborn & Smith.[4] These authors make a number of points of considerable importance. First, they draw attention to the rapid expansion in the use of diagnostic X-ray procedures, adducing evidence that in England and Wales the number of X-ray examinations may, at the present time, be increasing by about 12% per year, and that in 1954 between 17 and 18 million examinations were made. They point out, however, that the adverse effect of this expansion, so far as the gonad dose is concerned, is offset to some extent by technical advances which have reduced the amount of necessary radiation exposure. Secondly, they draw attention to the important fact that only a small number of examinations, amounting to about 7% of the total, contribute the major portion (about 75%) of the gonad dose. These examinations are those of the hips, the lumbo-sacral spine, the pelvis, the urinary tract (intravenous and retrograde pyelography) and pelvimetry. By and large these findings are comparable to those of Martin.[2]

Of serious import is the widespread use of pelvimetry and other X-ray obstetrical examinations of the abdomen, not to speak of examination of the maternal abdomen for other than obstetrical purposes. According to Osborn & Smith, *at least* 26,000 pelvimetries are carried out annually in England and Wales and 86,000 other obstetrical X-ray examinations. These authors calculate that the maternal gonad dose from pelvimetry alone amounts to 3% of the gonad dose to the total population of both sexes, and the foetal dose to as much as 15.6%.

The real criticism of the work of Osborn & Smith lies in the fact that their results are based on a sample of only five hospitals—two teaching and two non-teaching hospitals and one children's hospital. It is doubtful whether this is an adequately representative sample of the hospitals of England and Wales, particularly as there is some evidence, especially from a study of pelvimetry, of considerable variations in radiographic techniques.

THERAPEUTIC RADIOLOGY

The great majority of patients treated in radiotherapy centres are suffering from malignant disease and are either actually or effectively past the reproduc-

[1] Submitted by Dr. W. M. Court Brown, Director, Group for Research into the General Effects of Radiation (Medical Research Council of Great Britain), Radiotherapy Department, Western General Hospital, Edinburgh, Scotland.
[2] Martin, J. H. (1955), Med. J. Aust. 2, 806.
[3] Clark, S. H. (1956), Bull. atom. Scient. 12, 14.
[4] Osborn, S. B. & Smith, E. E. (1956), Lancet, 1, 949.

tive age. However, a proportion of young persons are treated for a variety of nonmalignant conditions. These conditions are grouped into those treated during childhood and those treated during early adult life. In the former group are haemangiomata, keloids, hypertrophic tonsillar tissue, bone cysts, etc. The conditions treated during early adult life are mainly ankylosing spondylitis, skin diseases, keloids, some menstrual disturbances and, occasionally, bone cysts.

The childhood conditions chiefly occur on the upper half of the trunk and on the limbs, and, on the whole, are treated with low-voltage radiation given on small localized fields. It is unlikely that these treatments contribute to the gonad dose to any great extent.

Of the conditions irradiated in early adult life, so far as experience in Great Britain is concerned, the treatment of ankylosing spondylitis is likely to contribute an appreciable fraction to the gonad dose. Some tentative estimates which have been made of the size of this fraction are given below. No information is available on the contribution from the treatment of skin diseases, but there is every reason for believing that it may also be appreciable.

ANKYLOSING SPONDYLITIS

During the past year an epidemiological survey has been carried out to determine the incidence of leukaemia among patients treated with X-rays for ankylosing spondylitis. This survey covered all the radiotherapy departments of England and Wales, and of Scotland, at present operating under the respective National Health Services. In the course of the survey, data were recorded concerning 13,352 patients, who were treated between the years 1935 and 1954, inclusive—presumably the majority, if not the great majority, of the patients treated for this disease in Great Britain during the period in question.

It is possible to make some rough and very preliminary estimates of the minimum contribution to the gonad dose as the result of the treatment of ankylosing spondylitis in Great Britain, on the assumption that the testes are not shielded during treatment.

The beneficial effects of X-ray treatment for this disease were only widely recognized during the Second World War, and there was a steady annual increase in the number of new patients irradiated up to 1950, since when there has been a tendency for the number to diminish slightly. For the period 1949 to 1954 the average number of new cases per year was 1336, of which 1109 (83%) were males. Of these 53% were under the age of 35. Of the females, 43% were less than 35 years of age.

The average standard first course of treatment for a male patient has been taken as follows (based on dosage information from a random sample of approximately one in six of the whole population):

(a) A single 15 cm x 10 cm posterior field centred over the sacro-iliac joints, the large axis of the field being horizontal.

(b) A series of fields irradiating the whole length of the spine and extending from the upper margin of the sacro-iliac field to the upper part of the neck. On an average, the breadth of these fields is 7.5 cm.

(c) A total skin dose to each field of 1500 r (half value layer 1.6 mm).

From measurements made in a phantom man it appears that a dose of about 45 r is received in the male gonads during such a course of treatment.

Measurements have not been made of the gonad dose received by females. In some centres the female sacro-iliac area was treated similarly to the male area; but in many others definite attempts were made to avoid the ovaries. The direct irradiation of the sacro-iliac region to 1500 r is almost certain to induce permanent sterility. For present purposes it is assumed that the ovarian dose received is on an average 45 r.

The contributions to the gonad doses every year from the treatment of ankylosing spondylitis have been calculated (see table below) on the basis of the following *de facto* populations for England, Wales and Scotland (1952):

	15-34 years	All ages
Males	6.6×10^6	2.37×10^7
Females	6.8×10^6	2.56×10^7
Total	1.34×10^7	4.93×10^7

Gonad dose (in milliroentgens) per head of population per year in England, Wales, and Scotland

	15-34 years	All ages
Males	4.0	2.1
Females	0.6	0.4
Both sexes	2.3	1.2

These estimates can be considered to be minimal ones. No less than 46% of the males and 44% of the females were given more than one course of treatment. Many of these additional courses were given to the lumbo-sacral region and to the hip joints. However, two other factors which tend to diminish either the size or the effectiveness of the gonad dose must be taken into consideration. First in some radiotherapy centres it was standard practice to provide some form of lead shielding for the testes; this was by no means a universal habit, however, and a number of instances of male infertility, some of which could have been the result of X-ray exposure, were discovered. Secondly, it may well be that sufferers from spondylitis are sub-fertile. The disease is a crippling one and is frequently accompanied by pulmonary tuberculosis and ulcerative colitis. There is, however, no published evidence on this point.

To sum up, the radiotherapy of patients suffering from ankylosing spondylitis in Great Britain will give a gonad dose per year of at least 1% of the natural background, and possibly appreciably higher. A more rigorous examination of the contribution from this source, and an analysis of the contribution from other types of radiotherapy, including the use of radioactive isotopes, could well bring the total contribution from radiotherapy up to the level of about 8% suggested by Clark [3] for the USA, or perhaps to an even higher level.

DISCUSSION

So far as the writer is aware there is no direct evidence of a steady upward trend in the incidence of any of the undesirable traits that might be expected with any increase in the mutation rate due to the steady expansion of medical radiology. The direct epidemiological approach to this problem is clearly beset with difficulties, not the least being the very large population that would have to be kept under observation and the inaccuracy and inadequacy of death certification.

There is, however, some evidence from which it could be argued indirectly that an increase in undesirable traits may already be taking place. For example, recent work has demonstrated a significant increase in the mortality from leukaemia among persons treated with X-rays for ankylosing spondylitis, and it has also been possible to demonstrate the existence of a relationship between the annual incidence of leukaemia in these patients and the radiation dose to the bone-marrow. Some preliminary data have been published (Court Brown & Doll [5]). On the evidence as it stands it seems possible that radiation leukaemogenesis in man is a non-threshold effect, and that over the range of dose met with in ordinary civilian life the dose-response relationship is a simple proportional one, analogous to that for the induction of mutations. If this were finally shown to be the case, two deductions would become valid. First, that a proportion of naturally occurring cases of leukaemia are probably due to natural background radiation; and, secondly, that any increase in the background radiation from artificial sources will be associated with an increased mortality from leukaemia.

The mortality from leukaemia is known to be rising. Thus, the annual crude death-rate for both sexes rose in England and Wales from 26 in 1940 to 49 in 1954. The corresponding figures for Denmark are 48 and 71, for Canada 30 and 51, and for the USA 39 and 63 (in 1953). Undoubtedly part of this increase is due to changes in diagnostic criteria and improvements in diagnostic techniques. There is, however, a general feeling that it may in part be real

[5] Court Brown, W. M. & Doll, R. (1956), Appendix B: Leukaemia and aplastic anaemia in patients treated with X-rays for ankylosing spondylitis. In: Great Britain, Medical Research Council, The hazards to man of nuclear and allied radiations, London, p. 87.

and absolute. If this be the case, and if the dose-response relationship over the relevant range of dose be linear, then part of the increased mortality from leukaemia may well be due to the expanding use of radiations, particularly diagnostic radiology. If the incidents of such traits as haemophilia, muscular dystrophy, and achondroplasia could be examined in the same way, it is more than probable that at least a qualitatively similar upward trend would be found.

Annex 7

Mutation in Man [1]

1. INTRODUCTION

The study of gene mutation in man has two aspects. The first concerns the ascertainment of spontaneous mutation rates at specified loci. This gives information about human evolution in general as well as about the causation of certain rare diseases and defects. The second aspect, which has only recently become significant, concerns artificially produced mutagens and, in particular, the genetical effects of ionizing radiation. In order to estimate the magnitude of these effects a knowledge of spontaneous mutation rates at given loci is required and the sensitivity of these loci to radiation needs to be ascertained.

2. MEASUREMENT OF SPONTANEOUS MUTATION RATE

Estimation of mutation rate in man, in relation to any given hereditary trait, depends upon ascertaining three things, the incidence of the trait in the general population, the nature of the genetical contribution to the cause of the trait, and the fitness of the genotypes concerned. These phenomena are not necessarily constant. As seen in the population at the present time they may not represent the true picture over a long series of generations, during which natural selection has been acting. They only give us the first clue to conditions which govern genic equilibrium in human populations.

There are two standard methods of approach, the direct and the indirect.

(*i*) *Direct observation*

The most favorable case for estimating mutation rate directly occurs when the gene studied is detectable with certainty or regularity in heterozygotes. Instances of fresh mutation can then be observed in families where a gene appears in an offspring although it was not present in the parents. The ideal kind of regular dominance required for this is rarely (perhaps never) found in human genetics. Man is a wild species, under natural selection, unlike laboratory stocks, and consequently most single gene effects, especially those shown in heterozygotes, are subject to modification. Even with the most reliable characters, such as blood group antigens, suppression is possible by gene interaction (Levine et al. 1955); such events could easily be misinterpreted as evidence of mutation by the unwary.

The situation for sex-linked genes is quite favourable, theoretically, for direct observation of fresh mutation because modification of a character shown in homozygous males is usually slight. Occasional families will be observed in which the probability is very great that the disease in the propositus is due to fresh mutation. The proportion of sporadic cases can also be inferred if the sibships show an excess of sporadic propositi.

For recessive traits the problem is much more difficult because heterozygous carriers are not detectable in ordinary circumstances. In cases where special techniques have been developed for identification of carriers the problem is resolved into one of detection of mutation for a dominant condition, as demonstrated by Vanderpitts et al. (1955) for sickle cell trait. Direct observation of cases of recessive diseases due to fresh mutation is very unlikely to be possible because only a very small proportion of cases of a recessive trait in a given generation can be attributed to fresh mutation in a parent. For diseases in which a single gene is only a part cause and in which environment has a great effect upon manifestation, the contribution of spontaneous mutation is likely to be even less significant. The same applies to conditions due to the interaction of many genes. For none of them can mutation rates be directly determined.

---

[1] Submitted by Dr. L. S. Penrose, Galton Professor of Eugenics, University College, London, England.

(*ii*) *The indirect argument*

When the total effects of a gene are very disadvantageous an indirect line of argument can be used for estimating mutation rate, even though the gene may not be manifest in the heterozygous state. Principles on which the indirect estimation of mutation rates can be based were laid down by Haldane (1932). The assumption can be made that the human population is in a state approaching genetical equilibrium under natural selection. It is supposed that disadvantageous genes could not persist in the population unless their extinction by selective mortality were completely balanced by the resurrence of mutation.

In the case of dominant or sex-linked characters associated with very high mortality, the direct measurement of mutation rate can be supplemented, and its plausibility greatly strengthened, by the indirect argument. The best situation for this combination occurs in the case of a very deleterious dominant trait. This is a rare circumstance. If the disease is not very lethal, there will be difficulty in measuring the unfitness conferred by the gene: if it is very lethal, there is difficulty in providing the dominant mode of inheritance, as it will seldom last even for two generations. Sometimes the problem might be solved for a locus, which had several different known alleles, some producing milder and others severer types of disease. Then, in each of the severest cases, mutation of a lethal allele will be observable. This possibly occurs in both epiloia and chondrodystrophy. For mild alleles, which last for several generations, the proportion of cases due to fresh mutation is correspondingly smaller.

Estimates which are entirely indirect, are untrustworthy but they have actually been made for a variety of genes recognized only by their recessive effects. One cause of uncertainty with recessive traits is that allowance has to be made for the results of inbreeding. Another likely source of error is that genetical equilibrium can be maintained not only by mutation but also by slightly advantageous effects in heterozygotes. That is to say, on the balance, the total effect of a gene may be much less bad than appears from studying abnormal homozygotes and then an indirect estimate of mutation rate will give much too high a value.

8. SOME STANDARD ESTIMATES OF HUMAN MUTATION RATES

Mutation rates have been calculated for quite a large number of genes in man. It is preferable to express them in terms of loci per generation, if we wish to avoid controversy, because slightly different forms of the diseases concerned can be accounted for by the same allele or by different alleles. If there are several very closely linked loci giving rise to a pseudoallelic system, the real mutation rate for each separate element is lowered by a factor depending upon the number of elements in the complex.

(*i*) *Dominants*

The most exact estimates for supposedly single loci are probably those for very deleterious dominant traits (see Table 1). Allowing for the probability that more than one disease entity may be classified under each heading, they are maximal values. The average value for six conditions is about 14×10^{-6}.

Owing to the classification of more than one type of chondrodystrophy under the same heading the rate given is likely to be considerably too high. According to Grebe (1955) there are several clinical types; and some cases may be due to recessive genes. Furthermore, these different types may have different mutation rates.

Another, dominant, condition, which apparently has a relatively high mutation rate, namely, retinoblastoma, occurs perhaps not infrequently as a phenocopy (Vogel 1954) not transmissible to the next generation. The same idea could be applied also to other conditions listed in Table 1, such as microphthalmos.

The indirect argument, which supports all these estimations, can only be used when there is a strong selection against the gene studied. Theoretically it should be possible to obtain mutation figures for several blood antigens, e. g. *ABO* or *MNS*, but selection against any of these genes is too slight and indefinite to be used as indirect support for the mutation hypothesis. On the other hand, the indirect argument can be extended to cover certain cases in which the combination of several genes at different loci is lethal or very deleterious. Thus a lethal condition, caused by the simultaneous presence of two heterozygous genes, will imply that each of the genes concerned mutates frequently enough

to make good the loss occasioned when it occurs in conjunction with the other.

Taking all these considerations together we can reasonably assume that the mutation rates for loci giving rise to dominant genes, though somewhat too high, are of the right order of magnitude. It seems that, for most of these dominant diseases, the rate should be considered to be about 5×10^{-6}.

(ii) Sex-linked loci

The prevalence in man of sex-linked diseases which are very lethal is difficult to explain except on a mutation hypothesis. Direct evidence based upon observed low incidence of haemophilia in sibships and in maternal collateral relative also supports this explanation. The matter has been repeatedly investigated by Haldane (1946, 1955) and there seems to be some evidence that mutation more commonly occurs in males than in females. The two sex-linked diseases which have given information about human mutation rate are haemophilia and pseudohypertrophic muscular dystrophy. In both cases there are many types of illness easily confused with one another clinically. Sex-linked types are identified by pedigree studies and by their occurrence in males only but, by this process, some autosomal cases may occasionally be incorrectly included. A characteristic difficulty is the exclusion of autosomal sex-limited conditions.

In the standard examples of haemophilia and sex-linked muscular dystrophy, mutation rates have been estimated several times but always on the assumption that, in each disease, there is only one locus involved. These rates, as shown in Table 1, are considerably higher than the direct estimates for autosomal dominants. Perhaps the X-chromosome is peculiar in that it has many complex loci or distinct loci with similar effects.

(iii) Recessive traits

A recessive trait in man can be defined as one which depends upon a gene in homozygous form. There may be mild manifestations detectable in heterozygotes (e. g. thalassaemia, galactosaemia, cystinuria) but the disease in the homozygote is the effect with which we are concerned. The indirect estimates of mutation rates for recessive diseases, shown in Table 2, assume that the heterozygote is neutral in its effect upon fitness. If the heterozygotes were deleterious, as suggested by Böök (1953) for schizophrenia, the values would have to be increased. Conversely if heterozygotes were slightly favourable, the values would have to be reduced.

TABLE 1.—*Estimates of spontaneous mutation rates of some human genes: (a) Dominant inheritance; (b) sex-linked inheritance*

Trait	Mutation rate per million loci per generation	Region	Source [1]	Date
Epiloia (a)	8	England	Gunther & Penrose	1935
Chondrodystrophy (a)	45	Denmark	Mørch	1941
Do	70	Sweden	Böök	1952
Aniridia (a)	[2] 5	Denmark	Møllenback	1947
Microphthalmos without mental defect (a)	5	Sweden	Sjögren & Larsson	1949
Retinoblastoma (a)	15	England	Philip & Sorsby	1947
Do	23	USA	Neel & Falls	1951
Do	4	Germany	Vogel	1954
Partial albinism & deafness (a)	4	Holland	Waardenburg	1951
Haemophilia (b)	20	England	Haldane	1953
Do	32	Denmark	Andreassen	1943
Do	27	Switzerland and Denmark.	Vogel	1955
Pseudohypertrophic muscular dystrophy (b).	95	USA	Stephens & Tyler	1951
Do	45	Northern Ireland	Stevenson	1953
Do	43	England	Walton	1955

[1] See Penrose (1956a).
[2] This estimate differs by a factor of 2 from that given by the author but it is based on his material.

TABLE 2.—*Indirect estimates of spontaneous mutation rates on the assumption of recessive inheritance*

Trait	Mutation rate per million loci per generation	Region	Source	Date
Juvenile amaurotic idiocy	38	Sweden	Haldane	1939
Albinism	28	Japan	Neel et al.[1]	1949
Icthyosis congenita	11	Japan	Neel et al.[1]	1949
Total colour blindness	28	Japan	Neel et al.[1]	1949
Infantile amaurotic idiocy	11	Japan	Neel et al.[1]	1949
Amyotonia congenita	20	Sweden	Böök [1]	1952
Epidermolysis bullosa	50	Sweden	Böök [1]	1952
Cystic fibrosis of pancreas	700	USA	Goodman & Read	1951
Sickle cell anaemia	10,000	USA	Neel	1951
Thalassaemia	400	USA	Neel	1951
Spastic diplegia	2,000	Sweden	Böök	1953
Microcephaly	49	Japan	Komai et al.[1]	1955
Phenylketonuria	25	England	Penrose [1]	1956
Schizophrenia	500	England	Penrose [1]	1956

[1] See Penrose (1956a).

A very slight amount of heterozygous advantage is sufficient to keep a rare recessive lethal in stable genic equilibrium in the absence of mutation so that the calculation of mutation rate is very easily invalidated. This is an extremely important principle and is worthy of detailed consideration.

Most well known recessive traits cannot easily be supposed to have arrived at their existing levels of gene frequency (e. g. 1/100 for phenylketonuria) by chance or by "drift". The situation for commoner genes is even more striking. For tnalassaemia and sickle cell trait (Neel 1951), cystic fibrosis (Goodman & Reed 1952), spastic diplegia (Böök 1953) and schizophrenia (Penrose 1956a) improbably high mutation rates have to be postulated. Indeed the maximum rate for sickle cell trait, derived from direct observation on heterozygotes, is much lower than that calculated indirectly (Vanderpitte et al. 1955). These common traits could not have easily established themselves unless the heterozygotes had some advantage. The advantages may have been local ones in the distant past, for example, ability to withstand infections, plagues, famines, abnormal climates and so on.

It is not necessary to postulate any virtue in the heterozygote as such. It could be sufficient if the mutant alleles were favourable at one epoch and unfavourable at another epoch, in different circumstances or at different stages of the same life cycle. The principle of genetical stability produced by heterozygous advantage or, more accurately, homozygous disadvantage, is one which has been understood for a long time (Fisher 1930) but only recently taken seriously. In human genetics it is exhibited by such a system as may be present in relation to the sickle cell trait in Africans. The disadvantage of one homozygote, SS, which suffers from anaemia, is balanced to some extent by disadvantage of the homozygote, *AA*, which is especially susceptible to malaria caused by *P. Falciparum.* Balanced human genetical systems are shown in metrical traits because the extreme types, which tend to be homozygous, are relatively unfavourable. Examples are stature, birth weight and intelligence level. For intelligence, in particular, there is a marked fertility differential in one direction and a viability differential in the other. That is, low intelligence levels are associated with low viability and high levels with low fertility.

In all such cases of balanced polymorphism the variation, which is apparently, reduced each generation by loss of extreme types, is not maintained by fresh mutation. It is maintained simply because the heterozygotes, who tend to have medium metrical value, are the parents of most children in each successive generation. In these circumstances it is quite useless to attempt to estimate the mutation rates of component genes: any indirect estimate will be far too high.

It has been suggested by Haldane (1939) that the converse may be true, namely that mutation rate estimates for recessive traits are often too low. The argument used is that the true incidence, which recurrent mutation would theoretically balance, has in the past been much greater than it is at the present time. This is likely because inbreeding, which facilitates the appearance of recessive

diseases, has been gradually diminishing for many decades in all civilized communities. I believe this argument to be unsound because the incidence of rare recessive traits in man is extremely irregularly distributed. Tay-Sachs disease is almost confined to Jewish communities as also is pentosuria, Cooley's disease has centre in the Po delta. Phenylketonuria, on the other hand, does not occur among Jews. Sickle cell anaemia is common in Africans. Juvenile amauratic idiocy is commonest in Sweden and acatalazaemia has only been found in Japan. These facts suggest that recessive mutations are very rare but that occasionally they have spread for unknown reasons probably connected with heterozygous advantage at one epoch or another. If mutations were not very rare the same set of recessive diseases would appear in all communities or at least in all inbred communities throughout the world.

To sum up the discussion on spontaneous mutation rate, my view is that, for a variety of reasons, most mutation rates already calculated are too high; points to be stressed are, first, that mutation may be mimicked by suppression of even the most regular kinds of dominant inheritance, secondly that different conditions are grouped under single clinical headings, and, thirdly, that heterozygotes of established recessive lethal traits are likely to have carried slight advantages in the past even if they do not at the present time.

4. EFFECT OF INDUCED MUTATIONS

The immediate effect of an increase above spontaneous mutation rate is most easily calculated when the gene is dominant. The rule, however, is quite general. The increase of incidence of any trait in the first generation, due to induced mutation, depends upon the proportion of cases due to fresh mutation in ordinary circumstances. For lethal dominants and sex-linked traits this proportion is large but in lethal recessives it is very small. It is also small for dominants which are very imperfectly manifested as with those contributing to multifactorial traits. The rule refers to the effect in the first generation or in closely succeeding generations, which especially interest people now living. The total quantitative effect on the population of altered mutation rate is theoretically the same whatever the manner of inheritance but, in the case of recessive or heavily modified dominants, a slight effect is maintained over such an enormous length of time, many thousands of years.

The proportion of cases of a lethal condition due to fresh mutation in any given generation can be estimated on the basis of the indirect argument. If the mutation rate, μ, is expressed as a function of the gene frequency thus,

$$\mu = f(q)$$

it follows formally that the proportion of cases in any given generation due to fresh mutation, a quantity which can be called M, is given by the approximation,

$$M = d\mu/dq.$$

For example, for a recessive lethal trait,

$$\mu = q^2, \text{ and so } M = 2q.$$

Substituting 1/40 000 for q^2, the frequency of juvenile amaurotic idiocy as estimated by Sjörgren (1931), we get M=1/100. In view of what has been said about the use of the indirect method this is probably an upper limit but it shows how little effect a change in spontaneous mutation rate would have upon the incidence in the next generation after it had occurred or, indeed, in any subsequent generation. Doubling the mutation rate would only increase the incidence by one or two per cent. in the first subsequent generation.

5. SENSITIVITY OF HUMAN LOCI TO RADIATION

Much has been written about the probable sensitivity of human loci to radiation using experimental data on lower animals as a basis for comparison. Direct observations on man, however, are essential and three sources of information are at present available.

(*i*) *The comparison of offspring of selected parents exposed to different quantities of radiation*

This is the method attempted in several comparative studies. Children of radiologists have been examined by Crow (1955) and also by Macht & Lawrence

(1955) and the exposed Japanese population by Neel and his colleagues (Neel & Schull 1954). A development of the same idea is implied in two other proposed types of investigation. One of these is the special examination of children of patients who have received large therapeutic doses of radiation before conception, as may be the case in sufferers from spondylitis. The other suggested method is to examine the incidence of mutations in areas where the natural background radiation is high. Each of these methods, though theoretically possible, has its own special technical difficulties. There is a general objection to all of them, however. Fresh mutation is a phenomenon which can only very rarely be observed even though it may be occurring all the time. To search for slight increases in incidence of traits which, in the case of known recessives, will not exceed one per cent. requires the collection of enormous quantities of data and results are likely to be inconclusive. These methods are, in fact, rather inefficient even after allowances have been made for sources of error peculiar to each type of enquiry.

(ii) The examination of parental history in known instances of mutation

An alternative and more efficient method, which has received scant attention hitherto, is the careful examination of the personal histories of parents and, in certain instances, of grandparents, for groups of cases where fresh mutation is suspected of having played a part in causing disease in the offspring. This method has already produced valuable results by using the simple test of parental age.

Clearly, the older the parent the more likely he is to have been subjected to mutagenic influences. If the influence is background radiation, at the age of 40 the dose will have been twice that received at the age of 20. The net effect on parental age distribution of diseases in the offspring caused by background radiation alone, though definite, would be slight. The expected average increase would be scarcely more than one year above normal parental age (Penrose 1955a). Marked effects confined to one or other parent have, however, been observed in several malformations. Marked increase of father's age has been found in chondrodystrophy and acrocephalosyndactyly, as shown in Table 3. On the other hand, the incidence of mongolism is associated solely with advancing age of the mother. It would appear, thus, that, in so far as these traits may have their origin in fresh mutations, the causes must be different. In particular, a marked increase in paternal age strongly suggests some process connected with cell division in the spermatogonial stage which might be chemical in origin. The effect does not appear in other traits thought to be often caused by fresh mutation, such as epiloia, neurofibromatosis and retinoblastoma, where only slight and statistically insignificant parental age increases have been registered. Mongolism would, by the same test, appear to have an entirely different cause.

TABLE 3.—*Mean parental ages in sporadic cases of diseases attributed to fresh mutation compared with control mean ages*

Disease	Source [1]	Control mean [2]	Number of cases		Excess over control mean (years)			
					Father's mean age		Mother's mean age	
Chondrodystrophy	Mørch (1942)	D	97	176	+5.4	+5.1	+3.5	+3.6
	Krooth (1952)	E	16		+6.8		+5.7	
	Grebe (1955)	G	63		+4.3		+3.1	
Acrocephalosyndactyly	Grebe (1944)	G	7		+5.5		+3.5	
Epiloia	Gunther & Penrose (1935)	E	12	33	+0.8	+0.5	+0.3	+0.4
	Borberg (1951)	D	21		+0.4		+0.5	
Neurofibromatosis	Borberg (1951)	D	49		+0.9		+0.8	
Retinoblastoma	Neel & Falls (1954)	M	64		+0.5		+0.7	
Mongolism	Schulz (1931)	G	80	664	+5.3	+5.8	+7.7	+6.9
	Øster (1953)	D	369		+5.3		+6.5	
	Penrose (1955)	E	215		+6.8		+7.4	

[1] See Penrose (1956b).
[2] E, England: father 30.9; mother 28.6. D, Denmark: father 33.3; mother 28.6. G, Germany: father 32.6; mother 28.9. M, Michigan, USA: father 30.5; mother 26.4.

The investigation of parental age is only one part of the problem. The history of parental exposure to X-rays and other kinds of radiation needs to be recorded;

occupational risks and possible exposure to chemical mutagens from external sources could also be made the subject of enquiry.

(*iii*) *Observations on somatic cells*

It has been suggested that a tissue culture treated by exposure to a known dose of radiation could serve to investigate the sensitivity of human cells. Techniques of this purpose will no doubt be developed in time though such experiments may never be critical because germ cells could have different sensitivity from that of somatic cells. This objection may be for the moment left on one side, however, while we search for existing data which might give clues to mutation rate in somatic cells. The obvious source of information is observations concerning inductions of tumours by radiation.

It has until recently usually been assumed that very small amounts of ionizing radiation have no effect on the induction of leukaemia. This is now doubted and the relation between bone marrow dose and incidence of leukaemia is thought to be not unlike the linear effect observed in the induction of X-chromosome lethals in *Drosophila*. Some idea of the dosage to bone marrow required to double the spontaneous leukaemia rate can be obtained from unpublished figures (Court, Brown & Doll 1956) and it is in the region of 30 to 50 roentgen units.

This line of thought leads to another interesting idea. The suggestion has been made that many sporadic cases of retinoblastoma arise as phenocopies. Is it not possible that these phenocopies are simply somatic mutations of the same gene which sometimes is carried in the germ track causing a dominant type of inheritance?

6. THE "LOAD" OF ABNORMAL GENES IN MAN

Finally, I would like to mention one or two points about the total effect of mutation on man since this has been so much discussed recently. Consider the total number of zygotes formed in a generation. We have no idea how many fail to pass through the first few divisions and never develop into embryos. Indeed it is impossible to estimate how many embryos are lost in the first six weeks after fertilization. According to Yerushalmy (1945) 15 per cent. of human pregnancies are known to terminate in miscarriages or abortions. Beyond this, three per cent. are stillborn and two per cent. are neonatal deaths. In addition, early mortality after the first month amounts to three per cent. These are figures for European and North American communities, where infectious diseases and malnutrition are under efficient control. In many parts of the world they would be gross underestimates. Among those who survive to adult status, 20 per cent. are unmarried and of those who do marry some 10 per cent. are infertile. How much of this continuous loss of zygotes, which may amount to about 50 per cent., is genetic is not known; by analogy with results obtained on ordinary metrical traits such as stature and intelligence, about half of this loss of zygotes might me directly hereditary. Perhaps the main factors are recessive lethals. If this were so, the indirect argument would lead to the conclusion that about a quarter of zygotes are lost each generation and that the genes which are thereby eliminated are replaced by fresh mutations. This points to the further conclusion, that a large increase in mutation rate, say permanent doubling, would eventually increase this lethal lead to a half and would greatly reduce human fitness, though the immediate effects would be small. However, for reasons given earlier, I do not suppose this picture to be an accurate one. Much of the permanent lethality which we experience is likely to be due to balanced genetical mechanism which do not require the assumption of appreciable amounts of mutation to maintain them. As I have previously pointed out (Penrose 1955b) improved living conditions are likely to reduce the frequencies of recessive genes whose prevalence is due to heterozygous advantage. Thus genetic damage which may be done by increase of mutation rate, due to industrial and medical uses of radiation, may be offset in the future by the improvements in hygiene which are taking place at the present time all over the world.

REFERENCES

Böök, J. A. (1953) A genetic and neuropsychiatric investigation of a North-Swedish population with special regard to schizophrenia and mental deficiency. *Acta genet.* *4*, 1.

Court Brown, W. M. & Doll, R. (1956) Leukaemia and aplastic anaemia in patients treated with X-rays for ankylosing spondylitis. *Hazards to man of nuclear and allied radiations*, Appendix B. London: HMSO.

Crow, J. F. (1955) A comparison of foetal and infant death rates in the progeny of radiologists and pathologists. *Amer. J. Roentgenol. 73*, 467.

Fisher, R. A. (1930) *The genetical theory of natural selection.* Oxford: Clarendon Press.

Goodman, H. O. & Reed, S. C. (1952) Heredity of fibrosis of the pancreas, possible mutation rate of the gene. *Amer. J. hum. Genet. 4*, 59.

Grebe, H. (1955) *Chondrodysplasie.* Roma: Ed. dell'Istituto Gregorio Mendel.

Haldane, J. B. S. (1932) *The causes of evolution.* London: Longmans Green & Co.

Haldane, J. B. S. (1939) The spread of harmful autosomal recessive genes in human populations. *Ann. Eugen. (Camb.)*, *2*, 232.

Haldane, J. B. S. (1946) Mutation rate of the gene for haemophilia and its segregation ratios in males and females. *Ann. Eugen. (Camb.)*, *13*, 262.

Haldane, J. B. S. (1955) Mutation in the sex-linked recessive type of muscular dystrophy. A possible sex difference. *Ann. hum. Genet. (Lond.)*, *20*, 344.

Levine, P., Robinson, E., Celand, M., Briggs, O. & Falkinburg, L. (1955) Gene interaction resulting in suppression of blood group substance B. *Blood, 10*, 1100.

Macht, S. H. & Lawrence, P. S. (1955) National survey of congenital malformations resulting from exposure to roentgen radiation. *Amer. J. Roentgenol. 73*, 442.

Neel, J. V. (1951) The population genetics of two inherited blood dyscrasias in man. *Cold Spr. Harb. Symp. quant. Biol. 15*, 141.

Neel, J. V. & Schull, W. J. (1954) *Human heredity.* Chicago: University of Chicago Press.

Penrose, L. S. (1955a) Parental age and mutation. *Lancet, 2*, 312.

Penrose, L. S. (1955b) Genetics and medicine. *Advanc. Sci. 11*, 387.

Penrose, L. S. (1956a) The spontaneous mutation rate in man. *Hazards to man of nuclear and allied radiations*, Appendices C, D and E. London: HMSO.

Penrose, L. S. (1956b) The mutational origin of hereditary eye diseases. *Franceschetti Festschrift* (in the press).

Sjögren, T. (1931) *Die juvenile amaurotische Idiotie.* Lond: Berlingska Boktryckeriet.

Vanderpitte, J. M., Zuelzer, W. W., Neel, J. V. & Colaert, J. (1955) Evidence concerning the inadequacy of mutation as an explanation of the frequency of the sickle cell gene in the Belgian Congo. *Blood, 10*, 341.

Vogel, F. (1954) Ueber Genetik und Mutationsrate des Retinoblastoms (Glioma retinae) *Z. Konstlehre, 32*, 308.

Yerushalmy, J. (1945) Infant and maternal mortality in the modern world. *Ann. Amer. Acad. pol. Soc. Sci. 237*, 134.

Annex 8

Possible Areas With Sufficiently Different Background-Radiation Levels To Permit Detection of Differences in Mutation Rates of "Marker" Genes [1]

The impressive developments in atomic research in recent years and the increasing application of nuclear energy in many fields of human endeavour have brought to the forefront serious problems concerning the long-term effects of radiation on man and his environment. In order to obtain a better appreciation of the totality of effects on human populations, it is necessary first to take into account the magnitude of the various components of natural background radiation as well as the radioactive elements normally present in the body and environment. The magnitude of the contributions of cosmic rays, radioactive radiations from the earth's surface and radioactive elements in the body is shown in Table I.

In his paper (see p. 1761), Professor R. M. Sievert has dealt with the naturally occurring sources of radiation and measurements of low-level radioactivity. No attempt will therefore be made here to traverse this ground again.

MONAZITE AREAS

In this paper, the writer will endeavour to show that conditions in certain parts of India are particularly favourable for studies of the differences in muta-

[1] Submitted by Dr. A. R. Gopal-Ayengar, Head, Biology Division, Department of Atomic Energy, Indian Cancer Research Centre, Bombay, India.

tion rates due to differences in background radiation. But before doing so, it may perhaps not be out of place to say a few words about thorium ores and monazite, since it is these constituents on the earth's surface that contribute to the buildup of natural background levels in certain areas.

Like uranium, thorium is a derivative of acid rocks and is for the most part concentrated in granites, syenites and their corresponding pegmatites. There is, however, a basic geochemical difference between the two radioactive elements, in that while the weathering of thorium ores supervenes by and large a process of physical comminution, that of uranium ores is acted upon by chemical processes. In consequence, thorium becomes incorporated into sediments as discrete, detrital grains of primary mineral, whereas secondary uranium in sedimentary formation occurs as a diffuse, chemically absorbed entity on carbonaceous matter, phosphates and clays. The usual ore minerals of thorium are monazite, thorite and thorianite. Of these, mention will be made only of monazite, the principal ore.

Without entering into any detailed description of the mode of formation of monazite or the manner in which it finds its way into the sea—aspects which legitimately belong to the province of geology—the writer will pass on to the stage where it is found to accumulate as beach-sand deposits in different parts of the world.

As to geographical areas, menazite deposits have been found admixed with ilmenite, rutile, zircon and other rare-earth elements in patches along the coastline of India. The monazite has accumulated along the sea-shores by a process of natural concentration out of the products of rock decay in the course of long geological ages. The heaviest deposits occur in Travancore-Cochin State, in the south-west part of India. Here, over a stretch of about 100 miles (160 km), the coastline is characterized by patches of this radioactive sand. The most concentrated distribution is to be found over a 12-mile range from Neendakara to Kayankulam and in another stretch, a mile long, at Manavala-Kurichi. Although the monazite constitutes only 1% of the beach sand, the thorium content in it is one of the highest in the world, amounting to about 10.5%. Small pockets of the littoral belt also contain even higher amounts (33%).

Other areas of the world worth mentioning in this context are: Brazil, Ceylon, Indonesia, Australia, the Belgian Congo, parts of the USSR, South Africa, Madagascar, Korea, Spain and the USA. According to Davidson,[3] the beach-sand deposits of Brazil are situated in the States of Rio de Janeiro, Espirito Santo, Bahia, Parahyba and Rio Grande do Norte, extending along a coastline of more than 1000 miles, the largest accumulation being at Comaxatiba and Guaratiba in Bahia, Guarapary in Espirito Santo, Barra do Itabopoana in Rio de Janeiro and the so-called "fossil bar". Although the ratio of monazite to the other minerals is higher than in the Indian and most other sands, the Brazilian concentrate apparently has only 5–6% of thoria, as compared with 10% in Travancore.

MEASUREMENT

A preliminary sample survey of the monazite areas of Travancore-Cochin was carried out recently by the Health Physics and Air Monitoring Divisions of the Department of Atomic Energy, Government of India, to estimate the internal and external radiation exposures of the population living in the coastal areas. A more extended series of measurements is under way to obtain detailed information on the levels of activity in the areas of highest background radiation, where there is also a high population density. Measurements have also been taken in and around the houses at the active area.

The external radiation exposure in the region is caused by:

(a) β–γ radiation from natural uranium and thorium contained in the monazite, and

(b) β–γ radiation from radon, thoron and their decay products in the air (Table II).

The external exposure to alpha radiation is not important because of the small range of the particles.

Measurements were made with a thin window β–γ, Geiger-Müller counter with a thickness of 20–30 mg/cm.^{2}. The measuring instrument was calibrated with a thin walled ionization chamber. Measurements of this nature have also been carried out in Sweden by Professor Sievert who, as we have seen has been interested mainly in the measurement of low levels of activity, particularly

[3] Davidson, C. F. (1956) Mining Mag. 94, 197.

the γ radiation from living subjects. On the basis of his painstaking studies he has built up what is probably the most complete body of knowledge obtained so far on radiation exposure in human material of all age groups.

The internal exposure to the population on the monazite sands is caused by the intake of radioactive substances through air, water and food. Moreover, radon and thoron emanating from monazite will add to the contamination of the air in the vicinity. However, these gases decay in the air, and their decay products get attached to fine dust particles in the air from whence they settle down on the soil or on the population. When the air is breathed, a considerable portion of the active dust is retained in the respiratory system, where it undoubtedly acts on the epithelium. The intake of soluble compounds of uranium and thorium through food and water would increase the body burden of uranium and thorium through ingestion and become a permanent source of internal irradiation. The accompanying schematic diagram gives an idea of the disintegration of thorium and its decay products.

Representative series of measurements taken on the beach, at the surface and in the air, as well as those in the houses, are given in Tables III to VI. These relate to Neendakara Chavara (Table III), Shakthikulangara (Table IV), Panderathuruthu (Table V) and parts of Midalum (Table VI).

It will be seen that there is considerable variation in the intensities of the radiation at different points in the measured areas. While the actual amount of radiation that the population receives must remain speculative to a certain extent at this stage, the balance of evidence seems to point to the fact that the population is subjected to fairly high doses. The estimated values in terms of γ doses for different regions range from 200 mr/yr to about 2.6 r/yr. It is further estimated that the population would be exposed to a total γ dose of about 10.20 r over a reproductive span of 30 years. It may be mentioned in this connection that Travancore State has the highest density of population in India; the estimated number of inhabitants in the monazite area is of the order of 100,000. It should be stressed that the total β–γ dose in all cases was 3.5 times higher than that due to γ alone. Although in normal circumstances the β dose could be considered to have a negligible effect, the fact that the decay products of thorium, mesothorium 2 (2.1 Me.) and thorium C (2.25 Me.) are high β-energy emitters should not be lost sight of, especially when it is considered that the people come into close contact with the surface of the soil every time they sit or sleep on it. A correction factor would therefore have to be applied to the γ doses in order to estimate the total dose to the hole body as well as to the gonads.

POSSIBILITIES OF DETECTING DIFFERENCES IN SPONTANEOUS MUTATION RATES

It has become all too clear that the compilation of exact genetical data of man is beset with numerous difficulties: for one thing, there are no pure strains to work with; for another, the generation times are inordinately long. There is also the probability that many the radiation-induced genetic changes would be lethal. A considerable number of recessive mutations are passed through successive generations and may express themselves as physiological aberrations that weaken but do not necessarily kill the individual. In such cases, distinction from incidental disease processes may be difficult if not impossible, and may therefore never be resolved. Many mutations induced by radiation may be expected to affect the fertilized ovum, and hence abortions, foetal deaths, stillbirths, infant mortality, malformations, sex ratios, viability and fertility, etc., are the genetic changes most readily seen and analysed statistically. But what we need to look into also are the possibilities of detecting differences in the mutation rates of "marker" genes of populations exposed to radiation of the order present in the monazite area of Travancore. A careful study of the population structure in this area should furnish information concerning gene frequencies and their distribution in time and space, as well as data on mutation rates. Several years ago, Muller[3] raised the question of the possibility that genic erosion will result in a piling up of deleterious mutations following ameliorative medical practices. Now, in the monazite belt of Travancore we have an almost unique situation, where there would appear to be no relaxation of the forces of selection on the population, since the alleviating action of modern medical services has not found its expression to any appreciable degree. The

[3] Muller, H. J. (1950) Amer. J. hum. Genet. 2, 111.

population has been more or less stationary for generations and might be expected to show differences in mutation rates for particular traits—autosomal dominants or sex-linked recessives of the type already discussed by Professor Stevenson in his paper (see page 5 Annex 9). A control population of comparable dimensions, with similar demographic conditions and normal background radiation exists in the nearby areas.

An inquiry of such a nature would obviously be a long-range one, but would be well worth doing in view of the possibility of thus obtaining some direct evidence on the genetic consequences of naturally occurring high background radiation. The investigation would also be likely to shed light on the dosage relationships for doubling the spontaneous mutation rate and other cognate problems. Moreover, it might also reveal interesting somatic effects, such as the incidence of leukaemia, cancer and other conditions.

TABLE I.—*Level of exposure of human body to background radiation*

[Dose Unit: Milliroentgens per year]

I. RADIOACTIVE ELEMENTS IN THE BODY

Radioactive carbon (15 disintegrations per minute per gram of carbon)	2
Radioactive potassium (1980 disintegrations per minute per gram of potassium)	19
Radium (3.7×10^{10} disintegrations per second per gram of radium	(7?)

II. COSMIC RAYS

	Equator	High latitudes
Sea level	33	37
5,000 feet	40	60
10,000 feet	80	120
15,000 feet	160	240
20,000 feet	300	450

III. RADIOACTIVE RADIATIONS FROM EARTH'S SURFACE		TOTAL: I+II+III AT EQUATOR (SEA LEVEL)
Granite rock abundance in parts per million (Typical): U 4, Th 13, K 3×10^4	90	21+33+90=144
Sedimentary rock U, Th and K about one quarter as abundant as in granite	23	21+33+23=77
Ocean Abundance in parts per million U 2×10^{-3}, Th 10^{-5}, K 4×10^2	0	21+33+0=54
Uranium (ore content, 0.1% U) rock		
rock surface	2800	
inside mine (2 x 2800)	5600	
Phosphate (fertilizer) rock: 280–700 (U content, about 0.01–0.025%) rock surface		

93299°—57—pt. 2——51

TABLE II.—*The thorium series*

Name	Symbol	Half-life	Energy of radiation (Mev.)		
			α	β	γ
Thorium	${}_{90}Th^{232}$	1.39×10^{10} years	4.03		0.05
Mesothorium 1	${}_{88}Ra^{228}$ ($MsTh_1$)	6.7 years		0.02	0.03
Mesothorium 2	${}_{89}Ac^{228}$ ($MsTh_2$)	6.13 hours		2.1, 1.7, 1.0	0.06, 0.97
Radiothorium	${}_{90}Th^{228}$ (RdTh)	1.90 years	5.42, 5.34		0.084, 0.087
Thorium X	${}_{88}Ra^{224}$ (ThX)	3.64 days	5.68, 5.45, 5.19		0.24, 0.05
Thoron	${}_{86}Pa^{220}$ (Th)	54.5 seconds	6.28		
Thorium A	${}_{84}Po^{216}$ (ThA)	0.158 seconds	6.77		
Thorium B	${}_{82}Pb^{212}$ (ThB)	10.6 hours		0.33, 0.57	0.24, 0.30, 0.11, 0.25
Thorium C	${}_{83}Bi^{212}$ (ThC)	60.5 minutes	6.05, 6.09	2.25	0.04, 2.2
Thorium C^1	${}_{84}Po^{212}$ (ThC^1)	3×10^{-7} seconds	8.78		
Thorium C^{11}	${}_{81}Tl^{208}$ (ThC^{11})	3.1 minutes		1.79	2.65, 0.58, 0.51, 0.23, 0.86
Thorium D	${}_{82}Pb^{208}$ (ThD)	Stable			

TABLE III. INVESTIGATIONS AT NEENDAKARA-CHAVARA BEACH*

Beach				Road
62 52 48	82 18 16	5.4 5 4.6	14 12 12	2.2 2 2
62 54 50	44 36 32	hut 10 9	6.5 6 6	10 8 7
72 62 56	34 30 26	16 12 10	7.5 7 7	14 12 10
64 56 46	44 38 32	12 10 9	hut •	28 24 20
30 24 22	12 10 8.5	• 10 8.4	4.4 4 3	6.5 6 5.8
36 30 28	12 10 8	8.4 8.4 7.5	hut	16 12 12
22 18 14	10 9 8.5	hut • •	32 26 20	24 22 20
32 28 24	22 18 16	5.4 5 5	cowshed	2.2 2 2
22 18 16	12 10 8	hut • •	8.5 8 7.8	7.8 7.4 7
10 8 7.5	8.5 8 7.5	6 7.6 5.2	4.2 4 3.8	16 14 12

* An area of one mile by about 500 yards was scanned by taking ten points along the length and five across the width.

Reading from left to right, the three figures in each square refer to actual counts of surface $\beta+\gamma$, surface γ, and air γ, respectively.

For $\beta+\gamma$, 100 counts = 10.15 r per year

For γ, 100 counts = 2.86 r per year

TABLE IV. INVESTIGATIONS AT SHAKTHIKULANGARA (SOUTH OF NEENDAKARA-CHAVARA BEACH GRIDLINES)*

←100 yards→

30–50 yards

56 44 40	36 28 24	18 14 12	14 10 8.6
72 58 50	12 9 7.5	14 10 8 A	12 9 7.4
72 58 48	22 18 14	12 10 8	12 10 8.2
44 36 30	20 14 12 B	72 60 48	12 10 8.4
20 16 14	28 20 16	32 22 18	16 12 10
16 12 10	28 20 16	32 24 20	14 12 C 10
8 7.5 7	32 24 22	22 18 16	22 18 14

* Most of the area is covered with coconut plantations, the coconut pits being filled with the sand from the beach. Cross-road activity, one furlong from beach, is 7.

Reading from left to right, the three figures in each square refer to actual counts of surface $\beta+\gamma$, surface γ, and air γ, respectively

For $\beta+\gamma$, 100 counts = 10.15 r per year

For γ, 100 counts = 2.86 r per year

A: hut entrance	8	B: hut entrance	24	C: hut entrance	22
yard (black sand)	30	floor	12	floor	12
floor	7.5	wall	10	wall	10
wall	6.4				

TABLE V. INVESTIGATIONS AT PANDARATHURUTHU*

Beach				Canal
18 16 14.2	12 9.4 8.8	16 14 12	10.4 9 8.4	8 7.5 7
9.2 8 7.6	16 14 12	9 8.2 7.8	12 10 10	7.6 7 6.4
16 14 12	12 9.6 9	hut	10 8.8 8	7.6 7 6.6
22 18 14	10 8.6 8	14 12 10	9.6 9 8.6	8.4 8 7.6

* An area of 100 yards by 50 yards, one mile down towards Chavara from Cheriaakhiakal, was scanned by taking four points along the beach and five across it.

Reading from left to right, the three figures in each square refer to actual counts of surface $\beta+\gamma$, surface γ, and air γ, respectively.

For $\beta+\gamma$, 100 counts = 10.15 r per year

For γ, 100 counts = 2.86 r per year

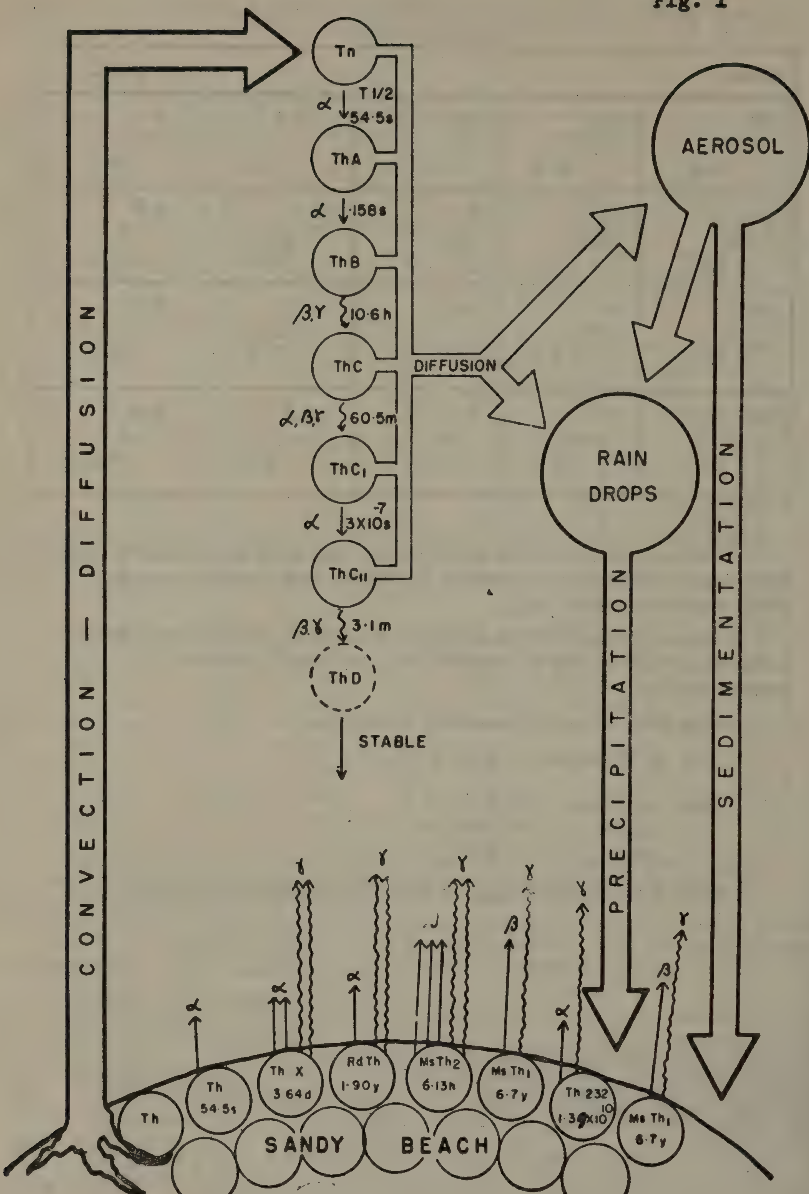
Hut:				
	entrance	10	8.6	8
	floor	10	8.4	8
	wall		8.0	

TABLE VI.—*Investigations at Midalum (Midalum teri sandhill)*

Location	Surface $\beta+\gamma$	Surface γ	Air γ
Black sandy patch	18	14	12
Black sandy stream, 1,000 yards from the sea, 500 yards from sandhill	76	70	60
In centre of six huts	24	22	20
Black and yellow spots on another stream	------------	90	80
Black and yellow spots on another stream [1]	90	80	80
Nearby garden, 100 yards upstream	18	14	12
Inside the hut:			
Entrance	9.5	8.8	8
Floor	13	12	12
Wall	------------	12	------------
Centre of 20 huts in locality	------------	8	7.5
Market	14	12	11.5
In centre of locality on wet ground	------------	12	------------
Hotel floor	12	10	9.5

[1] An underground measurement made here gave a γ count of >100.

Fig. 1



Tn
T 1/2
α
54·5s
ThA
α
·158s
ThB
β,γ
10·6h
ThC
DIFFUSION
α,β,γ
60·5m
ThC₁
α
3X10⁻⁷s
ThC₁₁
β,γ
3·1m
ThD
STABLE
AEROSOL
RAIN DROPS
PRECIPITATION
SEDIMENTATION
CONVECTION — DIFFUSION
Th
Th 54·5s
Th X 3·64d
Rd Th 1·90y
Ms Th2 6·13h
Ms Th1 6·7y
Th 232 1·39X10¹⁰
Ms Th1 6·7y
SANDY BEACH

ANNEX 9

COMPARISONS OF MUTATION RATES AT SINGLE LOCI IN MAN[1]

This note is concerned with the practical problems that arise in attempting to compare phenotype frequency, gene frequency and mutation rates between different communities. Such a comparison may be desirable for a number of reasons and, indeed, it is always something which is inherently of great interest in human population genetics. In one context, however, the comparisons which have been suggested as particularly desirable are those between communities known to have been exposed to widely different total radiation.

It seems most unlikely that it *would* be feasible to detect differences of the small order of magnitude which would be expected in such communities, or if they were detected, to attribute them with confidence to differences in background radiation alone. It would appear necessary to elaborate these and other points of criticism, however, because the suggestion has been made so frequently that it is perhaps better not to ignore, but rather to analyse, the difficulties inherent in such comparisons. This must be the writer's excuse if what follows appears largely destructive.

OUTLINE OF PROBLEMS INVOLVED

The difficulties may be summarized as follows:

1. There are the statis tical problems inherent in attempting to detect small differences in very low frequencies based on small numbers, such as mutation rates in man. Suppose in two populations of about 3,000,000 each it was desired to compare the frequency of a dominant or a sex-linked trait with an approximate expected frequency of 1/30,000. Suppose, also, that a background radiation of 3 r per generation to the gonads was expected to cause 10% of all mutations. If the two areas compared had a difference of 4 r in radiation exposure, then it would be necessary to interpret a difference of some 10–20 in the number of affected individuals in the two populations.

2. There are the hazards of assuming that any differences detected in mutation rates between two areas are, in fact, caused by different exposure to radiation. The proportion of mutations attributable to radiation is not known, and there are other factors—racial, dietary and demographic—each of which separately may determine more variation in rates than that determined by background radiation. The evidence of a close relationship between mutation and parental age is very strong for several of the genes whose expression would be suitable for comparative purposes, so that conventions of age at marriage and of the age interval between the men and women might well greatly influence the mutation rates. There are used in medicine and industry naturally occurring and synthetic substances known to be mutagenic in some lower organisms. Their effects on mammals are unknown.

3. In calculating mutation rates by the indirect method the value adopted for relative fertility of the specific phenotype could be rather critical. Yet in different populations the actual fertility of the phenotype may vary for social reasons, and because of inadequate or differing sources of demographic information it might be that only a very unsatisfactory method could be used in the comparison of two communities. For example in Denmark (Mørch[2]) a high proportion of adult achondroplastics have had offspring, while in Northern Ireland at present only one living achondroplastic is known to have had children (Stevenson[3]). In the State of Michigan, USA, the numbers of offspring born to people of both sexes at different ages can be compared (Falls & Neel[4]) whereas in England and Wales this information is available in respect of females only, and in Northern Ireland there are no national statistics of this kind. Hence the available background demographic information may not be comparable in areas otherwise suitable.

4. An essential for adequate comparison of mutation rates in two communities is that the complete patterns—not the truncated patterns which result from different degrees of ascertainment—should be available. With the best will in the world it is impossible to arrive at complete ascertainment unless the medical services are reasonably well organized, records are good and available, and

---

[1] Submitted by Professor A. C. Stevenson, Department of Social and Preventive Medicine, the Queen's University of Belfast, Institute of Clinical Science, Belfast, Northern Ireland.
[2] Mørch, E. T. (1941) Op. dom. Biol. hered. hum. (Kbh.), 3.
[3] Stevenson, A. C. (1956). (In press.)
[4] Falls, H. F., and Neel, J. V. (1951) Arch. Opthal. (Chicago), *46*, 367.

co-operation is readily given by medical and other authorities . To take a specific example, it is extremely difficult to find the older surviving sporadic boys with Duchenne-type sex-linked muscular dystrophy. If they have not had medical attention for many years, if they only attended a long time previously a hospital which kept poor records, or if they live in an area where the education authorities tend to ignore children who do not attend school, they may be well nigh impossible to find. Any comparisons between communities would therefore need to be supervised by physicians or other persons with plenty of practical experience of the difficulties and of the devices for checking and cross-checking the efficiency of ascertainment.

5. Lastly, there are the dual problems of diagnosis and of the complications introduced when it is appreciated how diverse the apparently simple relationship between gene and trait usually is. As might be expected from animal work, experience is constantly showing that human syndromes can be determined by genes at various loci or by different alleles. In addition there is, of course, the complication of the occurrence of phenocopies. The more carefully single-gene traits in man are studied, the more complex the picture becomes, so that there is hardly a trait known where there is not either some evidenec or a suspicion that the trait may be determined by various mechanisms. This does not by any means depend only on the text-book descriptions of many traits described as being caused sometimes by dominant, sometimes by recessive and sometimes by sex-linked genes. Many of these may well have arisen from the misinterpretation of pedigrees or from the selective publication of the data of remarkable families. However, clinical and biochemical separation of traits and the increasing practice of studying all families with affected members in a community constantly seem to suggest such probabilities. Perhaps more will come to light when more data are available for the analysis of various measurable characteristics of traits, such as measurements between sibs and between all the phenotypes. Perhaps, too, we shall get help in the future from some further knowledge of the morbid anatomy and histology of genetical conditions. The most remarkable example of all is probably hereditary deaf-mutism, where the evidence is strong that many recessive genes and a few dominant ones can each determine deafness which cannot be separated clinically. It is interesting to see that in mice, where similar evidence is available, even histology fails to reveal differences in lesions produced by different genes In deaf-mutism, too, the so-called "congenital" cases or phenocopies seem to constitute as many as one-third of all cases (Stevenson & Cheeseman [5]).

Such reflections make it difficult to suggest which traits would be suitable for use as markers for comparisons of the kind suggested, and indeed they leave a nasty suspicion in one's mind that certain traits might be chosen as satisfactory largely because not enough is know about them.

TYPES OF TRAIT SUITABLE FOR USE AS MARKERS

It seems worth while here to review briefly the kinds of trait which might be used as markers for mutation rates at specific loci and to point out the practical problems just mentioned as they arise. It will be generally accepted that autosomal-recessive gene traits are quite unsuitable for the purpose. In the first place, they are individually rare. Secondly, variations in inbreeding ratios, difficult to detect except in the crudest form of full-cousin rates in man, together with the fact that if small isolates with close inbreeding produce a number of cases of a rare trait the total rate will be very markedly affected, render it very hazardous to estimate a gene frequency as a preliminary to calculating a mutation rate.

In contrast, autosomal-dominant and sex-linked gene traits offer some opportunities for direct estimations and, being less dependent on vagaries from random mating, offer the chance of indirect calculation of mutation rates based on the theoretical distribution of genotypes at equilibrium developed by Haldane.[6]

It may be presumed with some confidence that the genes for which mutation rates have already been estimated are those whose characteristics are most likely to be suitable for comparative purposes. As will be seen from Tables I and II in which mutation rates for some dominant and sex-linked gene traits are given, the number of adequate calculations made is unfortunately very small and the

[5] Stevenson, A. C., and Cheeseman, E. A. 1956) Ann hum. Genet. *20,* 177.
[6] Haldane, J. B. S. (1935) J. Genet. *31,* 317.

data from which they are derived are frequently rather meagre or dependent on indirect estimates rather than on actual counts. This is intended not so much as a criticism as an indication of the difficulty—and, at times, impossibility—of assembling data which are sufficient in quantity and quality.

AUTOSOMAL DOMINANT GENES

Autosomal dominant genes would seem to offer the best opportunity for collecting data for comparative purposes, and the ideal trait would be one with the following charatceristics:

(b) that only one gene can determine the trait;
phthalmus, heart defect and rudimentary tail, and two had cleft palate, polyerrors in estimating gene frequency and in identifying new mutant phenotypes);

(c) that the phenotype cannot be mimicked by a phenocopy;

(d) that the condition is recognizable at birth or in early life but that its possessors do not die too young (this is important, in that otherwise defferential mortality experience makes estimates of frequency difficult);

(e) that the frequency of the condition is reasonably high (the lower the frequency, the larger the population which will be needed to detect differences in frequency and, hence, mutation rates):

(f) that selection against the phenotype is marked. (If it is not, direct estimates of mutation rates would be well nigh impossible to obtain as new mutant phenotypes would very seldom be observed. Indirect estimates of the mutation rate would also be less reliable, as unless the dimunition of the effective fertility is big enough to be recognized and measured, such estimates could not be made. In a low frequency trait, with little negative selection, a small number of mutations a few generations previously could result in great differences in phenotype frequency.)

Looking at the traits in Table I and the mutation rates calculated, it is unfortunately easy to point out the deficiencies of each for comparative purposes. Perhaps it may be worth just mentioning them for the benefit of non-medical readers, who may not be familiar with the clinical aspects.

Achondroplasia

There are three certain objections, and another possible one, to the use of achondroplasia as a marker. In the first place, it is fairly clear that achondroplasia as commonly recognized at birth as "different" from achondroplasia as commonly seen in older subject. Pooling the obstetric history and the foetal condition of Mørch's eight and the writer's nine cases of achondroplasia recognized at birth and born to normal parents (Stevenson, 1956), it appears that of the seventeen (six males and eleven females), six were stillborn; eight died shortly after birth; one lived for one year and died of pneumonia; one lived for eighteen months, but never walked or had any teeth, and died of pneumonia; and one lived to twenty-nine years of age. In this last case (one of Mørch's), however, the condition of the father was not known.

Further in this combined series of cases, six of the mothers had hydramnios in pregnancy and three of the babies had other gross anomalies: one had micro-
(a) 100% manifestation in the appropriate genotype (this would obviate dactyly and syndactyly.

In Northern Ireland a complete ascertainment has been made of 37 subjects presumed to have received fresh mutations, only three of whom were recognized at birth. All this suggests that the cases recognized in hospital at birth have the type of maternal history and foetal appearance which we usually associate with congenital, but not necessarily hereditary, anomalies. It further raises the question whether the possible survival of mild cases of this type may not complicate the gene frequency and fertility issues in the living. It may also be noted here that it would be hazardous to compare the incidence of this condition in births in various hospitals, as the amount and quality of ante-natal care may alter the incidence. For example, of nine cases born in the Royal Maternity Hospital, Belfast, from 1 January 1938 to 30 June 1956, six mothers were admitted either because of hydramnios or because of pre-natal X-ray diagnosis (four cases).

Secondly, from time to time cases have been reported of two achondroplastic subjects being born to normal parents. Helwig-Larson & Mørch[7] and Grebe[8]

[7] Helwig-Larson, H. G., and Mørch, E. G. (1950) Nord. Med. *43*, 180.
[8] Grebe, H. (1952) Z. Kinderpsychiat. *71*, 437.

have reported such instances, in one of which the parents were cousins. There are two such families in Northern Ireland, and again in one case the parents are full cousins. This suggests that there may be a recessive gene or allele, and introduces another complication.

Thirdly, there are the difficulties of diagnosis. The taller achondroplastic subjects are usually discovered only as the parents of affected children. Several cases about 5 foot in height have been reported. The writer has seen a man 5 foot 1 inch in height, and neither he nor his colleagues can decide whether this man is affected. If he had an affected relative in an appropriate relationship, the issue would probably be beyond doubt.

The separation of achondroplasia from Morquiö's syndrome is perhaps not as easy as is commonly assumed. For example, some cases of achondroplasia seem typical as far as limb-strength, shape of head and hands are concerned, yet radiographs of the spine show vertebral changes commonly assumed to be characteristic of Morquiö's disease.

Finally, as already mentioned, in the indirect estimation of the frequency of mutation, the value taken for the relative fertility may greatly alter the figure calculated. In Denmark a very high proportion of subjects have had offspring, mostly illegitimate, whereas only one subject in Northern Ireland is known to have had any children. Thus, variation in social standards would interfere with comparisons.

Epiloia

Epiloia must, it would seem, be ruled out as a marker. The total frequency of the trait as measured is low. Gunther & Penrose [9] estimate 1/120,000 and the nine living cases in Northern Ireland represent essentially the same frequency (Stevenson & Fisher [10]). It is possible to estimate that the real frequency of the genotype is perhaps three times as great, but such speculations, although no doubt valid in some contexts, are hardly satisfactory when attempting to compare two frequencies.

The trouble is that the gene may not be manifested at all or may appear only in such mild or uncharacteristic formation that the condition will not be diagnosed unless attention is called to severely affected relatives. In addition, the condition may be impossible to diagnose before the characteristic skin affections appear, and there will almost certainly be some undetectable cases in any large group of young epileptic children. When one adds that subjects suffer a very high mortality, that relatively few survive for thirty years, and that many cases of tuberose sclerosis are only discovered at post-mortem examination, the difficulties are even more obvious.

Retinoblastoma

This would seem to be a trait more suitable in many ways for the purpose of comparing frequencies, provided that there are good ophthalmological services in the areas observed. Children with eye symptoms rapidly come to the attention of the doctor, and those with the kind of symptoms and signs likely to be caused by retinoblastoma would be referred quickly to an ophthalmologist and the diagnosis would be made, if not immediately, then soon afterwards. In a very high proportion of cases the eyes are enucleated, and histological examination is available to confirm the clinical diagnosis. However, as it seems likely that as many as one quarter of eyes which are enucleated as a result of retinoblastoma are otherwise affected, biopsy examination is essential.

Falls & Neel,[4] who have made the most complete study as yet carried out are not willing to exclude the possibility that more than one gene can cause the condition and that some cases, particularly the uni-ocular ones, represent phenocopies. Further, they raise the question of racial differences in frequency by pointing to the apparently low frequency in people of African as opposed to those of European origin. Finally, they were not satisfied that they had made a complete ascertainment.

Waardenburg's syndrome

Waardenburg [11] estimated that the interesting syndrome described by him (hair pigment, eye and hearing defects) had a frequency in the Netherlands of

[9] Gunther, M., and Penrose, L. S. (1935) J. Genet. *31*, 413.
[10] Stevenson, A. C., and Fisher, O. D. (1956) Brit. J. soc. Med. *10*, 134.
[11] Waardenburg, P. J. (1951) Amer. J. hum. Genet. *3*, 195.

about 1/42,000, but he had to make allowance for an estimated proportion of undiscovered cases. Indeed, a direct estimate would involve examining the whole population for minor signs. It is clear from Waardenburg's account that, in a given subject, only one of the triad of hair anomaly, deafness and eye signs may be present, and it would seem impossible on clinical grounds and in terms of the effort required to examine sufficient people to make a direct estimate of the frequency of the trait. For example, in Northern Ireland only one case has been discovered, and this was a sporadic case which turned up in work on hereditary deafness.

Pelger's leucocyte nuclear anomaly

This appears to be an uncommon trait even in continental Europe, although it is much commoner there than in North America or in the United Kingdom.[12] Indeed, until controlled studies determine whether these apparent differences are in fact real, and if so whether they are racial or geographic, it would appear rather hazardous to suggest that the trait might be used for the purpose of comparing mutation rates. Further, in the absence of easily recognizable external characteristics it would require examination of perhaps 500,000 samples of blood to find a reasonable number of cases.

Aniridia

The syndrome of aniridia and mental deficiency is estimated by Møllenbach[13] to have a frequency of about 1/100 000. The condition appears to be inherited as a dominant, but with considerable variation down to complete failure of manifestation. The history of some families also suggests that there is a recessive form, and the pattern of aniridia and other associated eye anomalies described within and between families suggests that we may be observing the effects of several different genes or of alternative alleles. It would seem that further studies of this condition are needed before it may be considered for use for our purpose.

Multiple polyposis of colon

A mutation rate for multiple polyposis of the colon was calculated by a most ingenious method by Reed & Neel,[14] but the incidence of the condition could hardly be counted directly. The condition is one of multiple small benign tumours of the colon and rectum, and cases only come to attention (a) when one of the benign tumours undergoes malignant change and causes symptoms, (b) when there is accidental bleeding from the tumours, (c) when the colon is examined by sigmoidoscopy for some other purpose, and (d) when found by chance at autopsy.

These would have to be the starting-points for all cases of ascertainment of sporadic cases and for investigations of families in familiar cases. Short of passing protoscopes and sigmoidoscopes on perhaps ten thousand people and chasing relatives with such instruments, it seems unlikely that this condition could be used as a marker!

Dystrophia myotonica

Lynas[15] in the Department of Social and Preventive Medicines, Queen's University of Belfast, has made the only complete ascertainment of dystrophia myotonica that the writer has been able to discover. The greatest single difficulty in this condition is again failure or partial failure of manifestation of the gene and variation in age of onset, so that the mildly affected mutant phenotype or the mildly affected members of the present generation of a family could hardly be ascertained. Possibly, very careful assessment of the neurological condition of persons presenting with pre-senile cataract would make it possible to find more cases but there would still be an element of doubt about many cases.

Marphan's syndrome

Lynas (unpublished data, 1956) has also made a complete ascertainment of Marphan's syndrome. Here again, all the difficulties arise which are inherent in dealing with a trait which is the variable manifestation of an irregular dominant gene. Precisely parallel difficulties to those mentioned for dystrophia myotonica are encountered: diagnostic doubts in mild cases, the impossibility

---

[12] Patau, K., and Nachtsheim, N. (1940) Schr. Naturforsch. *1*, 345.
[13] Møllenbach, C. J. (1947) Op. dom. Biol. hered. hum. (Kbh.), *15*.
[14] Reed, T. E., and Neel, J. V. (1955), Amer. J. Hum. Genet. 7, 236.
[15] **Lynas, M. A. (1956) Ann. hum. Genet. *21* (in press).**

of ascertaining mild cases unless there are more severe cases in the family, and so on.

SEX-LINKED GENES

Of the sex-linked gene traits, only haemophilia and Duchenne-type muscular dystrophy appear to be sufficiently frequent and well-defined for possible use as markers. The question of differential mutation rates in males and females—raised by Haldane [16, 17] for both these conditions—must be regarded meanwhile as unproven. It should be remembered that we must rely on indirect estimates involving an estimate of relative fertility in calculating rates for sex-linked recessive genes.

Haemophilia

This seems to be a reasonably suitable condition for the purpose, provided that there are adequate clinical, pathological facilities for differentiating between haemophilia and allied disorders. Curiously enough, apart from Andreasson's work in Denmark[18] and possibly Fonio's inquiry in Switzerland,[19] no one has made a complete ascertainment of the condition, and with new techniques presenting opportunities of separating out different haemoglobins, such work seems overdue. The increasing life-span and fertility of haemophiliacs make for some difficulty in assessing mutation rates, but these do not seem insurmountable.

Duchenne-type muscular dystrophy

Three complete ascertainments of Duchenne muscular dystrophy have been reported. Stephens & Tyler's [20] and Stevenson's [21 22] data are strictly comparable clinically but Walton's [23] include two females, and one male who lived to 40 years of age, who would certainly not be accepted by the other authors. However, the clinical details given by Walton make it possible for the data to be equated, and there is reasonable agreement between the three on gene frequency and mutation rate, though perhaps Walton's ascertainment would seem to be less complete on internal evidence.

CONCLUSIONS

To sum up, it would appear unlikely that communities of sufficient size could be found which would have sufficiently different exposures to background radiation to permit detection, far less measurement, of differences in mutation rates.

The basic problem is likely to be statistical. In addition, however, problems in ascertainment, in clinical diagnosis and in the complexity of the underlying genetical mechanisms would add further to the difficulty in using the "single gene traits" which have been suggested as markers.

Finally, since it seems wise not to end on too pessimistic a note, the following points may be worth considering:

1. Suppose the proportion of mutations due to a background radiation of 3 r is not 10% but, say, 20%, the upper limit suggested in the report of the Medical Research Council of Great Britain.[24] Then given a population of 3,000,000 and a dominant trait with a frequency of about 1/30,000 as before, a difference of just under 5 r near the 3 r level would seem theoretically to give expected differences of the trait of about 30 cases, which might be interpreted as significant. If only 10% of the mutation rate is determined by radiation, then the same numerical difference in cases would require about 9 r difference in background radiation.

2. If, in spite of the difficulties outlined, mutation-rate comparisons are thought to be fundamental, then another type of planned observation than straight comparison between two areas might be more satisfactory. For example, serial comparisons of a number of defined areas, for several traits with carefully planned control of diagnostic standards and ascertainment, and the simultaneous collection of background radiation information would perhaps be more valuable than a comparison between two areas.

---

[16] Haldane, J. B. S. (1947) Ann. hum. Genet. *13,* 267.
[17] Haldane, J. B. S. (1956) Ann. hum. Genet. *20,* 344.
[18] Andreasson, M. (1943) Op. dom. Biol. Hered. Hum. (Kbh.), *6.*
[19] Fonio, A. (1954) Die erblichen und sporadichen Bluterstamme der Schweiz, Basel.
[20] Stephens, F. E., and Tyler, F. H. (1951) Amer. J. Hum. Genet. *3,* 111.
[21] Stevenson, A. C. (1953) Ann. Eugen. (Camb), *18,* 50.
[22] Stevenson, A. C. (1955) Ann. hum. Genet. *19,* 159.
[23] Walton, J. N. (1955) Ann. hum. Genet. *20,* 1.
[24] Great Britain, Medical Research Council (1956) The hazards to man of nuclear and allied radiations, London.

TABLE I.—*Estimations of mutation rates of autosomal dominant gene traits*

Trait	Basis of estimation of mutation rate	Estimated rate per million	Source
Achondroplasia	*Direct:* 8 sporadic cases in 94 073 hospital births.	43	Mørch [1]
	Indirect: $\mu=\frac{1}{2}(1-f)x=\frac{1}{2}(1-0.098)\frac{86}{3\ 793\ 000}$ (Denmark)	10	
	Direct: 6 sporadic cases in 44 109 hospital births. (South Sweden)	68	Böök [2]
	Direct: 9 sporadic cases in 31 753 hospital births.	142	Stevenson [3]
	Direct: 37 sporadic cases in 1 387 000 living subjects.	13	
	Indirect: $\mu=\frac{1}{2}(1-f)x=\frac{1}{2}(1-0.09)\frac{39}{1\ 387\ 000}$ (Northern Ireland)	14	
Epiloia	*Direct:* Estimated frequency $\frac{1}{30\ 000}$, one quarter of the cases being sporadic. (South-East England)	8–12	Gunther & Penrose [4]
Retinoblastoma	*Direct:* 51 sporadic cases from an established number of about 1 500 000 births. (London)	17	Philip & Sorsby (unpublished data, 1947) [5]
Retinoblastoma	*Direct:* 49 sporadic cases in 1 054 985 births (State of Michigan, USA)	23	Falls & Neel.[6]
	Direct: 47 sporadic cases in 1 376 000 births (Germany)	17	Vogel.[7]
Waardenburg's syndrome (hair pigment, eye and hearing defects).	Based on proportion of cases observed in deaf mutes, an estimate of "penetrance" and the frequency of deaf mutism (Netherlands)	(4) 4	Waardenburg.[8]
Multiple polyposis of colon.	Based on frequency of condition at autopsy and proportion of cancer of colon autopsies showing some polyposis (State of Michigan, USA)	13	Reed & Neel.[9]
Dystrophia myotonica	$\mu=\frac{1}{2}(1-f)x=\frac{1}{2}(1-\frac{1}{5})\frac{33}{1\ 370\ 921}$ (Northern Ireland)	8	Lynas.[10]
Marphan's syndrome	$\mu=\frac{1}{2}(1-f)x=\frac{1}{2}(1-\frac{1}{2})\frac{36}{1\ 370\ 921}$	5	Lynas (unpublished data, 1956)
Aniridia	28 sporadic cases (1875–1944) and 13 isolated cases in 1944 in population of 3,844,000. Estimated frequency $\frac{1}{200,000}$.	5	Møllenbach.[11]

[1] Mørch, E. T. (1941), Op. dom. Biol. hered. hum. (Kbh.), *3*.
[2] Böök, J. A. (1952), J. Genet. Hum. *1*, 24.
[3] Stevenson, A. C. (1956) (in press).
[4] Gunther, M., and Penrose, L. S. (1935), J. Genet. 31, 413.
[5] Based on data of Griffith and Sersby (Griffith, A. D., and Sorsby, A. (1944), Brit. J. Ophthal. *28*, 279).
[6] Falls, H. F., and Neel, J. V. (1951), Arch. Opthal. (Chicago), 46, 367.
[7] Vogel, F. (1954), Z. KonstLehre, *32*, 308.
[8] Waardenburg, P. J. (1951), Amer. J. Hum. Genet. 3, 195.
[9] Reed, T. E., and Neel, J. V. (1955), Amer. J. Hum. Genet. 7, 236.
[10] Lynas, M. A. (1956), Ann. hum. Genet. *21* (in press).
[11] Møllenbach, C. J. (1947), Op. dom. Biol. hered. hum. (Kbh.), *15*.

TABLE II.—*Estimations of mutation rates of sex-linked recessive gene traits*

Trait	Basis of estimation of mutation rate	Estimated rate per million	Source
Haemophilia	Estimates frequency in London as between 35 and 175 per million births and relative fertility of affected male subjects as 0.25. (London)	50	Haldane.[1]
	$\mu=1/3(1-f)x=1/3(1-0.286)\times 1.33\times 10^{-4}$	32	Andreasson [2] modified by Haldane.[1]
	$\mu=1/3(1-f)x=1/3(1-0.333)\times\frac{3.163}{4\ 092\ 025}$ Based on data of Fonio [4] and Andreasson.[1] (Switzerland and Denmark)	27	Vogel.[3]
Duchenne-type muscular dystrophy.	18 cases in 67 000 male live-births $\mu=1/3(1-f)x=1/3\times1\times\frac{18}{67\ 000}$ (State of Utah, USA)	95	Stephens & Tyler.[4]
	36 cases in 162 488 male live-births $\mu=1/3(1-f)x=1/3\times1\times\frac{36}{162\ 448}$ (Northern Ireland)	74	Stevenson [5] also unpublished data 1956.
	16 cases in 138 403 male live-births $\mu=1/3(1-f)x=1/3\times1\times\frac{16}{138\ 403}$ (England)	39	Walton.[6]

[1] Haldane, J. B. S. (1935), J. Genet. *31*, 317.
[2] Andreasson, M. (1943), Op. dom. Biol. Hered. Hum. (Kbh.), 6.
[3] Vogel, F. (1955) Z. ges. Blutforsch. *1*, 91.
[4] Stephens, F. E., and Tyler, F. H. (1951) Amer. J. Hum. Genet. *3*, 111.
[5] Stevenson, A. C. (1955) Ann. hum Genet. *19*, 159.
[6] Walton, J. N. (1955), Ann. hum. Genet. *20*, 1.

ANNEX 10

SOME PROBLEMS IN THE ESTIMATION OF SPONTANEOUS MUTATION RATES IN ANIMALS AND MAN [1]

In view of the known species differences both in the genetic structure of populations and in the apparent genetic responses to irradiation, when considering the genetic impact of increased exposure to ionizing radiation we should prefer not to attempt to extrapolate from other species to man, but rather base our thinking entirely on human data. Unfortunately, as has already become abundantly clear, the necessary data on man are not yet at hand, nor is it likely that they will be for some time to come. Under the circumstances, our thinking must for the present be guided to a large extent by what we know about the genetics of other species.

Attempts to quantitate the effects of radiation on human populations have usually been based on five factors. These are:

(1) The spontaneous mutation rate/locus/generation.

(2) The induced mutation rate/locus/*r*.

(3) The total gene number.[2]

(4) The 'accumulation factor', i. e., the ratio of nominally recessive genes already present in the population to those arising spontaneously each generation through mutation.

(5) The manner in which selection operates on the total gene complex.

Although I was asked to speak on "extrapolation from animals to man: the problem", this is so very broad an assignment that rather than utter a few

[1] Submitted by Dr. J. V. Neel, Associate Geneticist, Heredity Clinic, Institute of Human Biology, University of Michigan, Ann Arbor, Mich., USA. The material presented in this paper is drawn in large part from Chapter XV of a forthcoming monograph by J. V. Neel and W. J. Schull, entitled "The Effect of Exposure to the Atomic Bombs on Pregnancy Termination in Hiroshima and Nagasaki", and is reproduced by permission of the publishers, the National Academy of Sciences-National Research Council (USA).

[2] The product of (1)x(3), or (2)x(3), is the rate of mutuation pergamete, spontaneous or induced, as the case may be. It is possible in suitably designed experiments to estimate this directly (cf. Muller, 1955), and so decrease the number of variables involved in the calculation.

generalities about each of the factors just mentioned, and how they are manipulated to give estimates of the quantitative risks of radiation, I would like to examine in some detail the present state of knowledge as regards just one of these. The factor to be singled out for special consideration is 'spontaneous mutation rates'. There is no deep significance in the choice of this topic, i. e., any one of the four other factors of importance to attempts at extrapolation might almost equally well have been chosen for detailed consideration. In what follows, attention will repeatedly be drawn to gaps in knowledge. This should in no way detract from the past accomplishments of investigators in the field, but is submitted in the belief that the primary purpose of this meeting is to discuss what remains to be done if we are to place in proper perspective the genetic risks of ionizing radiation to human populations.

Current thinking concerning the rate of mutation of mammalian genes is for obvious reasons strongly influenced by what is known concerning drosophila rates. No less pertinent, but more difficult to fit into our conceptual framework for man at present, is the extremely important data emerging from the study of other inframammalian forms, such as the recent work on bacteriophage of Benzer (1955). We will accordingly first consider briefly what seem to us to be some of the more pertinent data concerning drosophila. For methodological reasons it is customary to distinguish on the basis of their physiological effects between three categories of mutations, namely, those associated with visible effects, those associated with lethal effects, and those which express themselves through a reduction of viability in the absence of detectable somatic effects, the so-called semi-lethal mutations (1–10 per cent. viability) and deleterious mutations (over 10 but less than 100 per cent. viability). Terminology in this field leaves something to be desired. Thus, the 'deleterious' mutations must have an organic basis, so that many of them would be found on careful study to be also 'visibles'. By the same token, most 'visibles' are also 'deleterious'. Finally, the dividing line between 'lethals' and 'semilethals' may be altered by culture conditions. Be that as it may, the division into these three categories has an operational usefulness, as we shall now see.

Beginning with the pioneer attempts of Muller (1934; see also Kerkis, 1935) and Timoféeff-Ressovsky (1935) a number of efforts have been made to establish the relative frequencies with which these types of mutations are represented among all mutations. These attempts have involved radiation-induced rather than spontaneous mutations because of the much more laborious nature of the problem if attacked through the study of spontaneously occurring mutations. In view of the possibility that the relative frequency of lethals is higher among the radiation-induced mutations because of the increased proportion of minute deletions, the estimate of the ratio, (semi-lethals + deleterious)/lethals, may be a minimum estimate. Muller (1954; see also Falk, 1955) places this ratio at 3–5 to 1. This same author goes on to state that ". . . the ratio may indeed be considerably higher than this, since the technique was hardly refined enough for the detection of detrimentals with a viability greater than some 85 per cent. of normal. Other studies have shown that 'invisible' mutants causing sterility or lowered fertility of some degree also form a very large group. This group, however, overlaps, to an extent not yet well investigated, that of the detrimental mutations (p. 396).

The significance of information concerning the relative frequency of mutants with viability in the 85–99 per cent. range in attempts to quantitate the genetic risks of ratiation is of course enormous. A related problem concerns the frequency of mutations for which the organism *at the time* is able to compensate completely, the undetectable mutations. Lately considerable attention has been directed towards the genetic basis and evolutionary implications of physiological homeostasis (refs. in Lerner, 1955). The possibility cannot be ruled out that the principle of homeostasis enables some organisms to compensate entirely, under particular sets of circumstances, for the effects of certain mutations.

It may be argued that there is no reason to be concerned about the relative frequency of mutants with undetectable effects in a consideration of the deleterious effects of radiation. However, these mutations are undetectable only under the conditions set by the observer. Under other conditions, set by nature and not by man, they might have decided effects. It is not at all difficult to argue that the mutants with over 85 per cent. viability which cannot now be studied in drosophila may in evolutionary importance far outweigh the visibles.

Muller (1950), in a discussion of the question of the numerical relationship between lethals, on the one hand, and semi-lethals and deleterious mutations, on the other hand, has made the following statement: "However, studies carried

on in drosophila during the past year by Meyer, Edmondson, and the writer indicate that in this organism the assumption of an equal distribution of detrimental mutations throughout all i_{ho} values[3] (when represented on an arithmetic scale) does not hold. Instead, it appears that, following the high but descending peak formed by complete lethals (i_{ho}=100 per cent.) and nearly complete lethals (i_{ho} = between 98 per cent. and 100 per cent), there is a marked drop in the frequency of mutations. The mutations studied were induced in an autosome (the second chromosome) by ultraviolet light acting on an interphase stage (in the polar cap). Along with 208 complete lethals there were 20 mutants found in the range of i_{ho} between 98 per cent. and 100 per cent., and again only 20 in the range of i_{ho} between 90 per cent. and and 98 per cent., although this range is four times as wide as the preceding one. If the rest of the distribution, as far as i_{ho}=10 per cent. had only the same frequency of mutations as in the range between 90 per cent. and 98 per cent. there would have been only 240 detrimentals in the entire interval between 100 per cent. and 10 per cent. to set against the 208 complete lethals found. But since we know from other work, previously cited, that the detrimentals in this interval are in reality several (about five) times as numerous as the complete lethals, it is evident that their frequency must, at lower degrees of detriment (lower i_{ho}), rise very much above that existing in the 90 per cent. to 98 per cent. range. The distribution of frequencies of i_{ho} therefore forms a bimodal curve with one peak at the left origin, lethality (i_{ho}=100 per cent.), and another peak somewhere to the right.

"Little more than this is yet known definitely about the shape of the curve in question, important though this genetic question is. However, there are grounds, both theoretical and observational, for regarding it as very unlikely that the second peak is near the first or that the rise towards it is sharp. Hence it is probable that detrimental mutations, instead of having an even distribution with respect to values of i_{ho}, form a curve which, except for its peak of near-lethals at the left end, is massively skewed towards the right, with its mean at a value of i_{ho} significantly beyond the middle (0.5)." (pp. 140–141).

If we consider these remarks of Muller in conjunction with the possibility of 'invisible' mutants discussed earlier, then the problem of estimating the relative frequency of lethal mutants versus those viable to some degree assumes new complexity. Fig. 1 attempts to present some of this complexity graphically. The abscissa of this figure represents viability of the homzygous genotype in some arbitrary environment. In this connexion, it is apparent that the term 'lethal' is relative, some lethal mutations having effects under no known circumstances compatible with life, other lethal mutations having far lesser effects. Likewise, the term 'normal' as applied to viability is relative, some normals being more normal than others, with the differences brought out only under unusual circumstances. Thus far, observations have been limited to the range of lethality and 1–85 per cent viability. As Muller has pointed out in the statement quoted above, there is great doubt concerning the shape of the curve of numerical relationships within this range. We have indicated two of the principal alternatives. Curve A assumes a mode at 60–70 per cent. viability, from which it would seem likely that the proportion of mutations in the 85–100 per cent. and normal viability range is small. Curve B assumes that the mode is farther to the right with the corollary that there is a considerable group of mutations not now being detected. How large that group is depends of course on the shape of the curve.

The question of the relative frequency of lethal mutations as contrasted to visibles, is on somewhat more secure footing than the question of the ratio of lethal mutations to mutations reducing viability to a lesser degree. In tabulating the results of radiation experiments by five different workers, Schultz (1936) found this ratio to be 7.4 : 1. In view of the well-recognized differences in the ability of individuals to recognize mutant phenotypes, the true ratio is probably somewhat lower. We refer, for instance, the ratio of 5.2 : 1 which obtained in the extensive and meticulous experiments of Spencer & Stern (1948). Even this ratio may be too high. Thus, in the control cultures, Spencer & Stern obtained a ratio of sex-linked lethals to visibles of 4.3 : 1 and in the irradiated cultures, a ratio of 5 : 3 : 1. In a study on spontaneous mutations in a 'high mutation rate' line, the ratio of sex-linked lethals to visibles was 3.6 : 1 (Neel, 1942). The ratio of visibles :lethals :semi-lethals and deleterious mutations may, as an approximation, be said to be someplace between 1 : 4 : 16 and 1 : 6 : 30, with, as noted above, the most uncertainty centring about the magnitude of the third figure in the ratio.

[3] i_{ho} = the amount of impairment produced by a gene when homozygous.

The important question of the mutation spectrum at individual loci remains in its early stages because of the amount of labour involved in securing reliable data. The effort involved in studying this problem through the use of spontaneously occurring mutations appears almost prohibitive. Attempts to study the problem using induced mutations again encounter the question of how precisely the mutational spectrum obtained with mutagenic agents parallels that derived from the study of spontaneous mutations. However, there is some preliminary evidence that the ratios just given may vary significantly from locus to locus. Thus, although there are many instances of lethal and visible mutations arising at the same locus, there are also some few cases in which a locus does not appear to be essential to life, in the sense that flies with a deficiency for this locus may live although they are of reduced viability (e. g., yellow and achaete, Muller, 1935). These loci, then, would not produce at least one type of lethal mutation. Finally, for methodological reasons, localizing 'deleterious' mutations to specific loci is extremely difficult, so that studies relating these to the loci producing lethals and visibles are in an early stage.

It should also be pointed out that the question of the total relative frequency of mutation at different loci is in a very unsettled state. Although there seems no doubt that the rate of recovery of mutations differs from locus to locus, care must be exercised in reasoning as to the magnitude of the true differences (cf. Neel & Schull, 1954). In the following discussion of mutation rates at specific loci, the fact that these are *selected* loci must constantly be borne in mind.

With respect to the rate of occurrence of spontaneous 'visible mutations' at specific loci in drosophila, data are available from five extensive series of observations. These are summarized in Table 1. Time does not permit us to give this table the detailed attention it deserves. In most of these studies, some 'special circumstance' occurred that requires at least very brief mention. Thus, Muller, Valencia, & Valencia (1950) observed in other experiments with the same strain used for their 'visible mutation' series that the rate of occurrence of sex-linked lethal mutations in this strain was 0.7 per cent., a rate some fourfold greater than usual. From this they argue that "the frequency of gene mutations at the nine loci would *ordinarily* average between 10^{-5} and 7 x 10^{-6} per locus in females" (p. 125). However, in view of the possibility that these 'high mutation rate' lines contribute significantly in nature to the total of spontaneous mutation (Ives, 1950; see also Neel, 1942), it seems appropriate simply to average this finding with the others. From the data of Glass & Ritterhoff (1956), it would appear that the mutation rates of males are higher than females, but this is scarcely substantiated by the difference in the findings of Alexander (1954) and Muller, Valencia, & Valencia (1950). Accordingly, we have simply averaged all the findings without regard to sex. No attempt has been made to take into account the effect on the observed results of possible differences in the age of the flies tested. The paper of Glass & Ritterhoff contains additional data on mutation rates emerging incidentally to their study—it has seemed preferable in this summary to utilize only data on loci 'pre-selected' for mutation rate estimates. It would appear that Muller et al., and Schalet, did not score as mutants flies with mutant phenotypes which were infertile, whereas Glass & Ritterhoff did. In any mutation rate study, a considerable proportion of apparent mutants prove sterile. Schalet encountered one 'cluster' of ten 'cut' mutants, presumably due to a mutation occurring in a spermatogonium. One can argue as to whether this should be scored as 1 or 10 mutations in the context of the present discussion. Finally, in all these studies where mutation gives rise to one mutant in a culture of wild type, there is the question, probably not so serious in studies on man, of the extent to which the less vigorous mutants are eliminated prior to the inspection of the culture, and the further problem of the human factor in recognizing the mutant.

While, then, it is possible to 'correct' the data of Table 1 in several ways, I have made no attempt to do so. In a total of 4 625 945 locus tests, at least 41 mutations were recovered, a rate of 0.9 x 10^{-5}. However, *this applies only to 'visible mutations', or to lethal and deleterious mutations which in combination with a mutant allele have visible effects.* If other lethals, semi-lethals, and deleterious mutations are arising at these same loci which are without detectable visible effects with the test-crosses employed, the mutation rate must be higher. There is no way at present to estimate the amount of mutation not detected with current specific locus techniques, but if, for instance, the ratio of visibles/

(undetected lethal+semi-lethal+deleterious+sterile mutations) at these loci were as high as 1:4, the mutation rate per locus becomes 4×10^{-5}. In other words, there are some grounds for feeling that the commonly quoted mutation rates for specific loci for drosophila are conservative. If, on the other hand, the mutation spectrum at specific loci is restricted, as some evidence suggests it to be in the sense that some loci give rise predominantly to 'visibles' and others to 'deleterious' mutations, then the 'true' mutation rate may be closer to the 1×10^{-5} which emerges from specific locus studies than the 4×10^{-5} just suggested.

Two other studies involving individual loci should be quoted. Lefevre (1955), in a paper which contains an excellent discussion of the problem of estimating spontaneous mutation rates, reports that the rate of appearance of mutants with visible or lethal effects at the *y* locus is "about 1 per 75,000" (p. 379). On the other hand, Bonnier & Lüning (1949), in a paper criticized by Muller (1954) because the rate of recovery of spontaneous mutations appeared to be too low in comparison with certain other findings, observed only one mutation at the white and forked loci among 153 579 flies tested for visible mutations, a rate of 3×10^{-5}. Again, both of these estimates do not take into account the semi-lethal and deleterious mutations which may not be detected by the techniques being employed.

Utilizing a somewhat different approach, Dobzhanzky, Spassky, & Spassky (1952) have estimated the average rate of mutation to lethals, semi-lethals (1–20 per cent. viability), and visibles *per lethal producing locus* in different species. These estimates, which they felt are more likely to be overestimates than underestimates, are reproduced in Table 2. Again, the estimate does not include the deleterious mutants.

In summary, then, it would appear that depending on one's view of the representativeness of the loci studied, and the problem of the relative frequency of mutations not detected by current techniques, there is room for a divergence of opinion concerning the average rate of mutation of drosophila genes, with the range of possibilities perhaps extending from 0.5×10^{-5} to 5×10^{-5}. In our opinion, even this range of estimates must be applied with great caution to human problems.

Turning now to mammals, we find that significant studies are available for only two species, the house mouse and man himself. The figures for the house mouse were derived in much the same fashion as the figures quoted for drosophila, namely, through a search for mutant individuals among animals simultaneously heterozygous at multiple loci. Russell (1954), in connexion with his important observations on radiation-induced mutations in the house mouse, has found that in his control material the rate of appearance of *visible* mutations in 265 076 locus tests was 0.8×10^{-5}. The observational error is of course large. Again it must be recognized that these tests very probably detect only a fraction of the mutations occurring at these loci.

There seems no reason to labour further the point that our knowledge of spontaneous mutation rates at specific loci is poor for any species. In some attempts to extrapolate from non-human material to man, the additional problem arises of the greater life span of man than of laboratory material, as well as the question of the type of species which man represents in terms of mutation rate. While I would be the first to defend the animal work as providing the best estimates available at the present time, it is my opinion that in the final analysis, we must have figures for man himself.

The available estimates for the frequency of occurrence in man of certain mutations with visible effects have already been summarized by Professor Stevenson and Professor Penrose. Many of the problems involved in estimating human mutation rates have been discussed by these two authors as well as previously (Haldane, 1948, 1949; Neel (1952) Neel & Schull, 1954; Nachtsheim, 1954) and will not be re-examined here. The average of the available estimates of the rate of mutation for the autosomal dominant and recessive sex-linked mutations thus far investigated in man is in the neighborhood of 2×10^{-5}/locus/generation. These estimates, now, are entirely limited to mutations with visible effects. Because of the nature of the design of observations on human mutation rates, it seems possible that a higher proportion of the mutations at the specific loci being studied is going undetected in man than in Drosophila. There is no way to estimate the magnitude of this difference at present, but a total mutation rate as high as 1×10^{-4} at these loci is a possibility. Although the

apparent correspondence between human and *Drosophila melanogaster* rates is noteworthy, because of the differences in the way the rates are obtained one must be cautious in the emphasis placed on the similarity. However, if the correspondence were indeed valid, this has interesting implications concerning the importance of 'aging' and failures in the 'copying process' at mitosis, and the role of background radiation, in spontaneous mutation rates.

The representativeness of these estimates for man has been repeatedly challenged. There can be no doubt that there is definite selection in the loci studied. How this influences our estimates is not at all clear. As we have pointed out elsewhere (Neel & Schull, 1954), mutation at any particular locus may be thought of in terms of these aspects: (1) the frequency of mutation at that locus; (2) the number of alternative forms of the gene which may occur at any locus, i. e., the number of multiple alleles; and (3) the ease with which the effect associated with each of these multiple alleles can be detected. We assume that some loci are more mutable than others because we detect the results of mutation more frequently at these loci. However, making allowance for 'unstable loci', the hypothesis has not been disproven that the inherent instability of all genes is, by virtue of their biochemical complexity, very similar, but that the results of mutation are more readily detected at some loci than at others because of the role of that particular locus in the animal's physiology. It is entirely conceivable that the loci [4] thus far selected for study in man are those at which a high proportion of all possible alleles at that locus results in readily detectable effects, but at which the per locus mutation rate is fairly representative of the human species.

For purposes of calculation, estimates of the rate of mutation of human genes have included 10^{-5} (Evans, 1949), 10^{-7} (Wright, 1950), and 2×10^{-5} (Muller, 1950; Slatis, 1955). In the current state of our knowledge, students of the problem can select and justify estimates differing from one another by a factor of 100.

If time permitted, we would do well to submit to the same kind of scrutiny our knowledge concerning the other factors that enter into quantitative treatments of the risks of irradiation, namely, (1) induced mutation rates at specific loci, (2) gene number (or, alternatively, gamete mutation rates), (3) the accumulation factor, and (4) the manner of action of selection. This will obviously be impossible. However, in closing I would like to say just a few words about the nature of selection in human populations. To begin with, there would seem to be little problem in extrapolating from animals to man, since there is practically nothing known concerning the detailed action of selection in animal populations on which to base an extrapoliation. For all our allegiance to the principal of natural selection, it is amazing how little we know of its actual detailed workings. True, it is easily demonstrated in experimental populations that grossly defective individuals seldom reproduce. But the problem of how the population as a whole maintains its fitness is virtually untouched. To mention only one important point, to what extent does the stability and adaptability of the species rest on the mechanism of balanced polymorphism, a mechanism not readily disturbed by an increase in mutation rates?

There is one final point I should like to emphasize. In our attempts to evaluate the genetic risks of increased radiation to the human species, I am a strong proponent of extensive animal experimentation. Through such work, possibilities can be explored which would either involve prohibitive amounts of time or be impossible for man. But when differences do appear between two animal species, as is already the case, only work on man will tell which of the species he resembles more closely. It is of tremendous significance to the practice of medicine and the development of atomic energy whether the 'permissible' population dose above background is 3 *r* or 30 *r* per generation. In reaching any final conclusions concerning permissible radiation doses in man, regardless of what is learned concerning other animal species, we must have accumulated far, far more data on man himself than are now available.

---

[4] In point of accuracy, we do not know but what any particular mutation rate study in man is detecting mutation at several loci.

TABLE 1.—*Frequency of occurrence of spontaneous "visible mutations" in various species*

Author	Chromosome	Number of organisms	Number of loci	Total locus tests	Mutations	μ
Drosophila:						
Muller, Valencia, & Valencia, 1950.	X (females)	±60,000	9	540,000	15	2.8×10^{-5}
Altenburg, after Muller et al., 1950.	X (females)	±50,000	8	400,000	0	0
Alexander, 1954	III (males)	45,504	8	364,032	0	0
Glass & Ritterhoff, 1956	Multiple (females)	100,414	4	401,656	1	2.5×10^{-6}
	Multiple (males)	102,759	[1] 4	359,657	17	4.7×10^{-5}
Schalet, unpublished	X (males)	111,600	14	1,562,400	[2] 6	0.4×10^{-5}
	X (females)	71,300	14	998,200	2	0.2×10^{-5}
				4,625,945	41	0.9×10^{-5}
House mouse: Russell, 1954	Several	37,868	7	265,076	2	0.8×10^{-5}

[1] 1 sex-linked.
[2] One of these mutants appeared as a "cluster" of 10 flies. If one is concerned only with the rate of recovery of mutant phenotypes, then the entry here should be 15, and the average of the five studies quoted becomes 1.1×10^{-5}.

TABLE 2.—*Estimated average mutation rates per lethal-producing locus in several drosophila species, after Dobzhansky, Spassky, & Spassky (1952). These estimates are felt by the investigators to be more likely overestimates than underestimates*

Species	Second Chromosome	Third Chromosome
D. Melanogaster	1.1×10^{-5}	
D. pseudo-obscura		1.1×10^{-5}
D. willistoni	2.2×10^{-5}	3.0×10^{-5}
D. prosaltans	1.1×10^{-5}	2.1×10^{-5}

REFERENCES

Alexander, M. L. (1954) Mutation rates at specific autosomal loci in the mature and immature germ cells of drosophila melanogaster, *Genetics, 39*, 409–428

Benzer, S. (1955) Fine structure of a genetic region in bacteriophage, *Proc nat. Acad. Sci. (Wash.), 41*, 344–354

Bonnier, G. & Lüning, K. G. (1949) Studies on X-ray mutations in the white and forked loci of drosophila melanogaster. I. A statistical analysis of mutation frequencies, *Hereditas (Lund), 35*, 163–189

Dobzhansky, Th., Spassky, B. & Spassky, N. (1952) A comparative study of mutation rates in two ecologically diverse species of drosophila, *Genetics, 37*, 650–664

Evans, R. D. (1949) Quantitative inferences concerning the genetic effects of radiation on human beings, *Science, 109*, 299–304

Falk, R. (1955) Studies on X-ray induced viability mutations in the third chromosome of drosophila melanogaster, *Hereditas (Lund), 41*, 259–278

Glass, B. & Ritterhoff, R. K. (1956) Spontaneous-mutation rates at specific loci in drosophila males and females, *Science, 124*, 314–315

Haldane, J. B. S. (1948) The formal genetics of man, *Proc. roy. Soc. B.* 135, 147–170

Haldane, J. B. S. (1949) The rate of mutation of human genes, *Proc. VIII Int. Cong. Genetics. Hereditas Suppl.*, 267–273

Ives, P. T., (1950) The importance of mutation rate genes in evolution, *Evolution, 4*, 236–252

Kerkis, J. J. (1935) The preponderance of "physiological mutations", *Summ. Commun. XV Intern. Physiol. Congr.* (Leningrad-Moscow) pp. 198–200 (quoted from Muller, 1954)

Lefevre, G. Jr. (1955) Mutation isoalleles at the yellow locus in drosophila melanogaster, *Genetics, 40*, 374–387

Lerner, I. M.. (1955) Buffered genotypes and improvement in egg production, *Amer. Nat. 89*, 29–34

Muller, H. J. (1934) Radiation genetics, *Proc. IV Intern. Radiologen Kongr.*, *2*, 100–102

Muller, H. J. (1935) A viable two-gene deficiency phaenotypically resembling the corresponding hypomorphic mutations, *J. Hered.* *26*, 469–478

Muller, H. J. (1950) Our load of mutations, *Amer. J. hum. Genet.* *2*, 111–176

Muller, H. J., Valencia, J. I., & Valencia, R. M. (1950) The frequency of spontaneous mutations at individual loci in drosophila, *Genetics*, *35*, 125–126

Muller, H. J. (1954) The nature of the genetic effects produced by radiation. In: *Radiation Biology* (A. Hollaender, ed.) New York: McGraw-Hill Book Co., Vol. I, pp. 351–453

Muller, H. J. (1955) How radiation changes the genetic constitution. *Bull. atom. Scient.* *11*, 329–338

Nachtsheim, H. (1954) Die Mutationsrate menschlicher Gene, *Naturwissenschaften*, *17*, 385–392

Neel, J. V. (1942) A study of a case of high mutation rate in drosophila melanogaster, *Genetics*, *27*, 519–536

Neel, J. V. (1952) The study of human mutation rates, *Amer. Nat.* *86*, 129–144

Neel, J. V. & Schull, W. J. (1954) *Human Heredity*, Chicago: University of Chicago Press, 361 pp.

Russell, W. L. (1954) Genetic effects of radiation in mammals. In: *Radiation Biology* (A. Hollaender, ed.) New York: McGraw-Hill Book Co., Vol. I, pp. 825–859

Schultz, J. (1936) Radiation and the study of mutations in animals, In: *Biological Effects of Radiation* (B. M. Duggar, ed.) New York: McGraw-Hill Book Co., Vol. II, pp. 1209–1261

Slatis, H. M. (1955) Current status of information on the induction of mutations by irradiation, *Science*, *121*, 817–821

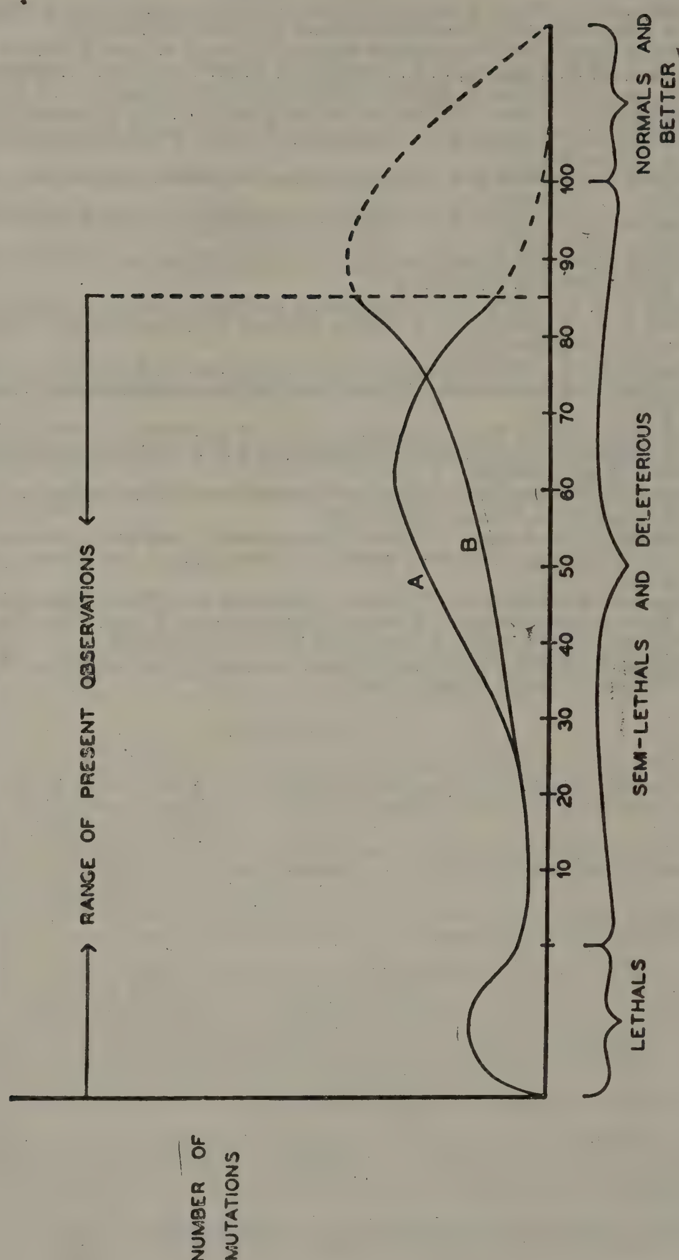
Spencer, W. P. & Stern, C. (1948) Experiments to test the validity of the linear *r*-dose/mutation frequency relation in drosophila at low dosage, *Genetics*, *33*, 43–74

Timoféeff-Ressovsky, N. W. (1935) Auslösung von Vitalitätsmutationen durch Röntgenbestrahlung bei drosophila melanogaster, *Nachr. Ges. Wis. Göttingen* Math.—physik. Klasse, Fachgruppen VI. Biol.) *1*, 163–180

Wright, S. (1950) Discussion on population genetics and radiation, *J. cell. comp. Physiol.* *35*, Suppl. 1, pp. 187–204

Figure 1

A schematic representation of two possible "mutation spectra" with reference to the degree of viability of the mutant, both compatible with the present data.



ANNEX 11

EFFECT OF INBREEDING LEVELS OF POPULATIONS OF INCIDENCE OF HEREDITARY TRAITS DUE TO INDUCED RECESSIVE MUTATIONS [1]

It is well known that the incidence of hereditary traits in populations depends not only on the respective gene frequencies, but also on the breeding structure of the populations. One of the important aspects of the prevailing mating pattern can be measured through the use of the coefficient of inbreeding, a population parameter practically impossible to evaluate in natural populations of animal species, but very easily determined in man.

INBREEDING IN BRAZIL

During the past six years, the writer has become very interested in this particular problem in human genetics and has tried to discover the inbreeding levels under which the present-day Brazilian populations live, as well as the magnitude of the same parameter during the last 150 years. The study of this problem in Brazil is greatly facilitated by the fact that much data can be obtained through the analysis of Catholic marriage records, a source of information which is available for the great majority of the population. The calculation of the coefficients of inbreeding has been done on the basis of data on the frequencies of marriages between uncles and nieces, aunts and nephews, first cousins, first cousins once removed, and second cousins. A few of the results obtained have already been published (Freire-Maia [2 3 4]), but the great bulk of the data will be presented in a paper now in preparation.

The following features of the breeding pattern of Brazilian populations have become apparent:

1. The degrees of inbreeding vary greatly in different regions: the rates are relatively low in the south and in parts of the east (a mean level of about 1% of first-cousin marriages has been found); are very high in large regions of the east and north-east (a level as high as 10% for first-cousin marriages has been detected); and are intermediate, but highly variable, in other regions.
2. There is, in general, a clear trend towards decreasing inbreeding rates, although a few reversals of this trend have been noted.
3. Geographic inbreeding gradients have been detected in some zones, with increasing inbreeding from the coast to the hinterland.
4. Although some of the Brazilian rates of inbreeding are higher than the highest so far found in other countries, the mean Brazilian coefficient of inbreeding (0.002) is relatively low because about one-third of the population lives at the level of 1% of first-cousin marriages.

An analysis of the factors probably responsible for the different degrees of inbreeding found in Brazil revealed that cultural pattern, economic level, migration, population density and degree of ruralization seem to be the most important.

EFFECT OF INBREEDING ON POPULATION STRUCTURE

It is known from theoretical analysis that inbreeding coefficients as high as 0.01 and 0.02, such as are found in some localities, produce negligible effects on common recessive traits, but have a considerable influence on the rare ones (Table I). Suffice it to say, for instance, that characteristics with a gene frequency of 1% will, under the action of a coefficient of inbreeding of 0.01 (which prevails in large populations in Brazil), have phenotype frequencies 99% greater than those expected in a model assuming no inbreeding (Table I) and 90% greater than those expected in populations with a coefficient of inbreeding of 0.001 (Table II). These almost 100% increases could hardly be detected in a direct way, through the analysis of the phenotype distribution in the populations, but may be appreciated through the incidence of the so-called "recessive" diseases in the offspring of consanguineous marriages, as well as through the incidence of consanguineous marriages among the parents of people presenting the same kind of genetic traits. Some data obtained on deaf-mutism show, for instance, a really very high inbreeding effect. In a population where the frequency of

[1] Submitted by Dr. N. Freire-Maia, Director, Laboratory of Genetics, University of Paraná, Curitiba, Paraná, Brazil.

[2] Freire-Maia, N. (1952) Amer. J. hum. Genet. 4, 194.

[3] Freire-Maia, N. (1953) Ciencia (Méx.), 1, 26.

[4] Freire-Maia, N. (1954) Carologia, 6, Suppl., p. 923.

first-cousin marriages has been estimated as 3.5%, there has been shown to be about 21% of first-cousin marriages among the parents of deaf-mute children (Aguiar & Freire-Maia[5] and unpublished data). Inasmuch as some of these children undoubtedly owe their defect to extrinsic factors, the frequency of consanguinity among the parents of children with genetically determined defect is of course even higher. This problem will, however, be discussed in detail elsewhere.

EFFECT OF INBREEDING UNDER INCREASED MUTATION RATE

In recent times one of the most important human genetics problems is the evaluation, on a quantitative basis, of the effects of increasing radiation levels on the genetic composition of populations. Unfortunately, no accurate mathematical treatment could be given to this subject up to the present, as no precise information has yet been collected on some basis phenomena—namely, the spontaneous mutation rates, the induced mutation rate per gene per roentgen, the total number of loci, etc. (see discussion in Neel & Schull[6]). Thus the treat ment to be presented below is *not* intended to show what happens under increasing radiation, but what could happen under increasing inbreeding. The emphasis will be put *not* on the rates of mutation frequency increment but rather on the fact that, assuming a given increment, the action of different inbreeding rates will produce different quantitative effects.

For instance, assuming that the probability of induced mutation per gene per roentgen in man is of the same order (2.5×10^{-7}) as that found in the mouse (Russell[7]), doses of 100 r would increase this probability to 2.5×10^{-5}. Thus, five different populations (A, B, C, D, and E), differing only in the intensity of inbreeding, with two given recessive genes at frequencies of 0.007 and 0.003, will have the frequencies of these genes increased respectively to 0.007025 and 0.003025. However, the increase of the frequencies of the recessive genotypes will depend on the inbreeding level of each population (Table III). Five inbreeding coefficients have been chosen for comparison: 0.001, 0.003, 0.006, 0.009 and 0.011. The first one, representative of European populations according to Haldane,[8] also holds true for southern Brazil; the third has been assumed to characterize the highly inbred Japanese populations as a whole (Neel et al.[9]); the fourth is the highest detected Brazilian coefficient for a large zone (the centre of the north-eastern region); the fifth is probably the coefficient now prevalent in the populations of this particular zone; and the second has been selected to represent an intermediate step between the "low" European level (0.001) and the "high" Japanese one (0.006). Table III shows how much different coefficients of inbreeding may change the phenotype composition of populations subject to the same radiation impact. With the initial frequency of 0.003, it is seen that the Brazilian coefficients 0.009 and 0.011 produce total increments (0.01125 and 0.01275) more than twice as great as that produced by the European and southern Brazilian coefficient of 0.001. With the initial frequency of 0.007, the effect is a little smaller. The action of the Japanese mean coefficient is somewhat intermediate, as expected. Other things being equal, then, it is to be expected that induced recessive mutations manifest their effects with much higher frequencies in populations with inbreeding rates like those found in some Brazilian regions than in some European or North American populations.

PROSPECTIVE GENETIC RESEARCH ON INBREEDING

Now that we possess the basic information on inbreeding rates in Brazil, it has been possible to discover some "modern" populations living at inbreeding levels probably comparable to those of European communities in the Middle Ages. In the focus of the highest inbreeding levels in Brazil, for instance, some localities have been found where as many as 1 out of 6 and 1 out of 5 of the marriages are contracted between first cousins, and 1 out of every 3 marriages is consanguineous up to and including second cousins. This situation would seem

---

[5] Aguiar, W. C., and Freire-Maia, N. (1953) Ciênc. e Cult. *5*, 203.
[6] Neel, J. V., and Schull, W. J. (1956) The effect of exposure to the atomic bombs on pregnancy termination in Hiroshima and Nagasaki (in press).
[7] Russell, W. L. (1951) Cold Spr. Harb. Symp. quant. Biol. *16*, 327.
[8] Haldane, J. B. S. (1947) Ann. Eugen. (Camb.), *14*, 35.
[9] Neel, J. V. et al. (1949) Amer. J. hum. Genet. *1*, 156.

to be of great potential usefulness in the study of the general effect of consanguineous marriages on the genetic make-up of populations and the discovery of the mean number of deleterious recessive genes per individual. Unfortunately, in the zones in Brazil where the very high inbreeding rates prevail, no analysis regarding the incidence of specific hereditary anomalies seems feasible because the level of medical practice there is lower than in the larger cities. Nevertheless, in these regions a study can be made of such population characteristics as the frequencies of abortion, miscarriage, stillbirth, infant mortality, malformations as a whole, etc., and should afford some interesting results. Furthermore, in Rio de Janeiro, Sao Paulo, and some other cities, where the inbreeding rates are probably from ten to twenty times higher than those prevailing in similar or even smaller cities in the USA (Glass;[10] Herndon & Kerley;[11] Steinberg (personal communication, 1956); Woolf et al.[12]), and where very good hospitals exist, a complete and detailed analysis is possible.

ACKNOWLEDGMENT

The writer is indebted to Dr J. V. Neel and Dr J. N. Spuhler for discussions in connexion with the preparation of this manuscript.

TABLE I.—*Effect of two coefficients of inbreeding (0.01 and 0.02) on incidence of recessive traits with different gene frequencies (q)*

$q\%$	$q^2\%$	$\alpha=0.01$			$\alpha=0.02$		
		$\alpha pq\%$	$q^2+\alpha q(\%)$	$\frac{\alpha pq(\%)}{q^2}$	$\alpha pq\%$	$q^2+\alpha pq(\%)$	$\frac{\alpha pq(\%)}{q^2}$
50	25	0.25	25.25	1	0.5	25.5	2
20	4	0.16	4.16	4	0.3	4.32	8
10	1	0.09	1.09	9	0.18	1.18	18
5	0.25	0.0475	0.2975	19	0.095	0.345	38
1	0.01	0.0099	0.0199	99	0.0198	0.0298	198
0.5	0.0025	0.004975	0.007475	199	0.00995	0.01245	398
0.1	0.0001	0.000999	0.001099	999	0.001998	0.002098	1 998

TABLE II.—*Effect of two coefficients of inbreeding (0.001 and 0.01) on incidence of recessive traits due to genes with frequency of 1%*

Incidence of the traits		Increment
$\alpha=0.001$	$\alpha^1=0.001$	$\frac{\alpha^1 pq}{q^2+\alpha pq}$
0.01099%	0.0199%	90%

[10] Glass, B. (1950) Cold Spr. Harb. Symp. quant. Biol. *15*, 22.

[11] Herndon, C. N., and Kerley, E. R. (1952) Cousin marriage rates in Western North Carolina (Paper presented at the annual meeting of the American Society of Human Geneticists, Ithaca, N. Y.; unpublished).

[12] Woolf, C. M. et al. (1956) An investigation on the frequency of consanguineous marriages among the Mormons and their relatives in the United States. (In press.)

TABLE III.—*Effect of inbreeding level of populations on frequency of recessive traits under increased mutation pressure, according to Haldane's formulae* [1 2]

Population	Coefficient of inbreeding (α)	Initial frequency of recessives $q^2+\alpha q(1-q)$ (A)	Increment of recessives $\Delta q(\alpha+2q)$ (B)	Increased frequency of recessives (A+B)	"Total" increment [3] (Bx30 000)
$q=0.003$ $q+\Delta q=0.003025$					
A	0.001	0.000011991	0.000000175	0.000012166	0.00525
B	0.003	0.000014973	0.000000225	0.000015198	0.00675
C	0.006	0.000026946	0.000000300	0.000027246	0.00900
D	0.009	0.000035916	0.000000375	0.000036291	0.01125
E	0.011	0.000041901	0.000000425	0.000042326	0.01275
$q=0.007$ $q+\Delta q=0.007025$					
A	0.001	0.000055951	0.000000375	0.000056326	0.01125
B	0.003	0.000069853	0.000000425	0.000070278	0.01275
C	0.006	0.000090706	0.000000500	0.000091206	0.01500
D	0.009	0.000111559	0.000000575	0.000112134	0.01725
E	0.011	0.000125461	0 000000625	0.000126086	0.01875

1 Haldane, J. B. S. (1947), Ann. Eugen. (Camb.), 14, 35.
2 For details, see Neel et al., footnote 9 in text.
3 Assuming an identical behaviour of 30 000 loci in gametes (Spuhler, J. N. (1948) Science, 108, 279).

ANNEX 12

DETECTION OF GENETIC TRENDS IN PUBLIC HEALTH [1]

To assess the practical genetic consequences of irradiating human populations, one must either: (a) extrapolate from mutation-rate studies in exposed animals, or men, to the effects of the additional mutations on human health and fitness; (b) extrapolate from fitness studies in animals to health and fitness in man; or (c) measure the important changes in the genetic component of health and fitness directly in the human populations which are exposed to a rising background of radiation.

The first two of these approaches have received considerable emphasis because the experimental procedures are relatively straightforward, and because predictions are needed, however tentative they may be. Unfortunately, however, it is extremely difficult to extrapolate from increases in mutation rates to the magnitude of the resulting increases in amount of general ill health, or from the fitness of animal populations to the fitness of human populations. In fact, there is reason to doubt whether the extent of the effect of a given increase in background radiation can ever be adequately anticipated.

The logical complement to prediction lies in the development of some sensitive means of detecting important genetic trends before they have gone too far. Ultimately, of course, this detection is the only way our predictions can be tested.

The main deterrents to setting up a continuing survey aimed specifically at the detection of important long-term genetic trends are the absence of any certainty as to how sensitive a method can be devised, and the very considerable financial and organizational difficulties. However, since we lack the assurance that "prediction" alone will fill our needs, it seems important that the feasibility of "early detection" receive much fuller consideration than it has in the past.

The present account deals with the application of certain of the methods of vital statistics to such detection. It seems essential to make it quite clear that the suggestions which follow are not at present recommended lines of action for general adoption by central vital statistical or record departments. These suggestions have been made earlier with Canada, solely as personal recommendations, with a view to studying their feasibility. As presented here, however, the remarks are designed simply to show the possibility of collecting specific data on human variation which could not possibly be assembled on a comparable scale by the conventional *ad hoc* field inquires used in population genetics. The methodology is discussed in this paper in the hope of getting constructive criticism before we embark on research on such a large scale.

1 Submitted by Dr. H. B. Newcombe, Biology Branch, Atomic Energy of Canada Limited, Chalk River, Ont., Canada.

Sources of Information

In general, to discriminate between genetic and environmental causes (either in genetic conditions of individuals or in population trends) information is needed concerning the number of affected and unaffected individuals, their family relationships, and the environments to which they have been exposed. Considerable information of all three kinds exists in the routine registrations of births, deaths and marriages. The handling procedures are not at present designed to discriminate between the genetic and environmental contributions to the diseases which are reported on the death registrations, but if maximum use were made of all three kinds of information we could presumably make such a distinction, with at least some degree of success.

Since routine vital statistics are a recognized measure of the health of a population, one approach to the detection of genetic trends would seem to lie in supplementing the basic information where necessary, and in designing handling procedures to distinguish the genetic from the environmental causes of ill health.

The approach is limited at present with regard to the health information, which relates solely to causes of death and still-birth, but other routine sources could be tapped. For example, one Canadian province (British Columbia) has made use of the "Physician's Notices of Births and Stillbirths", which are quite separate from the birth registrations, to obtain details of still-births and congenital abnormalities. The problem of adequate ascertainment is undoubtedly soluble.

The chief advantage in the use of registrations of births, deaths and marriages, however, is that these contain, in raw form, the most reliable and complete information on the family relationships of the individuals who make up the population. They are in essence a family tree on a very large scale (complete with marriage dates, birth dates, and the dates of all deaths). To extract this family information manually, and to convert it into a usable form, would be a prodigious task, and for this reason we have concentrated much of our thinking on the development of mechanical procedures involving the matching and sorting of punch cards to form a "Family Register Index".

A "Family Register" would contain cards for all marriages, starting from a given year. To these would be added the cards for all births arising out of these marriages, which would be identified and sorted into their respective sibling groups. In addition, all cards for still-births and for the deaths of offspring would be similarly identified and sorted. A further procedure has been devised whereby any marriages between first cousins could be identified, together with the births and deaths of their offspring, without resorting to interviews and without reference to any other kind of record (see Fig. 1 and 2).

Thus, for each disease condition on which information is available, it would be possible to determine the incidence within three groups of individuals: the population as a whole, the offspring of first-cousin marriages, and the siblings of affected individuals. This seemed the most suitable use to make of the family relationship data in an initial study, but the information could be applied in many other ways.

In addition, the registrations contain a considerable amount of routinely recorded information on environment, which would permit a breakdown of any data by the following factors: rural or urban residence; socio-economic class, as derived from father's occupation; age of mother and of father at time of birth; family size and spacing; racial origin; gestation period; legitimacy or illegitimacy; and home *versus* institution birth. This is probably adequate for any initial study, and supplementary information could undoubtedly be obtained.

The "Family Register Index" is the one unique feature of the present proposals. In emphasizing the mechanical methods involved, it should be explained that it was felt that the personal-interview technique of obtaining pedigrees would become too laborious in any study involving both a large population and many of the common diseases. And yet, if the common diseases were not included, or if the study were limited to a few genetically simple "indicator conditions", it would be difficult to relate any trend observed to the general health of the population. And the latter is, of course, our ultimate practical concern.

In making these proposals it has been assumed that the present problem of genetic damage from radiation, and the related problem of the operation of other causes in the production of genetic trends, are of sufficient importance to justify any appropriate changes in the collection and analysis of statistics relating to the health of the whole population. Our prime concern is with broad categories

of ill-health, and methods of obtaining and handling information on the very large numbers of affected individuals and their relatives need to be developed.

Rationale

In general, the greater the complexity of the genetic and the environmental causes of a condition, the more information of the three kinds (i. e., pertaining to health, family and environment) will be required to disentangle the two.

Thus, to look for a trend in the frequency of a simple dominant "indicator" condition, it would only be necessary to observe the proportions of affected individuals in the population. And to detect trends involving a simple recessive gene it would be sufficient to know the proportions of affected individuals in offspring from consanguineous parents. However, if one extends the survey to conditions arising from a recessive gene with incomplete penetrance, it would be necessary, in addition, to know the corresponding proportion of affected individuals in the rest of the population. In this case the gene frequency would be calculated from the ratio of the two, referred to as a "K" value; and since penetrance affects both components of the ratio equally, both "K" and the calculated gene frequency would be essentially independent of penetrance.

The number of diseases can, of course, be extended still further to include those due to dominant genes of unknown penetrance and those due to multiple additive genes, using comparisons between siblings (or other closely related individuals). Formulae for the estimation of gene frequencies from data of this kind (using the ratio of the incidence in close relatives of affected individuals to that in the population as a whole, i. e., a "K" value) have been derived by Penrose[1] and applied to a number of common diseases.

Where our main interest is in the detection of changes in the gene frequencies, rather than in the absolute frequencies, the problem is considerably simplified. Attention centres on trends in the values of "K", and it is not essential to know whether the genes are recessive, dominant, or multiple additive.

Thus, a basic requirement for discriminating between the genetic and the environmental trends affecting public health is a knowledge of the proportions of affected individuals within three groups of people: the offspring of consanguineous unions, the close relatives of affected individuals, and the population from which these were drawn.

Environmental changes, when they affect penetrance uniformly throughout the population, are unlikely to produce spurious trends in the estimates of gene frequencies. This is true also of changes affecting the extent of the ascertainment, and of changes in diagnostic fashion, when they occur uniformly throughout the population. However, there are a number of sources of error, and additional information would be needed in order to detect and evaluate them. Such information would relate mainly to the environment.

Gene frequencies based on consanguinity data would be least subject to bias, the main source of which is the fact that marriages between close relatives tend to be more common in certain sectors of the population—notably, the rural groups. To eliminate errors from this source it would be necessary to obtain independent values of "K" from the various population groups (e. g., breaking the data down by: rural or urban residence; racial origin; socio-economic class; etc.) or, better still, from comparisons with offspring from the brothers and sisters of the individuals who married their cousins.

Gene frequencies based on "K" values for sibling comparisons may be biased in a number of ways. In general, where there are family-to-family differences in environment affecting the expression or penetrance of an hereditary disease, the increased tendency for affected individuals to appear within particular families will increase the value of "K" and bias the estimate of gene frequency downwards. Environmental heterogeneities which might give rise to this kind of bias could be associated with: (a) maternal effects due to the mother's hereditary constitution; (b) maternal effects due to the environment to which the mother has been exposed; and (c) effects due to the child's post-natal environment. It should be possible to detect any bias from these sources, and to estimate its magnitude.

Thus, where there are maternal effects due to the mother's heredity, these should make for a closer resemblance between the children and any of their first cousins by their mother's sisters, than with first cousins of the other three kinds (i. e., offspring of the mother's brothers, of the father's sisters, or of the father's

[1] Penrose, L. S. (1953) Acta genet. (Basel), 4, 257.

brothers). The extent of the discrepancy (allowance having been made for differences in the likelihood of inheriting similar X-chromosomes) should indicate the magnitude of the bias. Important environmental variables other than inherited maternal effects should be strongly correlated with the incidence of the condition in a suitable breakdown by environmental groups.

Environmental differences may operate by altering either the expression of a genetic condition (i. e., the "penetrance" or "expressivity") or the production of non-genetic effects which simulate the genetic condition (i. e., "mimics" or "phenocopies"). Variations in environment might tend to group the affected individuals into families by either mechanism, thus increasing the value of "K" and causing the gene frequency to be underestimated. But variations in the production of mimics could operate in the opposite manner through obscuring the grouping due to genetic causes. Both environmental influences will be observed in an appropriate breakdown of the data, as a correlation between environmental group and incidence of affected individuals.

The two effects will in many cases be distinguishable, however, by observing the "K" values for appropriate environmental groups. Where the influence is on penetrance, the value of "K" for any homogeneous group will tend to be less than that for the mixed population (and the estimates of gene frequency will be less biased). Where the influence is on mimic production, the value of "K" for a genetic condition would tend to be increased in homogeneous favourable groups where mimics are rare, and decreased in the unfavourable groups where they are common. In either case, the most reliable estimates of gene frequency would be obtained from groups living in the most uniformly favourable environments.

Such refinements, using information which is already collected as a matter of routine, would remove many of the sources of bias. Further, since genetic applications were not envisaged in the planning of the present system of vital statistics collection, improvements could undoubtedly be devised after any major attempt to apply the existing information. It is, of course, impossible to predict just how sensitive a means for the detection of genetic trends might eventually be developed; this can only be done as experience is gained in using the information which we already have in a readily available form.

In case the present proposals seem over-optimistic, it is worth noting that at least one serious attempt has already been made to detect a genetic trend in a complex quantitative character (namely, intelligence[2]) which is known to be subject to environmental influences, and that this attempt has made very little use of information on family relationships and environment, and of the refinements which these permit.

DETAILS OF FACILITIES AND PROCEDURES

Microfilms of the registration forms for all births, deaths and marriages occurring in Canada are kept centrally at the Bureau of Statistics. From each of these microfilms, a punch-card, bearing a non-repetitive serial number and containing particulars of the event and of the individuals involved, is prepared as a matter of routine. At present a modification in the punching of these cards is under consideration. The modification is designed to enable each birth card to be identified mechanically with the marriage card of the parents, matching by name of father, maiden name of mother, and parents' initials and birth years; while the death cards would in a similar manner be identified with the individual's birth cards, matching by family name, first name and initials, province and date of birth. A small proportion of apparent discrepancies are known to arise, almost all of which could be matched manually.

The new birth and death cards would have additional blank spaces into which could be transferred the serial number from the corresponding marriage card. This operation would be mechanical, and the serial number from the marriage card would then become a "family number", enabling all three cards to be readily identified into family groups.

The change in the method of punching would not add appreciably to the present costs, which are in the vicinity of $100,000 per year, while the additional matching procedures and the punching of the family number might perhaps double these costs. This estimate refers to a proposed ten-year pilot study, but in a continuing study the handling of an expanding file of cards would involve a further increase in cost which has not as yet been estimated.

---

[2] Scottish Council for Research in Education (1949) Report of the * * *, London.

In addition, to identify all marriages between first counsins, the microfilms of the marriage registration forms would be scrutinized, and those in which one of the bride's parents had the same family name as one of the groom's parents would be singled out. In the case of these registrations (which amount to about 1%–2% of all marriages in Canada), the birth records of the bride and groom, and then of the respective parents of similar family name, would be checked for positive identification of the marriages which are in fact between first counsins. One man can scan approximately 1000 marriage registrations a day for this purpose, and searching of birth records is a function which the provinces carry out routinely at a relatively small cost.

When a Family Register Index is created, it would be used in conjunction with an Ill-health Register of all "affected" persons, who will be identifiable by their names and by the dates and places of their births, if they have not already been identified by the "family number". The latter register would include still-births, infant deaths, other deaths, congenital malformations, hospital records, other medical records, etc. From the two registers one would derive the sizes of the sibling groups and the numbers of affected individuals in each. Weinberg's propositus method would be used to calculate the probability that a sibling of an affected individual will be similarly affected: $p=\Sigma x(x-1)/\Sigma x(s-1)$, where p is the required probability, x the number of affected persons in the families, and s the number of children of the individual families. The incidence of the condition in offspring of first-counsin marriages, and in the population as a whole, would be obtained directly.

The Family Register Index can be thought of as a major research tool, designed to do away with the need for obtaining pedigrees by personal interview and thus to pave the way for whole-population studies of common diseases. Such studies would seem to be an integral part of any attempt to measure the practical consequences of genetic trends in terms of the general health of the population.

Details of Suggested 10-year Pilot Study

It has been proposed that before embarking on a major continuing programme of an entirely new kind, the design should be tested in a preliminary special study. In the present case there is an additional reason for such a study.

The main programme, if started solely with current registrations of marriages, and of the births and deaths of the children arising out of these, would require approximately four years before any comparisons could be made in brother-sister groups, and about ten years before it would have expanded sufficiently to yield data for a breakdown by cause of death. Only then would it be possible to evaluate the design of the project.

To avoid loss of time a special study could be carried out, essentially similar to the projected continuing study, but using existing records on a "backlog" basis. In drawing up the specifications it was assumed that the special study would cover the ten-year period from 1946 to 1955 inclusive. An attempt has been made to foresee the amount and kind of data which might be expected from the special study. It is estimated that there would be in the vicinity of a million infants born to the marriages under study, and that approximately half of these would have at least one brother or sister with whom comparisons could be made.

The special study would deal mainly with infant deaths, and should indicate the extent to which deaths from various causes tend to be correlated in families and in the offspring of first-counsin marriages. These correlations (i e., the factors by which the various causes are more common in these two groups of individuals than in the population as a whole) are the values which will be expected to change with changes in gene frequencies. The study would show how large these factors are for the various causes of death, together with the confidence limits, and would at the same time indicate any changes which should perhaps be made in the design of the continuing study.

In addition, since the relatives of affected individuals constitute a group in which the frequencies of the predisposing genes are, as it were, artificially increased, the results should give us a better appreciation of the practical consequences of an increase in the incidence of deleterious genes in the population as a whole.

The extensive Family Register Index, developed during the special study, would be used in the continuing study so that current births from much earlier marriages could be included from the start. This would ensure an appreciable annual yield of data without having to wait until the marriages occurring in the first year of the study had yielded two children.

FUTURE FACILITIES AND THEIR POTENTIALITIES

With the present punch-card equipment a storage problem would eventually develop. The family Register Index must contain three cards for each individual who has been born, married and died, and family groups of cards will have to be retained until the last of the brothers and sisters has died. Probably the equilibrium number of cards would be in the vicinity of three for each living member of the population. The obvious solution is a system of miniature cards, capable of being mechanically sorted and matched. If, in addition, these cards had an increased information capacity and could be handled more rapidly, the usefulness of the Family Register Index would be enormously increased.

One such system—the Kodak Minicard, which has been described by Tyler, Myers & Kuipers [8]—is in the process of development. (Details: card size, 32 mm by 16 mm; storage space required, 15 inches by 30 inches by 50 inches per 2 000 000 cards; digital information capacity, between five and six times that of the standard punch-cards, with room for a photographic image of the original registration form as well; handling speeds of 1800 cards per minute for sorting and selecting.) Such a medium could replace both the existing microfilm and the punch-cards, while taking approximately the same space as the microfilm alone.

The most important use for the additional information capacity would be in the identification of relatives more distance than brothers and sisters. The "family identity number" assigned at the time of marriage would be carried forward, not only to the children's birth and death cards, but to the children's marriage cards as well, and so on. The number of steps in this carryover would of course depend ultimately on the amount of digital information space allocated to ancestor-family numbers.

Let us assume, for example, that two generations of ancestor-family numbers are present on all cards (requiring, for 10-digit numbers, 60 out of the 420 spaces which will be available when the card contains a photographic image as well). Causes of death could then be compared in children, parents and grandparents, and in other relatives as remote as second cousins, using a single sorting and matching of death cards.

Another medium which might have applications is magnetic tape. (Information capacity, 100 characters per inch; handling speed for tabulation and other purposes, 15,000 characters per second.) Records from cards which had been suitably sorted in advance could be incorporated each year, together with the accumulated records of previous years, into a single master-tape, which would be revised annually. Assuming that such a tape contained records from 50 000 000 cards with 100 characters per card, it would require approximately 100 hours (not counting the changing of reels) to run the entire tape through a machine in order to tabulate the information in the required form.

In view of the rapid improvement in the designs of such equipment, the bulk of information to be processed, and the complexity of the operations involved, should not constitute more than a temporary limitation on any system of handling which was deemed necessary.

CONCLUSION

In this account it has been assumed that we may not be able to assess adequately the genetic damage occurring in irradiated human populations, either from a knowledge of the changes in mutation rates, or by observing the changes in fitness in similarly irradiated animal populations, or even by observing the prevalence of a number of genetically well-defined "indicator conditions". With each of these observations there remains an uncertainty as to the amount by which the ill-health of the population has been altered, and if the answer cannot be stated in such terms it is of only limited use.

This would seem to lead us to the much more arduous and exacting task of attempting to detect changes in the genetic factors which affect broad categories of human ill-health. The degree of precision which might be achieved is impossible to predict, but it is clear that we could not afford to waste any of the available information relating to the health of the individuals who make up the population, to their family relationships, or to the environments in which they have been reared.

Experience in effectively handling the masses of information of these three kinds which are at present readily available would seem to be one of our imme-

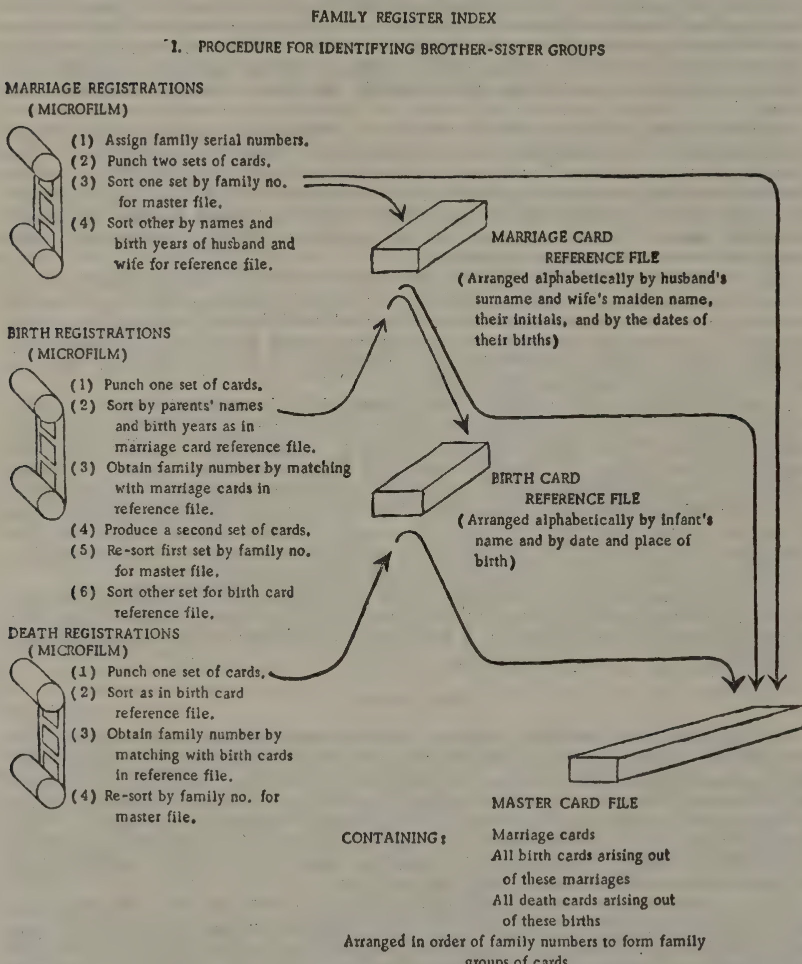
[8] Tyler, A. W., Myers, W. L., and Kuipers, J. W. (1955) Amer. Documentation, 6, 18.

diate needs, while improvement in the routine sources of information is another. It is with the first of these needs that the present paper has been primarily concerned.

ACKNOWLEDGMENTS

While the opinions expressed in this paper are entirely those of the writer, the procedures described are the outcome of numerous discussions with representatives of the Bureau of Statistics and the Department of National Health and Welfare, and with other geneticists. In the latter connexion, the writer would like to thank Mr Fraser Harris, Mr S. J. Axford, Mr Gordon H. Josie, Dr A. P. James and Dr F. Clarke Fraser for their generous co-operation.

FIG. 1. FAMILY REGISTER INDEX - 1



If both the family information and the vital statistics are included on a single card, tabulations from the Master Card File will yield family sizes and numbers of children "affected", from which sibling correlations can be derived. Note that with the use of a miniature card the storage space required for the Master Card File would be about the same as that needed for the microfilms of the corresponding registration forms.

FIG. 2. FAMILY REGISTER INDEX—II

FAMILY REGISTER INDEX

II. PROCEDURES FOR IDENTIFYING FIRST COUSIN MARRIAGES

A. Procedure to be used when family index is first started

(1) Examine all marriage registrations for cases in which a parent of the groom and a parent of the bride have the same surname (or maiden name).

(2) Where this is observed, search the birth records of the bride and groom for the birth years of the parents of similar name.

(3) Then, search the birth records of the parents of similar name. Where the bride and groom are first cousins the names of the common grandparents will be found on both registrations.

(4) Enter consanguinity on the marriage card in the family register index, and on all subsequent birth and death cards arising out of this marriage.

B. A more direct procedure using the family numbers assigned to the marriages in the two preceding generations (the method will be usable after the family register index has been in operation for about 40 years)

(1) Carry forward the following family serial numbers on to all new marriage cards:

(a) From the groom's parents' marriage
(b) From the bride's parents' marriage
(c) From the groom's paternal grandparents' marriage
(d) From the groom's maternal grandparents' marriage
(e) From the bride's paternal grandparents marriage
(f) From the bride's maternal grandparents' marriage

(2) Where the family number for (c) or (d) is the same as that for (e) or (f), the bride and groom are first cousins. Cards in which this is the case will be identified mechanically.

(3) Enter consanguinity on the marriage card, and on all subsequent birth and death cards arising out of the marriage.

The first of these procedures can be used immediately in the case of all provinces where the maiden names of the mothers of both bride and groom appear on the marriage registration form (i. e., all provinces in Canada except Quebec). The second procedure will enable consanguinity data to be obtained from all of the provinces, after the Family Register Index has been in operation for a sufficient period.

APPENDIX 4

THE BIOLOGICAL EFFECTS OF ATOMIC RADIATION AND EXCERPTS FROM PATHOLOGIC EFFECTS OF ATOMIC RADIATION, STUDIES BY THE NATIONAL ACADEMY OF SCIENCES, NATIONAL RESEARCH COUNCIL

THE BIOLOGICAL EFFECTS OF ATOMIC RADIATION

SUMMARY REPORTS FROM A STUDY BY THE NATIONAL ACADEMY OF SCIENCES

National Academy of Sciences—National Research Council, Washington, 1956

FOREWORD

The reports published in this volume summarize the first technical findings and recommendations of six committees established to carry on a continuing study of the biological effects of atomic radiations from the points of view of

genetics, pathology, meteorology, oceanography and fisheries, agriculture and food supplies, and the disposal and dispersal of radioactive wastes.

The members of these committees, numbering more than 100, are among the most distinguished scientists in their fields in the United States. They have given generously of their time and talents in making this analysis during the past several months because they are convinced that their fellow citizens should have the facts about the biological effects of atomic radiations based on all existing knowledge available to us. The members of the committees served as individuals, contributing their knowledge and their judgment as scientists and as citizens, not as representatives of the institutions, companies, or Government agencies with which they are associated.

The use of atomic energy is perhaps one of the few major technological developments of the past 50 years in which careful consideration of the relationship of a new technology to the needs and welfare of human beings has kept pace with its development. Almost from the very beginning of the days of the Manhattan Project careful attention has been given to the biological and medical aspects of the subject. By contrast, the automobile revolutionized our pattern of living and working, but we are only now beginning to appreciate the problems of safety, urban congestion, nervous tension, and atmospheric pollution which have accompanied its development. In the same way, the development of the aircraft industry outran our knowledge of how to meet the environmental needs of the human beings it intended to transport through the skies.

The reports now completed vary greatly as to the extent of technical detail they contain. The full reports of each committee, including technical appendices where these have been prepared, will be published at a later date by the National Academy of Sciences. Here only the essential facts, arguments and conclusions as seen today by each Committee are published. As further research provides new facts or further consideration sheds new light on what is now known these conclusions will almost certainly be modified. Moreover as time permits certain specialized aspects of the problem will be studies in more detail by the Committees. The results of these further analyses will be published from time to time as the National Academy of Sciences' study continues.

Douglas M. Whitaker, Vice President of the Rockefeller Institute, has provided coordination and liaison among the study committees with the assistance of Charles I. Campbell of the Academy staff. The study has been greatly assisted by consultations with many authorities in private and Government organizations. Particular mention should be made of the cooperation of the United States Atomic Energy Commission and the Department of Defense. Financial support of the Academy's study of the biological effects of atomic radiations is provided by the Rockefeller Foundation.

DETLEY W. BRONK,
President, National Academy of Sciences.

June 4, 1956.

MEMBERSHIP OF THE COMMITTEE ON GENETIC EFFECTS OF ATOMIC RADIATION

Warren Weaver, The Rockefeller Foundation, Chairman
H. Bentley Glass, Johns Hopkins University, Rapporteur
George W. Beadle, California Institute of Technology
James F. Crow, University of Wisconsin
M. Demerec, Department of Genetics, Carnegie Institution of Washington
G. Failla, Columbia University
Alexander Hollaender, Oak Ridge National Laboratory
Berwind P. Kaufmann, Department of Genetics, Carnegie Institution of Washington
C. C. Little, Roscoe B. Jackson Memorial Laboratory
H. J. Muller, Indiana University
James V. Neel, University of Michigan
W. L. Russell, Oak Ridge National Laboratory
T. M. Sonneborn, Indiana University
A. H. Sturtevant, California Institute of Technology
Shields Warren, New England Deaconess Hospital
Sewall Wright, University of Wisconsin
Consultants:
John S. Laughlin, Sloan-Kettering Institute
Ira Pullman, Nuclear Development Corporation of America

MEMBERSHIP OF THE COMMITTEE AND SUBCOMMITTEES ON PATHOLOGIC EFFECTS OF ATOMIC RADIATION

Committee Members:
- Shields Warren, New England Deaconess Hospital, Boston, Chairman
- Austin M. Brues, Argonne National Laboratory, Rapporteur
- Howard Andrews, National Institute of Health
- Harry Blair, School of Medicine, University of Rochester
- John C. Bugher, Rockefeller Foundation
- Eugene P. Cronkite, Brookhaven National Laboratory
- Charles E. Dunlap, School of Medicine, University of Tulane
- Jacob Furth, Children's Cancer Research Foundation, Boston
- Webb Haymaker, Armed Forces Institute of Pathology
- Louis H. Hempelmann, School of Medicine, University of Rochester
- Samuel P. Hicks, New England Deaconess Hospital, Boston
- Henry S. Kaplan, Stanford University Medical School, San Francisco
- Sidney Madden, School of Medicine, University of California at Los Angeles
- R. W. Wager, Hanford Atomic Products Operation, General Electric Company

Subcommittee on Acute and Long Term Hematological Effects:
- Eugene P. Cronkite, Brookhaven National Laboratory, Chairman
- Carl V. Moore, Washington University School of Medicine
- William N. Valentine, University of California Medical Center
- Victor P. Bond, Brookhaven National Laboratory
- William Moloney, Boston City Hospital
- George V. LeRoy, Billings Hospital, University of Chicago
- George Brecher, National Institutes of Health
- James S. Nickson, Memorial Hospital, New York
- Consultants:
  - James Hartgering, Lt. Col. (MC) USA
  - Karl Tessmer, Lt. Col. (MC) USA, Walter Reed Army Medical Research Institute

Subcommittee on Toxicity of Internal Emitters:
- Austin M. Brues, Argonne National Laboratory, Chairman
- Thomas F. Dougherty, University of Utah
- Miriam P. Finkel, Argonne National Laboratory
- H. L. Friedell, Western Reserve University
- Harry A. Kornberg, General Electric Company, Richland, Wash.
- Kermit Larson, University of California, Los Angeles
- Wright Langham, Los Alamos Scientific Laboratory
- Hermann Lisco, Argonne National Laboratory
- William P. Norris, Argonne National Laboratory
- J. Newell Stannard, University of Rochester
- Joseph D. Teresi, Naval Radiological Defense Laboratory
- Raymond E. Zirkle, University of Chicago
- Consultants:
  - R. J. Hasterlik, University of Chicago
  - L. D. Marinelli, Argonne National Laboratory
  - Jack Schubert, Argonne National Laboratory
  - Charles L. Dunham, U. S. Atomic Energy Commission

Subcommittee on Acute and Chronic Effects of Radioactive Particles on the Respiratory Tract:
- Ralph W. Wager, Hanford Atomic Products Operation, General Electric Co., Chairman
- Stanton H. Cohn, U. S. Naval Radiological Defense Laboratory
- John W. Healy, Hanford Atomic Products Operation, General Electric Company
  - Francis R. Holden, Stanford Research Institute
- James K. Scott, University of Rochester
- J. N. Stannard, University of Rochester
- George V. Taplin, University of California School of Medicine
- Consultants:
  - Averill A. Liebow, Yale University School of Medicine
  - C. C. Gamertsfelder, ANP Department, General Electric Co.

Subcommittee on Permanent and Delayed Biological Effects of Ionizing Radiations From External Sources:
- Henry A. Blair, Department of Radiation Biology, Chairman
- George W. Casarett, Department of Radiation Biology
- Louis H. Hempelmann, Division of Experimental Radiology
- John B. Hursh, Department of Radiation Biology
- Marylou Ingram, Department of Radiation Biology
- Thomas R. Noonan, Department of Radiation Biology
- James K. Scott, Departments of Pharmacology and Pathology
- Lawrence W. Tuttle, Department of Radiation Biology

All of the above personnel are members of the faculty of the University of Rochester School of Medicine and Dentistry, Rochester, New York.

MEMBERSHIP OF THE COMMITTEE ON METEOROLOGICAL ASPECTS OF THE EFFECTS OF ATOMIC RADIATIONS

Dr. Harry Wexler, U. S. Weather Bureau, Chairman
Dr. Lester Machta, U. S. Weather Bureau, Rapporteur
Colonel B. G. Holzman, Hdqtrs., Air Research and Development Command
Lt. Colonel N. M. Lulejian, Hdqtrs., Air Research and Development Command
Dr. H. G. Houghton, Massachusetts Institute of Technology
Dr. W. W. Kellogg, the RAND Corporation
Dr. Heinz Lettau, Air Force Cambridge Research Center
Mr. Merril Eisenbud, U. S. Atomic Energy Commission
Dr. R. R. Braham, Jr., Institute of Atmospheric Physics
Mr. Charles E. Anderson, Geophysics Research Directorate, Bedford, Massachusetts
Dr. William K. Widger, Geophysics Research Directorate, Bedford, Massachusetts
Mr. R. J. List, U. S. Weather Bureau
Mr. D. Lee Harris, U. S. Weather Bureau
Consultants:
- Irving H. Blifford, Naval Research Laboratory
- Joshua Z. Holland, U. S. Atomic Energy Commission
- Donald H. Pack, U. S. Weather Bureau

MEMBERSHIP OF THE COMMITTEE ON OCEANOGRAPHY AND FISHERIES

Roger Revelle, Scripps Institution of Oceanography, Chairman
Howard Boroughs, University of Hawaii
Dayton E. Carritt, Johns Hopkins University
Walter A. Chipman, U. S. Department of the Interior, Fish and Wildlife Service
Harmon Craig, Scripps Institution of Oceanography
Lauren R. Donaldson, University of Washington
Richard H. Fleming, University of Washington
Richard F. Foster, General Electric Company, Richland, Washington
Edward D. Goldberg, Scripps Institution of Oceanography
John H. Harley, U. S. Atomic Energy Commission
Bostwick Ketchum, Woods Hole Oceanographic Institution
Louis A. Krumholz, American Museum of Natural History
Charles R. Renn, Johns Hopkins University
M. B. Schaeffer, Scripps Institution of Oceanography
Allyn C. Vine, Woods Hole Oceanographic Institution
Lional A. Walford, U. S. Department of the Interior, Fish and Wildlife Service
Warren S. Wooster, Scripps Institution of Oceanography
Consultants:
- Theodore Folsom, Scripps Institution of Oceanography
- Theodore Rice, U. S. Department of the Interior, Fish and Wildlife Service
- George A. Rounsefell, U. S. Department of the Interior, Fish and Wildlife Service
- Paul Thompson (Alternate for Dr. Walford), Fish and Wildlife Service

MEMBERSHIP OF THE COMMITTEE ON EFFECTS OF ATOMIC RADIATION ON AGRICULTURE AND FOOD SUPPLIES

A. G. Norman, University of Michigan, Chairman
C. L. Comar, Oak Ridge Institute of Nuclear Studies
George W. Irving, Jr., U. S. Department of Agriculture

James H. Jensen, Iowa State College
J. K. Loosli, Cornell University
Roy L. Lovvorn, North Carolina State College
Ralph B. March, University of California, Riverside
George L. McNew, Boyce Thompson Institute for Plant Research
Boy Overstreet, University of California, Berkeley
Kenneth B. Raper, University of Wisconsin
H. A. Rodenhiser, U. S. Department of Agriculture
W. Ralph Singleton, University of Virginia
Ralph G. H. Siu, Office of the Quartermaster General
G. Fred Somers, University of Delaware
George F. Stewart, University of California, Davis
Consultants:
A. J. Lehmann, Food and Drug Administration
Robert Somers, Meat Inspection Service, U. S. Department of Agriculture
J. Wolfe, U. S. Atomic Energy Commission

MEMBERSHIP OF THE COMMITTEE ON DISPOSAL AND DISPERSAL OF RADIOACTIVE WASTES

Abel Wolman, Johns Hopkins University, Chairman
J. A. Lieberman, U. S. Atomic Energy Commission, Rapporteur
F. L. Culler, Oak Ridge National Laboratory
A. E. Gorman, U. S. Atomic Energy Commission
L. P. Hatch, Brookhaven National Laboratory
H. H. Hess, Princeton University
C. W. Klassen, Illinois State Department of Public Health
Sidney Krasik, Westinghouse Atomic Power Division
H. M. Parker, General Electric Atomic Energy Project, Hanford
W. A. Patrick, Johns Hopkins University
S. T. Powell, Consulting Engineer, Baltimore
Leslie Silverman, Harvard University School of Public Health
Philip Sporn, American Gas and Electric Company, New York
Conrad P. Straub, Oak Ridge National Laboratory
C. V. Theis, U. S. Geological Survey
Forrest Western, U. S. Atomic Energy Commission
Consultants:
Paul C. Aebersold, U. S. Atomic Energy Commission
Karl Z. Morgan, Oak Ridge National Laboratory

Report of the Committee on Genetic Effects of Atomic Radiation

FOREWORD

The National Academy of Sciences, with the approval of the top Government authorities, is carrying out an over-all Study of the Biological Effects of Atomic Radiations. One part of that general study is being made by a Genetics Committee, and the present report is a preliminary one from that Committee.

This Genetics Committee has sixteen members, whose names and positions are listed at the beginning of this report. Thirteen of these have been directly and extensively concerned with research in genetics. This number includes specialists on the genetics of lower forms of life, on the genetics of such mammals as mice, on the more mathematical aspects of population genetics, and on human genetics. One member is specially experienced in the general biological effects of radiation, one in radiological physics, and one in pathology.

The problems of the Atomic Age affect every man, woman, and child—in fact, every living thing—in our country, and of course in the whole world as well. Although many of these problems are technical in character, it is nevertheless of importance to our democracy that these matters be as widely understood as possible. Therefore every effort has been made that this report be generally understandable.

This necessitates a certain amount of explanation of technical matters; but this report will use just as few unfamiliar terms as possible, and will define those that are used. It should be understood that many of the statements made in this report would require various qualifications and a lot more detail to attain full technical precision.

The subject is an inherently complicated one, and the reader must be prepared for a certain amount of detailed explanation, some of which is not easy to grasp.

It is felt that the subject is important enough so that many citizens will wish to make the effort which is necessary to a careful reading of this report.

The simplifications and abbreviations which have been adopted in this report in order to achieve a generally understandable presentation will undoubtedly be recognized by, and it is hoped will not disturb, the more technical reader. The later sections of the present report will be supplemented by more detail and factual justification if this is later desired by any of the agencies (as for example, the National Committee on Radiation Protection, the Atomic Energy Commission, governmental and industrial groups concerned with radiation hazards, etc.), which have responsibility for the procedures and standards to which our recommendations apply.

This particular report is preliminary for two reasons. First, we wish later to make a fuller report with more technical detail. Second, the situation is changing at such a rate that there should be a continuing series of reports, each bringing the subject up to date.

The National Academy study is not directed toward the problems posed by wartime use of atomic weapons, nor toward the political aspects of atomic power. The study is only indirectly concerned with the social and economic aspects. In fact, the National Academy study, as its title indicates, is concerned with the *possible biological hazards* due to atomic and other radiations. And the present report, made by the Genetics Committee, is concerned with the *genetic aspects* of the possible biological hazards. As this report is read, it should become progressively clearer what these genetic aspects are.

(I) WHAT ARE WE WORRIED ABOUT?

The coming of the Atomic Age has brought both hopes and fears. The hopes center largely around two aspects: the future availability of vast resources of energy; and the benefits to be gained in biology, medicine, agriculture, and other fields through application of the experimental techniques of atomic physics (isotopes, beams of high-energy particles, etc.).

Gains in both of these areas can be of great benefit to mankind. Advances in medicine and agriculture are obviously desirable. The wide availability of power can also be of great benefit, if we use this power wisely. For not only should there be enough power to meet the more obvious and mechanical demands, there should be enough to affect society in much more far-reaching and advantageous ways, so as to reduce world tensions by raising the economic standards of areas with more limited resources.

On the other hand, the Atomic Age also brings fears. The major fear is that of an unspeakably devastating atomic war. Along with this is another fear, minor as compared with total destruction but nevertheless with grave implications. When atomic bombs are tested, radioactive material is formed and released into the atmosphere, to be carried by the winds and eventually to settle down at distances which may be very great. Since it does finally settle down it has aptly been named "fall-out."

There has been much concern, and a good deal of rather loose public debate, about this fall-out and its possible dangers.

Are we harming ourselves; and are there genetic effects which will harm our children, and their descendants, through this radioactive dust that has been settling down on all of us? Are things going to be still worse when presently we have a lot of atomic power plants, more laboratories experimenting with atomic fission and fusion, and perhaps more and bigger weapons testing? Are there similar risks, due to other sources of radiation, but brought to our attention by these atomic risks?

(II) WHAT COMPLICATIONS ARE MET IN REACHING A DECISION?

Now it is a plain fact, which will be explained in some detail later in this report, that radiations,[1] penetrating the bodies of human beings, are genetically undesirable. Even very small amounts of radiation unquestionably have the power to injure the hereditary materials. Ought we take steps at once to reduce, or at least to limit, the amount of radiation which people receive?

There are two major difficulties that make it very hard to decide what is sensible to do. First, although the science of genetics is as precise and as

[1] Throughout this report the word "radiation" is not used in its broadest sense, but refers to certain kinds of high-energy radiations which are described in Section V.

advanced as any part of biology, it has in general, and particularly in human genetics, not yet advanced far enough so that it is possible to give at this time precise and definite answers to the questions: just *how* undesirable, *how* dangerous are the various levels of radiation; just *what* unfortunate results would occur?

Second, even if the relevant questions concerning radiation genetics could be answered definitively that would be only part of the story. The over-all judgment (how much radiation should we have?) involves a weighing of values and a balance of opposing aims in regard to some of which the techniques of physical and biological science offer little help.

What is involved is not an elimination of all risks, for that is impossible—it is a balance of opposed risks and of different sorts of benefits. And in disturbing and confusing thing is that mankind has to seek to balance the scale, *when the risk on neither side is completely visible.* The scientists cannot say with exact precision just what biological risks are involved in various levels and sorts of radiation exposure (these considerations being on one pan of the risk-scale); nor can anyone precisely evaluate the over-all considerations of national economic strength, of defense, and of international relations (all on the other pan of the scale).

(III) MUST WE THEN MOVE ENTIRELY IN THE DARK?

Does this mean that geneticists have, at the moment, nothing useful to say on this grave subject? Fortunately, this is not the case. We do know something, though not nearly enough to give definite answers to a great many important questions. There is a considerable margin of uncertainty about much of this, and as a result, there are naturally some differences of opinion among geneticists themselves as to exact numerical values, *although no disagreement as to fundamental conclusions.*

Many people, moreover, suppose science to be definite—open or shut. Things are supposed to be so or not so. And, therefore, some persons may, quite mistakenly, conclude that geneticists are unscientific because they do not completely agree on all details.

In relatively simple fields, where both theory and experiment have progressed far, a comforting kind of precision does often obtain. But it is characteristic of the present state of human radiation genetics that one must carefully and painstakingly note a lot of qualifications, of special and sometimes very technical conditions, of cautious reservations. The public should recognize that the attitudes and statements of geneticists about this problem of radiation damage have resulted from deep concern and from attempts to exercise due caution in a situation that is in essence complicaed and is of such great social importance.

It is not surprising that our knowledge of genetics—and especially human radiation genetics—is so fragmentary. What goes on inside cells and the effects of radiations on these processes are extremely complicated and subtle problems. To attack them successfully requires a tremendous lot of time; for the inherent variability of certain of these effects is such that to establish something with certainty one must do not one experiment but many thousands of individual tests and observations. To attack these problems also requires a high degree of special skill—and perhaps most of all, imaginative ideas which can be tested.

Single-celled organisms, as well as fruit flies and corn plants, have been specially rewarding objects of genetic study. In evolutionary terms, however, insects and plants are clearly a long way from man, and we are really just beginning to get genetic information about the effects of radiation on some of the lower mammals, such as mice. Even so, several matters of profound importance have already become clear: bacteria or fruit fly, mouse or man, the chemical nature of the hereditary material is universally the same; the main pattern of hereditary transmission of traits is the same for all forms of life reproducing sexually; and the nature of the effects of high-energy radiations upon the genetic material is likewise universally the same in principle. Hence, when it comes to human genetics, where the impossibilities of ordinary scientific experimentation are clear and only a tantalizing start has been made, we can at least feel certain of the general nature of the effects, and need only to discover ways in which to measure them precisely.

(IV) HOW COULD WE REDUCE RADIATION RISK?

The major ways to reduce our present and future exposures to radiations would be: a) to reduce medical and other use of Xrays as much as is feasible; b) to

set and to observe regulations for the proper construction and the same operation of nuclear power plants and for the methods used to dispose of their radioactive wastes as well as the methods used in mining and processing the fissionable materials; c) to reduce the testing of atomic weapons and hence to reduce radioactive fall-out; d) to place limits on the human exposures involved in certain aspects of experimentation in atomic and nuclear physics.

To carry out the steps just mentioned would, in greater or lesser degree for the various items, reduce radiation risks. Progress with regard to step a) can doubtless be achieved, although to go too far in reducing the medical use of Xrays would of course lead to the risk of poorer diagnosis and less effective treatment of disease. But to carry out steps b), c), and d) would subject us to a different set of risks. We might thereby impede progress in the nuclear field. We might seriously weaken our country's position in the world. We might deny future generations some of the possible benefits of nuclear power and of other atomic discoveries.

(V) RADIOACTIVE MATERIAL AND RADIATIONS

Now that the problem has been posed, and now that we are warned somewhat about the difficulties, we must begin to consider some of the more technical issues involved. What is radioactive material, what are radiations, and what biological effects do they have?

By *radioactive material* is meant those naturally occurring substances such as radium, or those man-produced atoms resulting from atomic experiments, which are inherently unstable. Instead of remaining unchanged like ordinary atoms of familiar substances such as oxygen, gold, etc., the atoms of these radioactive substances act like alarm clocks set by mischievous gremlins for unknown times. Unpredictably (at least in individual instances, but predictably for the average behavior of a large number) these atomic alarm clocks "go off"; that is to say, they disintegrate.

When radio material disintegrates it emits, along with other less penetrating and hence less significant rays, certain high-energy rays known as gamma rays. Some of these rays are entirely similar to a beam of light, except for the important distinction that they readily penetrate human tissue which is nearly opaque to ordinary light. Also the energy of these rays is much higher than that of light, and this enables them to produce chemical and biological changes in the tissue they traverse. Rays of this sort, which transport energy from one point in space to some other point, are in general referred to as *radiation*. We also class as radiations beams of minute particles travelling at high speeds—such as electrons or neutrons which when they hit matter produce effects like those of the radiation mentioned.

As indicated above, gamma rays are emitted by naturally occurring radioactive substances, such as radium. They are also emitted by the radioactive materials which are produced in the nuclear fission which occurs in atomic weapons testing, in nuclear power installations, and in various sorts of experimental installations. These same rays, in dilute amounts, impinge on and penetrate all of us all the time. For radioactive material is, as an inevitable and hence normal procedure, built into the soil, rocks, plants, etc., and for that matter is also built into our own bodies. Similarly, such material exists on the luminous dials of our watches and clocks. The familiar Xrays of the hospitals and tuberculosis clinics, and in the offices of dermatologists and dentists, have properties of penetration and energy which are similar to gamma rays.

Throughout this report, the word "radiation" refers primarily to gamma rays and/or x-rays, sometimes to other sorts of radiations as will be more particularly mentioned later.

Everyone knows what a pound of beefsteak is, or a yard of cloth. We do not have that sort of familiarity with amounts, or units, or dosages of radiation. X or gamma radiation is measured in units called roentgens (abbreviated r; for example, "a dose of 3r"). Dental Xrays *involve* a dose (to the reproductive organs or gonads, that being the important matter from the point of view of genetics) of about 0.005 r; and a general fluoroscopic examination may involve a dose of 2r or even more.

(VI) SOME BASIC FACTS ABOUT GENETICS

Before we ask what effect radiations have on genetic processes, we must review a little basic information about genetics itself.

Every cell of a person's body contains a great collection, passed down from the parents, the parents' parents, and so on back, of diverse hereditary units

called *genes.* These genes singly and in combination control our inherited characteristics.

These genes, as was just stated, exist in every cell of the body. But from the genetic point of view the ordinary "body cells," which make up the body as a whole, are not comparably as important as the "germ cells" which exist in the reproductive organs, and which play the essential roles in the production of children.

The genes are strung together, single-file, to form tiny threads of genetic material called *chromosomes,* which are visible under a microscope. These chromosomes, in ordinary body cells, customarily exist as similar but not identical pairs. Human body cells normally contain 48 chromosomes, these constituting two similar but not identical sets of 24 chromosomes each. One of these sets of 24 chromosomes was inherited from the mother, for the egg cell carries a set of 24 chromosomes; and the other set of 24 chromosomes was inherited from the father, for the sperm cell also carries a set of 24.

All the genes that a person starts out with when the original egg cell is fertilized are in general kept unchanged as the cells divide and the person's body is elaborated and maintained. The process by which the dividing cells duplicate the genes may not always produce perfect copies, but it does so in general. But genes do nevertheless essentially change. They are changed by certain agents, notably by heat, by some chemical, and *by radiation.* It is with the last of these three agents of gene change that we are concerned in this report.

When a gene becomes permanently altered, we say it *mutates.* The gene in its altered form is then duplicated in each subsequent cell division. If the mutant gene is in an ordinary body cell, then it is merely passed along to other body cells; but the mutant gene, under these circumstances, is not passed on to progeny, and the effect of the mutant gene is limited to the person in whom the mutation occurred.

However, it cannot safely be assumed that the effect is a negligible one on the person in whom the mutation occurred, nor can it properly be said that this effect is nongenetic, even though passage to offspring is not involved. For various kinds of cellular abnormalities are known to be perpetuated within an individual through body-cell divisions; so these effects are genetic in the broad sense.

What is involved here is not only mutant genes, but also larger scale disruption of the genetic material, such as breakage of chromosomes.

The quantitative relations are not yet clear, but it is established that certain malignancies such as leukemia, and certain other cellular abnormalities can be induced by ionizing radiations. There is also some evidence that effects of this sort measurably reduce the life expectancy of the individual receiving the radiation. These risks have genetic aspects and therefore should receive mention in this report. Indeed these direct risks to the individuals exposed may well constitute another adequate genetic reason for limiting radiation exposures to the lowest practicable levels.

To return to a consideration of the risks which are passed on to progeny, the mutant gene may exist in a sperm or an egg cell as a result of a mutation having occurred either in that cell or at some earlier cell stage. In this case, a child resulting from this sperm or egg will inherit the mutant gene.

If we were to take the two chromosomes of a similar pair, stretch them out straight, and put them alongside each other, then each gene of one would be opposite a corresponding gene in the other. Thus the genes exist in pairs, as do the chromosomes. The two members of each pair of genes are not always identically the same. That is, in fact, why we call the chromosome pairs *similar* rather than *identical.* The two genes of a corresponding pair play similar roles, in that they both affect or help to determine the same characteristic of the whole organism. But one of the two may have a somewhat different, or a much more powerful effect than the other.

Thus of a certain pair of genes, both might be concerned with hair color. If both genes of this hair-color pair are the sort which favor red hair, then the person has red hair. If both genes are the sort which favor non-red hair (black, brown, or blond) then the person has non-red hair. But suppose that, of this pair of hair-color genes, one favors red hair and the other non-red hair. What happens then?

The answer (husbands and wives will understand this) is that one of the two usually dominates the situation and gets its way, although (and again this seems reasonable) the meeker one of the two usually manages to avoid being completely ignored.

Thus with one non-red gene (this being the powerful and dominant one of the two), and one red gene (this being the meeker one), the hair is ordinarily not red, but the red gene may nevertheless produce some effect, a little red showing in the hair so as to make it faintly rusty or tawny in color.[3]

The powerful type of gene, which gets all or most of its own way in contrast to its companion gene, is very naturally called a *dominant* gene. The less effective type is called a *recessive* gene. In this same terminology, non-red hair color is called a *dominant characteristic*, whereas red hair color is called a *recessive characteristic*. A recessive characteristic actually fully appears only if both of the relevant genes are of the recessive type. Of great importance for our present study is the fact that *mutant genes*—genes which have, for example, been changed by radiations—*are usually of the recessive type*.

It is now easy to see that any organism may have, latent in its genetic constitution, ineffectual or recessive genes that have not had much of a chance to become apparent in its developed external characteristics, since the recessive genes are masked by their dominant companion genes. Yet often, as we have seen, this dominance is incomplete and recessive gene is able to manifest itself partially.

When the two genes of a pair are alike (both recessive or both dominant) then they are called a *homozygous* pair; but when one is recessive and the other dominant, then the pair is called *heterozygous*. Thus a recessive characteristic (like red hair) can be fully expressed only when the corresponding gene pair is homozygous.

(VII) RADIATIONS AND GENETIC MUTATIONS

We are now in a position to indicate why it is that radiations, such as X-rays or gamma rays, can be so serious from the genetic point of view. For although the genes, as described above, normally remain unchanged as they multiply and are passed on from generation to generation, they do very rarely change, or *mutate;* and *radiation*, as we have already mentioned, *can give rise to such changes or mutations in the genes*. The change is presumably an alteration in the complicated chemical nature of the gene, and the energy furnished by the radiation is what produces the chemical change. Mutation ordinarily affects each gene independently; and once changed, an altered gene then persists from generation to generation in its new or mutant form.

Moreover, the mutant genes, in the vast majority of cases, and in all the species so far studied, lead to some kind of harmful effect. In extreme cases the harmful effect is death itself, or loss of the ability to produce offspring, or some other serious abnormality. What in a way is of even greater ultimate importance, since they affect so many more persons, are those cases that involve much smaller handicaps, which might tend to shorten life, reduce number of children, or be otherwise detrimental.

The changed character, due to the mutated gene, seldom appears fully expressed in the first generation of offspring of the person who received the radiation and thus had one of his genes mutated. For these mutant genes are usually recessive. If a child gets from one parent a mutant gene, but from the other parent a normal gene belonging to that pair, then the normal gene is very likely to be at least partially dominant, so that the normal characteristic will appear.

But this is not all of the story. For, like the red-hair gene, the harmful recessive mutant genes are not usually completely masked. Even when paired with a normal and dominant gene, that is to say even when in the heterozygous state, they still have some detrimental effect. This "heterozygous damage" is ordinarily much smaller than the full expression of the mutant when in the homozygous state, and yet there may be a significant shortening of the length of life or reduction of the fertility of the heterozygous carriers of the mutant. And the risk of heterozygous damage *applies to many more individuals*, indeed to every single descendant who receives the gene.

The relations of genes to ordinary traits (not to the most simply determined biochemical traits (are of course much more complex than the previous paragraph would seem to imply. Such gene-determined traits may vary from person to person, due perhaps to environmental differences, and often may not even appear at all. A single gene usually affects several such characters, and char-

[3] The accurate and complete genetic story about red hair is more complicated than has been stated here. There are less familiar characteristics—thalassemia and sickle cell anemia for example—which more strictly conform to the simple pattern here described.

acters are practically always affected by many genes. Also the effect of a gene may depend on what other genes are present, often in a complex way. For example, a mutation tending to increase weight might be harmful to certain persons, but beneficial to others.

Indeed it is likely that a large fraction of the genes that determine normal variability are of this rather ambiguous type that are sometimes deleterious, sometimes not. Mutations within this sort would not necessarily be harmful. Such mutations presumably occur, but geneticists do not know what fraction of all mutations are of this type, for they are not ordinarily detectable. However, the mutations that form the basis of this report are those that are relatively detectable, and these, as mentioned earlier, are almost always harmful.

Individuals bearing harmful mutations are handicapped relative to the rest of the population in the following ways: they tend to have fewer children, or to die earlier. And hence such genes are eventually eliminated—soon if they do great harm, more slowly if only slightly harmful. A mildly deleterious gene may eventually do just as much total damage as a grossly and abruptly harmful one, since the milder mutant persists longer and has a chance to harm more people.

In assessing the harm done to a population by deleterious genes, it is clear that society would ordinarily consider the death of an early embryo to be of much less consequence than that of a child or young adult. Similarly a mutation that decreases the life expectancy by a few months is clearly less to be feared than one that in addition causes its bearer severe pain, unhappiness, or illness throughout his life. Perhaps most obviously tangible are the instances, even though they be relatively uncommon in which a child is born with some tragic handicap of genetic origin.

A discussion of genetic damage necessarily involves, on the one hand, certain tangible and imminent dangers, certain tragedies which might occur to our own children or grandchildren; and on the other hand certain more remote trouble that may be experienced by very large numbers of persons in the far distant future.

No two persons are likely to weigh exactly alike these two sorts of danger. How does one compare the present fact of a seriously handicapped child with the possibility that large numbers of persons may experience much more minor handicaps, a hundred or more generations from now?

There are thoughtful and sensitive persons who think that our present society should try to meet its more immediate problems, and not worry too much about the long-range future. This viewpoint is in some instances supported by the belief that new ways, perhaps unimaginable at the moment, are likely eventually to be found for meeting problems.

There are other thoughtful and conscientious persons who think that we are specifically responsible for guarding, as well as we can now determine, the long future.

Recognizing the inevitability and propriety of both viewpoints, and recognizing that they lead different persons to express their concerns through different examples and with differing emphases, the fact of major importance for this present study is that, travelling by different routes, different geneticists arrive at the same conclusion: *Complexities notwithstanding, the genetic damage done, however felt and however measured, is roughly proportional to the total mutation rate.*

(VIII) MUTANT GENES AND EVOLUTION

Many will be puzzled about the statement that practically all known mutant genes are harmful. For mutations are a necessary part of the process of evolution. How can a good effect—evolution to higher forms of life—result from mutations practically all of which are harmful?

First of all, it is not mutations which, of themselves, produce evolution, but rather the action of natural selection on whatever combinations of genes occur. Much of evolutionary progress probably depends on changes within the range of normal variability, and thus depends on genes of very small effect, and of the type mentioned in the previous section which are favorable or unfavorable depending on what other genes are present. Thus evolution consists of a complex shifting of frequencies of such genes, accompanied by the continuous process of elimination of detrimental mutations and the occasional incorporation into the population of a favorable mutation.

Nature had to be rather ruthless about this process. Many thousands of unfortunate mutations, with their resulting handicaps, were tolerated, just so

long as an advantageous mutation could be utilized, once in a long while, for inching the race up slightly higher to a better adjustment to the existing conditions. The rare creature with an advantageous combination of genes was better fitted to survive and displace his less favored companions, and thus evolution was served, even though there were thousands of tragedies for every success.

The reader may be troubled by a second difficulty. If mutation results in at least some favorable types, and if these are building blocks of evolution, why is an increase in mutation rate regarded as undesirable? Why wouldn't an increase in mutation rate produce a larger total number of the favorable types and so speed up evolution? If the favorable types are normally quite rare, wouldn't it almost seem that increasing the mutation rate would be desirable? The answer to this question lies in the consideration that the bad effects of mutation must be balanced against the good. Some mutation is necessary for evolution, but if the mutation rate is too high, the unfavorable mutations will be so numerous that the species and its future evolution will be handicapped. Under present-day conditions of living and medical care, it seems unlikely that the unfavorable results of mutation are being eliminated nearly as rapidly as was formerly the case. In other words, one of the consequences of the amazing mastery of his environment which man has achieved has been an actual decrease in the severity of natural selection.

Geneticists in fact believe that although favorable mutations are rare compared with unfavorable ones, the human population probably already has, and will continue to have as a result of its present mutation rate and without additional mutations from increased radiation, a large enough total supply of favorable, partially favorable, and potentially favorable mutations. In other words, with our present mutation rate we shall continue to have a degree of genetic variability adequate for further evolution.

(IX) WHAT, THEN, CAN GENETICISTS SAY TO HELP RESOLVE OUR PROBLEM?

With the background furnished by the preceding discussion, we can now state rather concisely certain main points on which geneticists are in substantial agreement. Some of these points will partially repeat statements already made, but they are included here in order that this section be reasonably complete of itself.

(1) Radiations cause mutations.

Mutations affect those hereditary traits which a person passes on to his children and to subsequent generations.

(2) Practically all radiation-induced mutations which have effects large enough to be detected are harmful.

A small but not negligible part of this harm would appear in the first generation of the offspring of the person who received the radiation. Most of the harm, however, would remain unnoticed, for a shorter or longer time, in the genetic constitution of the successive generations of offspring. But the harm would persist, and some of it would be expressed in each generation. On the average, a detrimental mutation, no matter how small its harmful effect, will in the long run tip the scales against some descendant who carries this mutation, causing his premature death or his failure to produce the normal number of offspring.

Although many mutations do disturb normal embryonic growth, it is not correct that all, or even that most mutations, commonly result in monstrosities or freaks. In fact, the commonest mutations are those with the smallest direct effect on any one generation—the slight detrimentals.

(3) Any radiation dose, however small, can induce some mutations. There is no minimum amount of radiation dose, that is, which must be exceeded before any harmful mutations occur.

(4) For every living thing—bacterium, fruit fly, corn plant, mouse, or man—there exist mutations which arise from natural causes (cosmic rays, naturally occurring radiations from radium and similar substances, and also from heat and certain chemicals). These naturally occurring, and hence unavoidable, mutations are usually called "spontaneous mutations."

Like radiation-induced mutations, nearly all spontaneous mutations with detectable effects are harmful. Hence these mutations tend to eliminate themselves from the population through the handicaps or the tragedies which occur because the persons bearing these mutants are not ideally fitted to survive.

We all carry a supply of these spontaneous mutant genes. The size of

this supply represents a balance between the tendency of mutant genes to eliminate themselves, and the tendency of new mutants to be constantly produced through natural causes.

(5) Additional radiation (that is, radiation over and above the irreducible minimum due to natural causes) produces additional mutations (over and above the spontaneous mutations). The probable number of additional induced mutations occurring in an individual over a period of time is by and large proportional to the total dose of extra radiation received, over that period, by the reproductive organs where the germ cells are formed and stored. To the best of our present knowledge, if we increase the radiation by X%, the gene mutations caused by radiation will also be increased by X%.

The *total dose* of radiation is what counts, this statement being based on the fact that the genetic damage done by radiation is *cumulative.*

A larger amount of radiation produces a larger number of mutations. But within the limits of the radiation doses being considered in this report there is every reason to expect that these additional mutants would be of the same general sort as those produced by the natural background radiation. That is to say, mildly larger doses of radiation would produce *more*, but not *worse*, mutants.

(6) From the above five statements a very important conclusion results. It has sometimes been thought that there may be a *rate* (say, so much per week) at which a person can receive radiation with reasonable safety as regards certain types of direct damage to his own person. But the concept of a *safe rate* of radiation simply does not make sense if one is concerned with genetic damage to future generations. What counts, from the point of view of genetic damage, is not the rate; it is the *total accumulated dose to the reproductive cells of the individual from the beginning of his life up to the time the child is conceived.*

What is genetically important to a child is the total radiation dose that child's parents have received from their conception to the conception of the child. Since this report necessarily deals with averages, the significant total dose period should be, at least approximately, the number of years that normally elapses from the conception of a person to the average time at which offspring are conceived. In the United States, based on 1950 data, the average age of fathers at the births of all children is 30.5 years, whereas the average age of both parents is 28.0 years. It therefore seems sensible for us to use the round figure of 30 years, especially since this figure is the one usually chosen to measure a generation. Using this 30-year figure for characterizing the "total reproductive life radiation dose" would have the result that about half of the total offspring would receive the possible effects of a smaller, and about half the possible effects of a larger, radiation dose.

(7) The problems of defining and estimating genetic damage are very difficult ones.

There are at least three different aspects which must be considered. The first aspect places emphasis on the risk to the direct offspring and later descendants of those persons who, from occupational hazard or otherwise, receive a radiation dose substantially greater than the average received by the population as a whole.

The second aspect refers to the effect of the *average* dose on the population as a whole.

The third aspect refers in still broader terms to the possibility that increased and prolonged radiation might so raise the death rate and so lower the birth rate that the population, considered as a whole, would decline and eventually perish. We are at present extremely uncertain as to the level of this fatal threshold for a human population. This is one reason why we must be cautious about increasing the total amount of radiation to which the entire population is exposed.

These three approaches to the problem of genetic damage involve estimating the damage in successive generations and also the total damage in all generations, due to an increase in the amount of mutation. The relative emphasis one places on these three aspects depends in part on whether one thinks primarily in terms of distress to individual persons, or whether one thinks in terms of the population as a whole. Necessarily involved is the contrast between manifest harm to a few, and less evident but no less unreal harm to many. Also involved is the contrast between a more short-term and a more long-range point of view.

One way of thinking about this problem of genetic damage is to assume that all kinds of mutations on the average produce equivalent damage, whether as

a drastic effect on one individual who leaves no descendants because of this damage, or a wider effect on many. Under this view, the total damage is measured by the number of mutations induced by a given increase in radiation, this number to be multiplied in one's mind by the average damage from a typical mutation.

Measuring total damage in terms of the number of mutations does indeed necessarily involve this concept of the average damage from a typical mutation, and some geneticists find this concept difficult and illusive. They would point out that mutations may be grouped in classes that differ, on a subjective scale, many thousand-fold in the amount of damage per mutation. As examples they would cite a mutation which results in very early death of an embryo (which might cause very little social or personal distress), and a mutation which results in severe malformation to a surviving child (which would cause very great personal distress and which clearly involves a social burden).

Rather than utilizing this concept of the average total damage per mutation, some geneticists prefer to start with a consideration of the tangible damage which occurs now, as a result of the current rate of mutation and get an index of damage by multiplying this by the ratio of the expected new mutation rate to the current one. This procedure, however, admittedly deals with only *part* of the total damage; so an alternative difficulty faces those who prefer this procedure, namely the difficulty of estimating what part of the total damage they have dealt with.

As an illustration of the first aspect, suppose that ten thousand individuals were exposed to a large dose of radiation, of the order of 200 r. Then perhaps one hundred of the children of these exposed individuals would be substantially handicapped, this being in addition to the number handicapped from other causes. In this case the connection with the radiation exposure could be established by a statistical study.

As an illustration of the second aspect, suppose the whole population of the United States received a small dose of extra radiation, say 1 r. Then there is good reason to think that, among a hundred million children born to these exposed parents, there would be several thousand who would be definitely handicapped because of the mutant genes due to the radiation. But these several thousand handicapped children might be, so to speak, lost in the crowd. Society might be more impressed by the one hundred more obvious cases of the preceding paragraph than by the more hidden several thousand cases of this paragraph.

We should not disregard a danger simply because we cannot measure it accurately, nor underestimate it simply because it has aspects which appeal in differing degrees to different persons. Two conclusions seem to be clear and of importance: We should proceed with due caution as regards all agents which cause mutations; and we should vigorously pursue the researches which will in time give us a more precise way of judging all aspects of the risk.

(X) SOME REMARKS ABOUT APPROXIMATE ESTIMATES

Up to this point of the discussion the conclusions of the geneticist are pretty clear; the mutant genes induced by radiation are generally harmful, and the harm cannot be escaped.

But as yet this report has not furnished much of a basis for converting these conclusions into practical advice. Remembering that we must eventually balance risk against risk, it is obviously desirable to try to learn, as definitely as circumstances permit, the answer to the question: *how* great would be the genetic harm done by various doses of radiation?

Section XII of this report will respond to this question. But before giving the various replies, there should be some preliminary explanation concerning the nature of the answers given.

Science, and particularly the branch which deals with the physical world about us, has succeeded in giving highly precise answers to many questions. When one talks about the velocity of light he does not need to say that it is *something* like three hundred thousand kilometers per second: he is justified in saying that it *is* 299,793 km. per second, and that the final integer is almost certainly not off by more than two units.

But when you ask an experienced surgeon what your chances are of surviving a serious operation, and if he answers "something like nine chances out of

ten," then you accept that as a reasonable and helpful estimate. You do not distrust him because he gives you a rought estimate. Indeed you would have good cause to distrust him if he tried to give a highly precise answer.

In other words, there are many situations in which science can give only rough estimates. These estimates can nevertheless be very useful. No one should disdain such an estimate because it is rough, nor should anyone consider such estimates unscientific.

In Section XII there will be stated the results of certain approximate calculations. The theory behind these calculations is on the whole well understood; but it is seldom the case that one knows with much accuracy the numerical values that enter into the calculations. One may, for example, say, "I don't know, in any direct measured sense, how many mutants would result if all the genes in a human fertilized cell received one roentgen of radiation. But using a pretty definitely known value for the mutation rate in certain genes of the mouse; and also knowing fairly well (in this case from experiments with fruit flies) how to pass from the measured rate for a few genes to the rate which probably applies to a germ cell as a whole; and then making the unfortunate but necessary assumption that these mouse and fruit fly figures apply reasonably well to man—using this procedure I come out with estimates for the number of mutants which would be produced in man by a given dose of radiation. Because of the uncertainties, I think it prudent to state not a single final result, but rather a range of result with estimated lower and upper limits. I wish that we had direct experimental evidence which would firm up this estimate. But I don't have to be too apologetic, for a large amount of biological reasoning has been successfully based on this sort of procedure. Man differs widely from lower forms of life in all the obvious, and in many other, respects. But the fundamental processes inside cells tend to be curiously alike, from the simplest creature of a single cell, up to man."

It may turn out that the uncertainties in the quantities which enter the calculation are so great that the resulting uncertainty in the final answer is itself so very broad that the calculation simply does not furnish a useful estimate. But it may also turn out that, despite some considerable uncertainty in the constituent factors, the answer can be stated with a range of uncertainty which is small enough so that the estimate is useful.

It seems necessary to emphasize this matter of approximate estimation, so that no one will improperly conclude that a statement is unreliable because it involves a range of values. On the contrary, such a statement, when made in a situation like the present one, should be viewed as all the more dependable precisely because it does not pretend to an unwarranted accuracy.

(XI) HOW MUCH RADIATION ARE WE NOW RECEIVING?

If we are to talk about how harmful certain radiation doses may be, we should gain some idea of the amount of radiation we are already receiving from various sources.

The Committee will release a report specially devoted to this particular subject, which summarizes in detail all the kinds, sources, and amounts of radiation. In the present report, only that minimum amount of information will be given which is necessary for our current discussion.

Neglecting several minor contributions (all of which will be treated in the longer report), man is at present receiving radiations from the following:

(1) *Background Radiation*

This is the radiation which results from natural causes (cosmic rays, naturally occurring radium, etc.) not under our control. Each person receives on the average a total accumulated dose of about 4.3 roentgens over a 30 year period. At high altitudes this dose is greater, because of the increase of cosmic rays. Thus this background is as high as 5.5 r in some places in the United States.

(2) *Medical X Rays*

According to present estimates, each person in the United States receives, on the average, a total accumulated dose to the gonads which is about 3 roentgens of X-radiation during a 30 year period. Of course, some persons get none at all; others may get a good deal more.

(3) Fall-out from Weapons Testing

The Atomic Energy Commission[8] is doing a technically competent and a socially conscientious job of measuring fall-out: but it does not follow from this that one can answer, with high precision, all questions about the biological risks involved. What they usually measure (which, technically speaking, is a beta-ray activity in air) has to be translated over into what is genetically important (namely, the gamma ray dose to the gonads). The estimation of the latter of these quantities from the former is a pretty complicated business.

Besides those just mentioned, there are certain further uncertainties in the fall-out values. The measurements are necessarily taken far apart, and there is known to be considerable local variation due to meteorological conditions and topography. The radioactive dust, when it settles out of the air, is subject to weathering, as when it is washed off of buildings by the rain and carried to locations where it may affect fewer persons. Also individuals inside houses, or other shelters, will be considerably less exposed than those in the open air

Thus one cannot expect the figures on fall-out to be very precise ones. We have been informed that the AEC scientists are confident that the actual true dose figures are less than five times their stated estimates, and are also greater than one-fifth of these stated estimates.

It should be noted that the figures on fall-out as stated by the Atomic Energy Commission make only a conservative correction for weathering and shelter; and thus their figures, at least in regard to this point, tend to overstate the danger rather than the opposite.

With these understandings, it may be stated that U. S. residents have, on the average, been receiving from fall-out over the past five years a dose which, if weapons testing were continued at the same rate, is estimated to produce a total 30-year dose of about *one tenth of a roentgen;* and since the accuracy involved is probably not better than a factor of five, one could better say that the 30-year dose from weapons testing if maintained at the past level would probably be larger than 0.02 roentgens and smaller than 0.50 roentgens.

The rate of fall-out over the past five years has not been uniform. If weapons testing were, in the future, continued at the largest rate which has so far occurred (in 1953 and 1955) then the 30-year fall-out dose would be about twice that stated above. The dose from fall-out is roughly proportional to the number of equal sized weapons exploded in air, so that a doubling of the test rate might be expected to double the fall-out.

The figures just stated are based on all information now available from both the Atomic Energy Commission and the Armed Forces, and have been estimated as part of a study carried out for this Committee by Dr. John S. Laughlin, Chief of the Division of Physics and Biophysics, Sloan-Kettering Institute, and Dr. Ira Pullman, loaned to this study by the Nuclear Development Corporation of America. In their estimation correction has been made for weathering and shelter effects in accordance with the latest experimental data.

(4) Atomic Power Plants

As yet the general population has not received radiation from atomic power plants or from the disposal of radioactive wastes. These are future sources of radiation that might become dangerous.

(5) Occupational Hazards

The preceding four points apply to everyone. Unless proper precautions are taken, persons who are close to equipment emitting X rays who are engaged in experimental work in atomic energy, who operate atomic plants, who test weapons, who mine or otherwise handle radioactive material, etc., are subject to the risk of greater radiation exposure during their work.

(XII) HOW HARMFUL ARE RADIATION-INDUCED MUTATIONS?

As has already been indicated, there are various ways of estimating genetic harm, various attitudes which can be taken as to what is most serious and significant. But this situation should not be allowed to confuse or conceal the massive fact that, by whatever chain of argument or reasoning, all geneticists come out with the same basic conclusions.

[8] Under the Department of Defense other measurements, relating to fall-out, are also being made.

(A) Thus the first and unanimous reply to the question posed by the title to this section is simply this: *Any radiation is genetically undesirable*, since any radiation induces harmful mutations. Further, all presently available scientific information leads to the conclusion that *the genetic harm is proportional to the total dose* (that is, the toal accumulated dose to the reproductive cells from the conception of the parents to the conception of the child). This tells us that a radiation dose of 2X must be presumed to be twice as harmful as a radiation dose of X; but it still doesn't tell us the amount of harm we would be doubling.

(B) Second, we remember that mankind has for ages been experiencing, as the so-called spontaneous mutations, a certain rate of (generally harmful) mutations due to natural and uncontrolled causes (cosmic rays, heat, chemicals, etc.). It is not entirely unnatural to think of this burden of mutations as a sort of "normal" burden on society.[4] Therefore it seems to be illuminating to ask: how much additional "man-made" radiation will it take before this "natural" amount of genetic mutation (to which we are at least in some senses adjusted) will be doubled?

The calculations which lead to an estimate of this "doubling dose" necessarily involve the rates of both spontaneous and radiation-induced mutations in man. Neither of these rates has been directly measured; and the best one can do is to use the excellent information on such lower forms as fruit flies, the emerging information for mice, the few sparse data we have for man—and then use the kind of biological judgment which has, after all, been so generally successful in interrelating the properties of forms of life which superficially appear so unlike but which turn out to be so remarkably similar in their basic aspects.

In view of the inevitable uncertainties, it is rather surprising that the final estimates, as made by numerous specialists of this Committee and in other countries, do not differ more than they do. The lowest figure which has been responsibly brought forward for the doubling dose is 5 r, and the largest estimates range up to 150 r or even higher. Recent work with mice (which are, after all, mammals) gives some basis for thinking that the doubling dose is not as high as 150 r. The experience in Japan gives some basis for thinking that the doubling dose is larger than 5 r. Indeed it is clear that the doubling dose must be at least as large as the background radiation (which is between 4 and 5 r, over 30 years, in the United States). This, in fact, would be the value of the doubling dose if spontaneous mutations were due to background radiation alone, heat and chemical agents making no contribution.

Thus various arguments reduce the 5–150 r range, and several experienced geneticists have recently made estimates in the narrower range of 30 r to 80 r.

In summary then of this particular point: Each individual, on the average inevitably experiences during his reproductive lifetime a certain number of harmful spontaneous mutations from natural causes. He would experience an additional *equal number* of harmful mutations if he received a certain dose of radiation during that same period. This is known as the "doubling dose." The actual value of the doubling dose is almost surely more than 5 r and less than 150 r. It may very well be from 30 r to 80 r.

The first portion of this (Section XII) said that twice as much radiation gives twice as much harm. This second portion goes a bit further. It says that something like 30 r to 80 r (or at a further extreme, 5 r to 150 r) of extra radiation dose would do mankind twice the harm it is now experiencing from spontaneous mutations.

(C) The two preceding portions of this Section are clearly not really satisfying. They do indicate in quantitative terms how increases in radiation increase the harm. But anyone still wants to know in more specific terms, if possible, *how serious* is this harm that we may be doubling. If city traffic increases until the risk of crossing the street is doubled, then we will presumably still cross the street; for the risk per crossing is, after all, a very small one. If highway traffic increases until the risk in taking a thousand-mile drive is doubled, then many persons might well hesitate, for the risk is now unpleasantly high.

And this is the point at which it becomes most clearly evident that different geneticists find meaningful rather different approaches to the problem of genetic damage.

As has been stated previously, from one point of view the best index of genetic damage is the totality of tangible genetic defects of living individuals—

[4] There is some basis for hoping that we may eventually be able to control at least a part of both spontaneous and radiation-induced mutations.

say such things as mental defects, epilepsy, congenital malformations, neuromuscular defects, hematological and endocrine defects, defects in vision or hearing, cutaneous and skeletal defects, or defects in the gastro-intestinal or genitourinary tracts. Roughly 4–5% of all live births in the United States have defects of this sort; and of all of these, perhaps about half—or 2% of the total live births—have simple genetic origin and appear prior to sexual maturity.

If mankind were subjected to a "doubling dose" of radiation, then the present level of 2% of such genetic defects would rise, and would eventually be doubled. More explicitly, consider the next one hundred million births in the United States. This is about the number of children that will, in the future, be born to the presently alive population of the United States. Of these 100,000,000 children, something like 2,000,000 will experience genetic defects of the sort listed, these resulting from the deleterious "spontaneous" mutant genes which have been induced by natural causes excluding man-made radiation. If we were to be subjected, generation after generation, to an additional doubling dose of man-made radiation, then this present tragic figure of 2,000,000 would gradually increase by 2,000,000 more cases, up to an eventual new total of 4,000,000. It would, to be sure, take a very long time to reach this equilibrium double value. Perhaps 10% of the increase, or 200,000 new instances of tangible inherited defect, would occur in the first generation.

Since at various places this report considers a radiation dose of 10 r, it may be useful to state the tangible inherited defects from a dose of that size. A dose of 10 r would, on the above basis, give rise to some 50,000 new instances of tangible inherited defects in the first generation, and about 500,000 per generation ultimately, assuming of course an indefinite continuation of the 10 r increased rate and also assuming a stationary population.

These figures by no means measure *all* of the genetic damage that would result from a doubling dose; but they do make tangible and impressive the fact that a doubling dose of radiation would cause real personal and social distress.

(D) There is another way of looking at this problem of genetic damage, and that consists of trying to make some useful sort of really long-term, fully complete estimate. This consists of estimating the total number of mutant genes which would be induced in the whole present population of the United States and passed on to the next appearing 100,000,000 children, were this whole population to receive a certain total radiation dose to the gonads. In this instance we will use a dose of 10 r, since a dose of that magnitude appears later in this report in the recommendations. Having estimated this total number of transmitted mutants induced by a dose of 10 r, one then can only say, when he wishes to translate this over into harm or damage, that each one of these mutants must eventually be extinguished out of the population through tragedy. This statement does, of course, not hold in the detailed sense that one thinks of tracing each individual mutant gene until the line which bears and transmits it is overcome by the accumulating handicaps it imposes. The statement holds only in a statistical sense. Some lines of mutant gene will die out merely through normal chance procedures of inheritance. Others will multiply through these same chance procedures. But these normal chance effects cancel out; and the *statistical extinction* of the mutant genes is accomplished only through tragedy.

Concerning these estimates of total number of mutants, three things should be said. First, they are clearly not really satisfactory to any genetecist. Too much has to be assumed, too little is dependably known.

Second, this kind of estimate is not a meaningful one to certain geneticists. Their principal reservation is doubtless a feeling that, hard as it is to estimate numbers of mutants, it is much harder still, at the present state of knowledge, to translate this over into a recognizable statement of harm to individual persons. Also they recognize that there is a risk involved in extrapolating from mouse and Drosophila data to the human case.

Various remarks can, however, fairly be made in favor of this estimating attempt. Two largely independent methods lead to about the same results, and this increases one's confidence. Although the extreme ranges of the estimates differ widely, the mean estimate for any one geneticist is not very different from the mean for any other. Even the "guessing" which is involved hardly deserves that name, for it is based on long years of experience.

So that the final thing that should be said is that in spite of all the difficulties and complications and ranges in numerical estimates, the result is nevertheless very sobering.

Six of the geneticists of this committee considered the following problem: suppose the whole population of the United States received one dose of 10 roentgens of radiation to the gonads. What is the estimate of the total number of mutants which would be induced by this radiation dose and passed on to the next total generation of about one hundred million children? Each geneticist calculated what he considered to be the most probable estimate, and then bracketed this by his minimum and maximum estimates. Each thus said, in effect: "I feel reasonably confident that the true value is greater than my minimum estimate and less than my maximum. My best judgment, as stated in a single figure, is what I have labelled the most probable estimate."

The most probable estimates as thus calculated by the six geneticists do not differ widely. They bunch rather closely around the figure 5,000,000. Four of the six estimates are very close to that figure, and the other two differ only by a factor of 2.

These six geneticists concluded, moreover, that the uncertainty in their estimation of the most probable value was about a factor of 10. That is to say, their minimum estimates were about 1/10, and their maximum estimates about 10 times the most probable estimate.

This calculation assumes a stable value for the total population. This calculation is admittedly somewhat complicated and disappointingly vague. It is, to some geneticists, not a very meaningful way of looking at the problem. To others it adds up to something at least reasonably clear, and in any event very serious.

(XIII) FALL-OUT

There has been concern about the possible genetic harm due to the fall-out of radioactive material which results from the testing of atomic weapons. Certain aspects of this problem will be discussed in the reports of the other committees of this study (fall-out on grazing and cropland; fall-out in the sea and possible concentration in marine organisms; the distribution of fall-out material by the winds and in the upper atmosphere; possible pathological damage due to long-lived isotopes built into our bones; etc.). The present comments relate only to the question of genetic damage.

From the point of view of this Committee there are two summary remarks that should be made. First, since *any* additional radiation is genetically undesirable the fall-out dose is genetically undesirable.

Second, the fall-out dose to date (and its continuing value if it is assumed that the weapons testing program will not be substantially increased) is a small one as compared with the background radiation, or as compared with the average exposure in the United States to medical X rays.

(XIV) RECOMMENDATIONS

In light of the considerations which have been reviewed by this Committee, and which have been, at least in major outline, summarized in this report, this Committee has several recommendations.

These recommendations should all be interpreted in the light of the basic fact that *any* additional radiation is genetically undesirable. Therefore our society should hold additional radiation exposure as low as it possibly can. If certain figures (such as 10 roentgens) occur in a recommendation, it should most emphatically not be assumed that any exposure less than that figure is, so to speak, "all right": nor should it be for a moment assumed that disaster will suddenly descend if one of these figures is exceeded.

In any case in which a figure is stated, it is with the idea: stay just as far under this as you can; do not consider that this is an amount of radiation which is genetically harmless, for there is no such figure other than zero.

Opposing the fact that any further radiation is genetically bad is the practical fact that further radiation, from certain sources at least, is probably inevitable. The factors which argue for an increase in radiation are not genetic, and should obviously be appraised by a group much more representative than this Committee. Thus our recommendations will have to be evaluated by others, who must decide what decisions society should or must make. As geneticists we say: *keep the dose as low as you can.*

Thus we recommend:

(A) That, in view of the fact that total accumulated dose is the genetically important figure, steps be taken to institute a national system of radiation exposure record-keeping, under which there would be maintained for every individual a complete history of his total record of exposure to X rays, and to all other gamma radiation. This will impose minor burdens on all individuals of our society, but it will, as a compensation, be a real protection to them. We are conscious of the fact that this recommendation will not be simple to put into effect.

(B) That the medical authorities of this country initiate a vigorous movement to reduce the radiation exposure from X rays to the lowest limit consistent with medical necessity; and in particular that they take steps to assure that proper safeguards always be taken to minimize the radiation dose to the reproductive cells.

(C) That for the present it be accepted as a uniform national standard that X-ray installations (medical and nonmedical), power installations, disposal of radioactive wastes, experimental installations, testing of weapons, and all other humanly controllable sources of radiations be so restricted that members of our general population shall not receive from such sources an average of more than 10 roentgens, in addition to background, of ionizing radiation as a total accumulated dose to the reproductive cells from conception to age 30.

(D) The previous recommendation should be reconsidered periodically with the view to keeping the reproductive cell dose at the lowest practicable level. If it is feasible to reduce medical exposures, industrial exposures, or both, then the total should be reduced accordingly.

(E) That individual persons not receive more than a total accumulated dose to the reproductive cells of 50 roentgens up to age 30 years (by which age, on the average, over half of the children will have been born), and not more than 50 roentgens additional up to age 40 (by which time about nine-tenths of their children will have been born).

(F) That every effort be made to assign to tasks involving higher radiation exposures individuals who, for age or other reasons, are unlikely thereafter to have additional offspring. Again it is recognized that such a procedure will introduce complications and difficulties, but this committee is convinced that society should begin to modify its procedures to meet inevitable new conditions.

(XV) CONCLUDING COMMENTS

The basic fact is—and no competent persons doubt this—that rediations produce mutations and that mutations are in general harmful. It is difficult, at the present state of knowledge of genetics, to estimate just how much of what kind of harm will appear in each future generation after mutant genes are induced by radiations. Different geneticists prefer differing ways of describing this situation: But they all come out with the unanimous conclusion that the potential danger is great.

This report recommends that the general public of the United States be protected, by whatever controls may prove necessary, from receiving a total reproductive lifetime dose (conception to age 30) of more than 10 roentgens of man-made radiation to the reproductive cells. Of this *reasonable* (not *harmless*, mind you, but *reasonable*) quota of 10 roentgens over and beyond the inevitable background of radiation from natural causes, we are now using on the average some 3 or 4 roentgens for medical X rays. This is roughly the same as the unavoidable dose received from background radiation. It is really very surprising and disturbing to realize that this figure is so large, and clearly it is prudent to examine this situation carefully. It is folly to incur any X ray exposure to the gonads which can be avoided without impairing medical service or progress.

The 10 roentgen recommendation applies in an average sense to the population as a whole. We also include a recommendation concerning the upper limit of exposure that any one individual should receive. These limits would of course apply to persons whose occupations involve radiation exposure, but they are intended as broad and uniform regulations which apply to any and every individual.

The fall-out from weapons testing has, so far, led to considerably less irradiation of the population than have the medical uses—and has therefore been less detrimental. So long as the present level is not increased this will continue to be true; but there remains a proper concern to see to it that the fall-out does not increase to more serious levels.

One important lesson which results from this study is the following: The present state of advance in atomic and nuclear physics on the one hand, and in genetics on the other hand, are seriously out of balance. We badly need to know much more about genetics—about all kinds and all levels of genetics, from the most fundamental research on various lowly forms of life to human radiation genetics. This requires serious contributions of time, of brains, and of money. Although brains and time are more important than money, the latter is also essential; and our society should take prompt steps to see to it that the support of research in genetics is substantially expanded and that it is stabilized.

We ought to keep all of our expenditures of radiation as low as possible. Of the upper limit of 10 roentgens suggested in Recommendation C, we are at present spending about one third for medical X rays. We are at present spending less—probably under one half a roentgen—for weapons testing. We may find it desirable or even almost obligatory that we spend a certain amount on atomic power plants. But we must watch and guard all our expenditures. From the point of view of genetics they are all bad.

WARREN WEAVER, *Chairman*
GEORGE W. BEADLE
JAMES F. CROW
M. DEMEREC
G. FAILLA
H. BENTLEY GLASS
ALEXANDER HOLLAENDER
BERWIND P. KAUFMANN
C. C. LITTLE
H. J. MULLER
JAMES V. NEEL
W. L. RUSSELL
T. M. SONNEBORN
A. H. STURTEVANT
SHIELDS WARREN
SEWALL WRIGHT

SUMMARY REPORT OF THE COMMITTEE ON PATHOLOGIC EFFECTS

Appreciation of the pathologic effects of radiation on man has required of this Committee and its subcommittees, consideration of voluminous experimental work on animals, as well as such direct data on human beings as are available. When the results of controlled experimental studies are considered in the light of the human data, it is found that the sequence of pathological changes is indeed quite similar in man and in animals, although man has certain definable peculiarities of response.

The human data include:

Results of excessive exposure to X-rays and radium in the early days;

Results of more moderate exposure to different forms of radiation, as experienced by cyclotron workers;

Results of introduction of naturally occurring radioelements into the body, notably radium preparations and thorotrast;

Effects of exposure at Hiroshima and Nagasaki;

Observations on populations irradiated by fallout;

Additional observations from clinical radiotherapy, use of artificial isotopes in therapy, a very limited number of accidents in atomic energy work, and certain statistical surveys of large groups.

Experimental work covers the whole field and includes studies of acute and chronic effects on many species of animals.

Certain human effects have to be assumed from consideration of experimental knowledge: for example, early effects of high doses to the central nervous system, and results of absorption of most of the artificially produced isotopes, and it is fair to say that the lethal dosage of penetrating radiation for man is less well known than for many other species.

Radiation has been added to the means of production of casualties in warfare. Not only can radiation cause death or immediate, or delayed injury by itself, but exposure to it intensifies the seriousness of burns or other injuries. The acute lethal dose for half of a given population is in the range of 400 to 600 r.

Despite the existing gaps in our knowledge, it is abundantly clear that radiation is by far the best understood environmental hazard. The increasing contamination of the atmosphere with potential carcinogens, the widespread use of many new and powerful drugs in medicine and chemical agents in industry, emphasize the need for vigilance over the entire environment. Only with regard to radiaion has there been determination to minimize the risk at any cost.

It appears, however, that a fairly clear general picture of human radiation effects can be presented. Members of this group and of its subpanels, while recommending various points of departure for greater consideration and further research, were in no case of the opinion that any sort of "crash program" would be desirable or profitable.

The various means whereby persons may be overexposed to radiation will have a great deal of influence on the over-all effects. For example, the exposures at Hiroshima and Nagasaki and a few exposures in accidents in atomic energy plants, involved radiation to the whole body in which the clinical effects reflected mainly injury to the blood-forming tissues and intestinal tract. These tissues are very sensitive to radiation but have a great power of recovery.

Where, on the other hand, exposure has been suffered at a relatively low level from time to time over a period of years, a variety of injurious effects may be encountered, such as leukemia and skin cancer. Among those who have adhered to present permissible dose levels, none of these effects have been detected.

Shortening of life span may result from exposure to radiation not only as a consequence of damage to a specific tissue, as seen in the development of skin cancer and leukemia, but also as a result of such general factors as lowered immunity, damage to connective tissue, or premature aging. Older members of the populations seem to be more sensitive to this nonspecific damage. The shortening of life correlates roughly with dose of radiation, but has not yet been demonstrated at low doses. The following table indicates life shortening in radiologists, who may well have received doses in the course of their occupation ranging from very slight to about 1000 r.

Average age at death

	Years
Physicians having no known contact with radiation	65. 7
Specialists having some exposure to radiation (dermatologists, urologists, etc.)	63. 3
Radiologists	60. 5
U. S. population over 25 years of age	65. 6

Shielding of even a portion of the body from radiation lessens the effect out of proportion to the relative amount of tissue protected. Therapeutic radiation to a single portion usually is much greater than the lethal level of total body radiation.

Radiation may have its prominent effects in particular parts of the body when it is applied locally, and this may take place in two ways. First, an external source may be so handled as to direct its radiaion to a particular part; in this way many of the early radiologists suffered acute or chronic injury to the hands, which has also occurred in more recent atomic energy accidents.

In the second instance, a radioactive substance may be taken into the body and deposited where it is a source of constant local irradiation until it is eliminated. Bone disease in radium workers and lung disease in miners of radioactive ores (both leading to cancer as a late development) are well-known examples of this mode of exposure. It is worth noting that the atomic energy industry, through diligence, has apparently avoided exposures leading to this type of injury.

It is thus characteristic of the radiations that their effects may manifest themselves not only immediately, but perhaps only after a long period of intermittent radiation, or may even be long delayed after a single exposure. One of the particular tasks of the panel has been to see all of these effects in a common perspective. They will be discussed here in terms of the effects of radiation on the important organs and tissues of the body, since it is a well known fact that some are more readily injured by radiation than others, and that injury to some has more serious consequences than to others.

Among the more serious effects of radiation are those on the blood, since the vital blood forming organs are particularly sensitive to radiation injury. The white blood cells are decreased in number soon after radiation, and in fatal cases they almost disappear before death. Other acute changes in the blood give rise to disorders in the clotting mechanism and a bleeding tendency, and the formation of antibodies against infections is impaired. These changes lead to acute illness in the second week (perhaps a little later in man), heralded by decrease in the white cells.

In the next few weeks anemias may occur due to deficiencies in red blood cell formation and survival. Those victims living through the first month usually recover, but in certain individuals, or where radiation is continued, there is a further serious breakdown of blood cell formation.

Some late effects of radiation appear as leukemias, which are found to arise a few years after radiation. This disease, relatively rare in man, may show manifold increase in persons subjected to a nearly fatal single dose (Hiroshima data) or in those whose professional work has exposed them to higher than acceptable permissible dose rates.

Effects on the intestinal tract are also critical in the early period. Vomiting and diarrhea occur within a few hours. This is a common complication of X-ray treatment to the abdomen, but is not fatal. It seems to be mediated through the vegetative nervous system and is probably not related to later damage.

Within a few days (usually four or five) after radiation, more serious effects occur. Failure of the cells lining the intestine to replace themselves results in denudation of the surface, with intractable loss of fluid and salts; complicated by ulcerations, spread of infection, and bleeding.

Late effects are seen after heavy radiation therapy, and resemble those seen in some other heavily irradiated tissues: overgrowth of connective tissue (fibrosis) and decrease in the number of functioning epithelial cells. Cancer has occurred in animals given overwhelmingly large doses of isotopes in insoluble form by mouth.

Effects of radiation on skin have been widely observed. On the first day an erythema, resembling that of sunburn, appears but is transitory. A few days later a somewhat more persistent erythema occurs which may be associated with pigmentation. Ulceration may occur in this period after high doses. Much later, atrophic changes are seen, with marked deficiency of the blood supply and intractable ulceration; such a chronically damaged skin is a fertile bed for cancer development. The Marshall Island group, while receiving total body radiation insufficient to produce serious changes, had rather marked secondary skin lesions from direct contact with fallout material. Slight local vascular changes have been observed after two years, but serious after effects are not anticipated. Falling of hair was temporary in these persons; heavy dosages are required to make it permanent. In animals, destruction of the pigment cells causes regrown hair to be white, but such loss of pigment seems not to take place in men under comparable conditions.

Bone: Early radiation effects are not of note, except that retardation of growth of epiphyses of immature bones occurs and may produce serious results in children given local radiation therapy. Late effects are seen in radium poisoning, where we see repeated destruction and repair, culminating in widespread destructive changes in which bone sarcoma is likely to appear.

Lung: Early after large doses we see congestion and increased secretion. Here, again, the late-appearing changes are of greatest importance: fibrosis, and development of cancer, which has been very common in mining areas where large concentrations of radon gas were inhaled.

Thyroid: An early and persistent effect is depression in secretory activity, which is used as the basis of the radioiodine therapy of hyperthyroidism. No serious late local effects of thyroid radiation in adults have been recorded, although some leukemias have followed heavy radioiodine treatment. A small proportion of children treated with X-ray to the upper part of the body, however, develop thyroid cancer later on, suggesting a specially high sensitivity of the child's thyroid.

Eye: The only noteworthy lesion is cataract of the lens, which is a late response. It is much more readily produced by neutrons than by X-rays, therefore, has been most prominently observed in cyclotron workers.

Gonads: A single sublethal radiation dose to a male may result in sterility after two to three weeks, followed by a slow recovery. Chronic treatment results in a gradual reduction in number, motility and viability of sperm. This is the most sensitive indicator of chronic damage so far observed, being measurable in dogs at ten times the permissible dose rate. Larger doses (about equal to the total-body lethal dose) permanently sterilize males and females. Experience with the Marshall Islanders, the exposed Japanese, and certain accident cases indicate that total body doses up to about 40–50% of the lethal have no permanent effect on human fertility.

Central Nervous System: Observations in man are quite limited. Very high doses given to animals result in loss of coordination and excitement soon after

irradiation. At later stages, various effects are seen which indicate sensitivity of particular cells and areas.

Effects on Embryos: Treatment of embryos at various stages of development may lead to highly specific malformations depending on the exact developmental stage at the time of irradiation. At critical stages, relatively low dosages (those permitting survival of the mother) may cause serious malformations. These changes must be distinguished from genetic mutations, as one is often tempted to call abnormal offspring mutations. The type of malformation discussed here would not perpetuate itself genetically, and would result from radiation during gestation.

It must also be remembered that there are various other agents causing malformations during development, of which German measles is a well-known example.

A few factors influencing sensitivity might be mentioned. Very young or very old animals have increased sensitivity to lethal effects. Growing tissues are generally more readily damaged. States like hibernation delay the appearance of radiation damage but do not prevent it. Moderate stresses seem not to effect sensitivity but severe ones such as burns or exhausintg exercise, have a deleterious influence, augmenting sensitivity.

Local radiation in sufficient amount to almost any part of the body may produce cancer, the chance of tumor development being somewhat related to dose. Since the cancer cell is an altered type of a normal tissue cell, it has often been suggested that cancer is a somatic mutation, like a genetic mutation but arising in a tissue cell which perpetuates the character by its growth.

All types of induced and spontaneous tumors appear not to arise at once, but to pass through a serious of preliminary stages; and radiation induced tumors take a particularly long time to develop. Radiation induced cancer occurs in the absence of a generally abnormal state of the tissue of origin. Mouse experiments show that shielding of a part of the body will prevent radiation leukemia and that shielding of one ovary will prevent tumor from developing in the other; and several of the tumors appearing late after irradiation seem to be produced in response to indirect mechanisms. If somatic mutation is a necessary part of the induction of cancer, it would seem to play a minor role.

We have so far considered effects of overdosage of radiation in various forms. The question must necessarily be considered, as to whether much smaller amounts of radiation harmless to individuals, might be deleterious to large populations. Because of the striking difference of germinal and somatic cells the former carrying on from generation to generation injuries received, the Genetics Committee has recommended for large populations permissible dose levels of radiation lower than those which are safe for any one generation. As the permissible dose level which they have hypothesized as desirable for large populations were to be applied there would be no demonstrable somatic effect, although a theoretical minor shortening of life span could not be ruled out.

As regards internal contamination, independent data on Rongelap inhabitants and Japanese fishermen indicate that a considerable proportion of the lethal dose of external radiation was received by individuals who barely exceeded, and only for a short period, the permissible internal burden.

The only situation worth considering in relation to large-scale pathologic effects would then be widespread contamination with Strontium–90, which is a long-lived (half life 10,000 days) readily absorbed, bone-seeking isotope which tends to fall out generally over the earth rather than in accordance with the usual close or intermediate fallout pattern. It has already been found that some young individuals have retained 0.001 microcuries or one-thousandth of the permissible dose. This amount if maintained through life would yield 0.2 rep (equivalent r) to the skelton.

In developing an unequivocally safe amount, we can recall that a certain degree of radiation exposure has always been with us, even excluding X-rays, in the form of gamma radiation from minerals, cosmic rays, and radioelements normally in the body. These levels vary greatly from one location or altitude to another and are not considered to produce harmful effects.

There seems no reason to hesitate to allow a universal human strontium (very similar chemically to calcium) burden of 1/10 of the permissible, yielding 20 rep in a lifetime, since this dose falls close to the range of values for natural radiation background. Visible changes in the skelton have been reported only after hundreds of rep were accumulated and tumors only after 1500 or more.

In relation to world-wide contamination, food chains are important. Fallout contaminates plants through ground and leaf deposition; animals eat these plants. Because in fact milk and cheese are human sources of radiostrontium, being high in calcium. Throughout this chain, strontium is discriminated against relative to calcium, which reduces the hazard somewhat. It must be remembered that in regions where soil and water are low in calcium, calcium and strontium will be more readily taken up.

As to therapy of radiation injury: while treatment is difficult, some success has been achieved with antibiotics and properly timed blood transfusions. Shielding of a portion of the body appears to give a degree of protection disproportionately large for the mass shielded. Experiments set up to explain this fact may help in developing a rational treatment. Also, various forms of treatment given immediately before radiation have been devised, but do not appear in any sense practical. Studies of this sort may, however, provide a basis for future discoveries.

Because of the nature of this report, specific recommendations regarding needed research are omitted here, but will be published later when the subcommittee reports and other appendices are published in full.

SHIELDS WARREN, *Chairman*
AUSTIN M. BRUES, *Rapporteur*
HOWARD ANDREWS
HARRY BLAIR
JOHN C. BUGHER
EUGENE P. CRONKITE
CHARLES E. DUNLAP
JACOB FURTH
WEBB HAYMAKER
LOUIS H. HEMPELMANN
SAMUEL P. HICKS
HENRY S. KAPLAN
SIDNEY MADDEN
R. W. WAGER

OUTLINE OF APPENDICES

(To be published later)

I. Subcommittee on Acute and Long Term Hematological Effects of Atomic Radiation—E. P. Cronkite, Chairman:
  - Introductory Comments by Chairman.
  - Acute Hematological Response to Single Doses of Penetrating Radiation.
  - Long Term Effects on the Blood of a Single Exposure.
  - Effects of Repeated Low Level Exposure.
  - Usefulness of Hematologic Studies in Control of Radiation Injury.
  - Conclusions.
  - Bibliography, 1940–55.

II. Report of Subcommittee on Internal Emitters—A. M. Brues, Chairman:
  - Statement of the Problem.
  - Fallout Conditions.
  - Acute Toxicity.
  - Chronic Toxicity: Site of Injury.
  - Effects on the Lung.
  - Ruthenium; Cesium; Activation Products.
  - The Alkaline Earths:
    - Metabolism.
    - Toxicity.
    - Sr 90—Radium Comparison.
    - RBE, Alpha and Beta Rays.
    - Absorption of Strontium.
  - Radioiodine.
  - Radiation from Particles and Hot-Spots.
  - Permissible Dosage to Large Populations.
  - Therapy by Removal of Radioelements.
  - Bibliography.

III. Report of the Subcommittee on Acute and Chronic Effects of Radioactive Particles on the Respiratory Tract—Ralph W. Wager, Chairman:
  - Introduction.
  - Sources and Nature of Airborne Radioactive Particles.
  - Nuclear Detonations.
  - Description of the Respiratory Tract, Anatomy and Physiology.
  - Fate of Inhaled Particles.
  - Radiation Effects on the Respiratory Tract.
  - Conclusions.
  - Recommendations.
  - Bibliography.

Summary Report of the Committee on Meteorological Aspects of the Effects of Atomic Radiation

Chapter I. Debris from Nuclear Tests

A. Introduction

Nuclear weapons produce atomic clouds which rise to heights dependent principally upon the energy released and also on the type of burst (air, surface, underground, etc.). Weapons in the kiloton range leave most of their radioactive debris in the troposphere, while megaton weapons are powerful enough to inject significant quantities of radio-active material into the stratosphere. Once the debris is injected into the atmosphere, it is rapidly spread over the earth by atmospheric processes, and eventually deposited on the surface of the earth, in a complex manner. Among the many problems are included: the way in which debris is mixed and transported by the atmosphere, both vertically and horizontally, the mechanism of removal from the troposphere and deposition on the ground, and the rate of penetration from the stratosphere through the tropopause and into the troposphere for eventual removal.

1. *Categories of fallout.*—The problem of the removal of radioactive debris from the atmosphere and its deposition in the biosphere may be divided into three phases: (1) Early or "close-in" fallout, that which occurs within the first ten to twenty hours following a nuclear explosion; (2) Intermediate fallout, that which occurs during the first weeks following the burst; and, (3) Delayed fallout, the slow removal of small particles which may continue for months and even years, particularly after a high-yield thermonculear explosion.

The principal mechanisms by which the removal occurs are gravitational settling, scavenging of radioactive particles by falling precipitation, and deposition by diffusion resulting from the everpresent turbulent eddies of the atmosphere. Although all principal mechanisms of removal play a role in each phase of the fallout, the primary emphasis shifts from gravitational influences in the early fallout to precipitation scavenging in the intermediate phase to an as yet poorly understood combination of diffusion and scavenging in the delayed fallout.

2. *Measurements.*—The most direct measurement of radioactive deposition is that made from the soil since it represents the main natural surface onto which the particles fall. Difficulties arise from the fact that rain may remove some of the activity by runoff or soaking deeper into the ground. As a measure of the true radioactivity on the ground in determining plant or animal intake of strontium 90, for example, soil sampling is obviously the most acceptable solution. But, for an accounting of the amount which has been deposited, the soil analysis may be unsatisfactory if the sampling is performed, at say, yearly or multi-yearly frequency. Soil sampling on a frequent basis may be impractical.

Measurement of radioactivity by use of hand monitoring equipment is standard practice in areas where the radioactive deposition is significantly above normal background. This kind of observation is almost entirely useless outside of the areas of close-in fallout.

For daily, weekly or monthly fallout collections, the New York Operations Office of the Atomic Energy Commission recommends the use of a one-square-foot sheet of gummed film mounted horizontally on a stand three feet above the ground. An extensive, world-wide network of daily gummed film collection at about 250 locations has been operated by the Atomic Energy Commission for several years.

Finally, since there is evidence that much of the radioactivity deposited outside of the close-in area is brought down in precipitation, the collection of whole water samples is a method of obtaining the radioactivity of particles.

Air concentration.—Measurement of air concentration near the earth's surface has been achieved by a variety of sampling procedures. Filtration equipment of many types has been successfully employed, but the efficiency of the filter material for various particle sizes, particularly in the sub-micron range, must be determined before quantitative interpretation of the data can be made.

The fact that the upper atmosphere contains significant atomic debris has been known for several years. Sampling of the upper air by aircraft has been achieved by using the motion of the aircraft to pass air through a filter paper. The British report the presence of fission products at the peak altitude of their aircraft, 48,000 feet. The Japanese have measured the radioactivity by carrying aloft Geiger counters on balloons. By subtracting the cosmic ray counts from the total, the remainder is ascribed to fission products. American scientists do not view this procedure with favor for the low levels of radioactivity found over most of the world.

Instrumentation for the measurement of radioactivity by its effects on the electrical properties of the atmosphere also are of use only in those regions where the fission product concentrations are comparatively high.

B. Close-in fallout

1. *Description.*—Close-in fallout is the radioactive material from an atomic explosion which is deposited on the ground within a few hundred miles of ground zero, and which is down in some ten to twenty hours.

There is a fundamental difference between the fallout from an atomic device detonated at the ground and the fallout from one detonated so high that the fireball does not touch the ground. In the case of the surface burst, large quantities of surface material are broken up, melted, and even vaporized, and some of this material comes in intimate contact with the radioactive fission products. Then, after the atomic cloud has stopped rising and the violent updrafts associated with the explosion have subsided, the larger and heavier particles start falling back to the ground. The result is an area around ground zero and extending downwind which is covered in a more or less systematic way with radioactive particles.

In the case of an air burst in which the white-hot fireball never reaches the surface, the radioactive fission products never come into close contact with the surface material; they remain as an exceedingly fine aerosol. At first sight this might be thought to be an oversimplification, since there have been many cases in which the fireball never touched the ground, but the surface material was observed to have been sucked up into the rising atomic cloud. Actually, however, in such cases a survey of the area has shown that there has been a negligible amount of radioactive fallout on the ground. Though tons of sand and dust may have been raised by the explosion, they apparently did not become contaminated by fission products.

Experience has shown that an atomic device exploded on the surface distributes about 70–80 percent of its fission products on the ground within a few hundred miles of the burst point. A somewhat larger percentage will take part in the close-in fallout from an underground burst, and a smaller percentage will be scavenged from a near-surface burst or tower shot.

In order to make a quantitative study of the manner in which close-in fallout occurs, one must have a knowledge of the following parameters: wind structure, yield and height of burst, and kind of surface.

As each particle falls, it is carried horizontally by the wind at each level. The time during which it is falling through a given layer is inversely proportional to its rate of fall. Thus its horizontal travel during its entire fall from an initial height can be expressed as a summation of its horizontal travel in each layer. The rates of fall of atomic particles vary with particle size, shape, density, as well as the altitude.

Although no experimental information is available on the effects of precipitation during this initial stage of the atomic cloud, it is evident that significant deposition can occur from this cause. However, the effect would be most marked

from smaller yield bombs, since the bulk of the debris from larger bombs rises well beyond the rain-bearing strata.

2. *Height and size of the atomic cloud at the time of stabilization.*—It is evident that the physical size of the atomic cloud will have an effect on the distribution of the close-in fallout. The height to which the debris is carried will determine how far downwind a given particle size will drift, and the horizontal extent will serve to spread the fallout over a larger area.

In the first few seconds following an atomic detonation, the fireball grows rapidly, until the pressure inside the fireball is roughly that of the ambient air. At this point its temperature is still many thousands of degrees higher than that of the atmosphere around it, so it is much less dense, and the buoyancy of the atmosphere forces it to rise. However, it does not necessarily rise like a hot "bubble" or balloon, but in most cases, it develops a strong toroidal internal circulation and rises in the form of a smoke ring.

As the smoke ring rises, its internal circulation draws air in at the bottom and incorporates this new air into the cloud. The result is a very large growth in the size of the cloud as it rises, due mostly to the entrainment of the air from each level through which it passes.

It is clear that the cloud will gradually cool during its rise, due to radiation, the entrainment of the outside air and adiabatic expansion. When the mean temperature inside the cloud is the same as that of the ambient air at the same level, there will be no further buoyancy and the cloud as a whole will cease rising. However, at this point the kinetic energy of the toroidal circulation may still be considerable. For devices with yields of a few kilotons, the smoke ring circulation breaks up at about the same time that it reaches its point of stabilization, but for devices in the megaton range this toroidal circulation continues to pump air in at the bottom for ten to twenty minutes.

The net result of this pumping action after stabilization is a significant increase in the horizontal size of the atomic cloud, since the air which is drawn in at the bottom is forced out radially. Observations of this effect in the case of megaton devices are hindered by the fact that the structure of the cloud becomes confused.

The atmospheric stability will vary with season and latitude, and this accounts, in part, for the difference between the altitude of a cloud detonated in a tropical atmosphere and one of the same yield in a middle-latitude winter atmosphere. The most noticeable difference between these two regimes is the height of the tropopause.

3. *Distribution of radioactivity within the cloud.*—Since it is difficult to obtain enough samples of the radioactive debris while it is still within the cloud to determine its initial distribution, the most reliable estimates of this distribution have been based on the observed fallout and a reconstruction of what this initial distribution must have been.

It is clear from the observations of the rising cloud that almost all of the lighter debris is carried aloft in the smoke ring cloud. Apparently a certain fraction of particles are large enough to be thrown out of this ring, and these are left behind in the stem. However, in the stem there are violent updrafts for the first few minutes, so all but the very large particles will continue to be carried aloft.

For a surface or near-surface burst, the type of terrain must have a significant influence on the particle size and activity distribution within the cloud.

4. *Prediction of close-in fallout.*—At the outset it would be well to state what use can be made of a prediction of the fallout area from an atomic burst. At the risk of oversimplifying the case, here are some of the pertinent factors:

> Wind observations, now almost invariably made with sounding balloons, give winds which are not entirely representative of the winds which will affect the falling atomic debris. This is because winds change with time and place and because wind observations, as all meterologists recognize, are subject to a certain amount of error. Forecast winds, by the same token, are usually even further in error. A number of studies have been made of this subject. For example, a recent study by the Air Weather Service indicates that mean vector errors in 24-hr forecasts range from about 60 percent of the observed wind at middle altitudes to over 70 percent of the observed wind at 100 mb (about 53,000 ft.). These mean vector errors correspond to wind errors of 18 to 29 knots. It is perhaps significant that these forecast errors at the higher levels (40,000–55,000 ft.) are about the same as the root-mean-square deviation of the wind from the mean wind, and at lower levels (about 20,000 ft.) the 24-hr forecast error is about

half that of the normal climatological deviation. If one had to rely on forecasts 24-hrs old, he would be just about as well off if he used climatological data or persistence in computing the fallout.

The mushroom cloud from a multi-megaton device may rise entirely above the normal coverage of our radiosonde and RAWIN network, since it is generally considered impractical to plot and analyze current weather data at levels above 100 mb., or about 53,000 ft. Thus, unless special efforts are made there will simply be no wind data at all for the winds which will affect the debris during the first part of their fall. The effects of vertical motions in the atmosphere, possibly including currents arising from bomb-produced fires, may also be enough to alter the fallout pattern.

It should be fairly evident from the discussion in the preceding section that there are still a number of questions concerning close-in fallout about which we are still somewhat uncertain. Any fallout computation, even given perfect information on the wind field, will have a degree of uncertainty as a result of the assumptions on which it is based.

With these factors in mind, it appears unlikely that a weather forecaster, even given the computing aids which he would need to compute a fallout pattern, could on short notice and in a time of emergency give a detailed and reliable forecast of the close-in fallout. He could with a fair degree of assurance delineate the general sectors in which the fallout would be mostly likely to occur, but he could not tell where a given dose rate contour would lie. If one is dealing with a military situation in which an enemy is dropping atomic bombs, then the forecaster's problem is further complicated by the fact that he would presumably not have accurate knowledge of the height of burst and fission yield of the weapon.

It must be emphasized, however, that the above statements do not necessarily apply to the prediction of the fallout from a test device, where many of the uncertainties mentioned can be removed. It is possible, by the use of a special upper air-sounding network, to obtain wind information over a limited area which is considerably more reliable and current than that obtained from the routine upper air net, and which extend to a greater altitude. Moreover, there is usually no doubt about the yield and burst height of the device during a test. Thus, it is much more likely that an accurate forecast of the fallout pattern can be made under the favorable conditions which exist during a test. Even here, there remains a degree of uncertainty, as witnessed by the fallout which occurred on some inhabited atolls during the 1954 tests in the Pacific—though this might have been forecast if there had been the refined fallout computing aids which exist today.

Finally, if one does not have to make use of forecast winds at all, but can introduce all the detail of a careful synoptic analysis "after-the-fact", including the time variation of the wind at each level, and compute the fallout on a high-speed computer, it is possible to reproduce the fallout patterns which have occurred from the U. S. surface bursts with considerable accuracy. The radiological monitoring data show a certain amount of spread in the observations because of the detailed effects of terrain and atmospheric turbulence. When the reconstructed pattern or computed fallout patterns are compared with observed values, the minor differences are usually accounted for by small-scale features in the wind structure. Where the winds apparently behave as expected, predictions verify within a factor of two over most of the area. Where they do not, the peak dose rate is often correctly predicted at various distances from ground zero although displaced relative to the observed peaks.

C. Intermediate fallout

Although gravitational settling continues to play an important role for many days, and the downward diffusion of debris from the atomic cloud as it is moved about by the upper winds also becomes important, the primary removal of debris after the first day or two following a burst occurs in areas of precipitation. As the cloud of debris continues to be diluted by the atmosphere, concentrations decrease and it becomes necessary to collect the fallout and wait until the natural radioactivity has decayed before measurements can be made.

From Nevada test series, it has been found that less than 5% of the total beta radioactivity produced is collected by the gummed film network in the United States. Stewart, Crooks, and Fisher have estimated from observations in the British Isles that about half the radioactive dust in the troposphere from Nevada tests is deposited in approximately 22 days and that 80% of the deposition by rain occurs during the first transit of the cloud over England.

The importance of precipitation in bringing debris to the ground after the first day or so following an atomic explosion is strikingly shown in the average daily activity found on gummed films exposed in the United States during the Teapot Nevada test series in the Spring of 1955. In light rain, on the average, over twice as much activity is collected by the gummed film as compared to dry days and this increase becomes more apparent as the rain gets heavier. Various studies have shown that anywhere from four to more than ten times as much debris is deposited during periods of rain as compared with dry days.

On a few occasions, rain has coincided with the passage of a fresh cloud of debris from a Nevada test, resulting in local increases of background radiation to about 1 mr./hr. beyond a few hundred miles from the test site.

In the absence of precipitation, the effects of turbulence as well as gravitational settling are important.

Removal of debris by impaction on natural surfaces, buildings, etc., resulting from the movement of air around these surfaces must be appreciable. Various studies have shown radioactive particles are found on leaves, branches, etc. An experiment conducted at the Naval Research Laboratory with an 80-mesh stainless steel wire screen and with ordinary cheesecloth faced into the wind showed that in the absence of rain as much as 10 to 100 times the activity collected on the horizontal gummed film can be collected on the screen or cloth. In a two-month period during the Teapot series, a total of 50% more activity was collected on the cheesecloth than on a horizontal gummed film of similar size. Studies of the vertical distribution of chloride particles also indicate a depletion near the ground over land areas, presumably a result of impaction on natural surfaces.

D. Delayed fallout

In contrast to the results from the Nevada tests, measurements of radioactive debris concentrations in the troposphere showed a continued increase over England during the 10-month period following the thermonuclear tests in the Pacific in 1954. Similar increases in ground-level concentrations have also been observed by the Naval Research Laboratory in the United States and elsewhere.

This delayed fallout is a consequence of the extreme heights reached by debris from thermonuclear explosions, more than 80,000 feet, which results in the storage of large amounts of small particle-size debris in the stratosphere. The existence of such a distribution has been confirmed by aircraft measurements over the British Isles in August and September 1954 and again in early 1955 which show a very large increase in air concentration above about 35,000 feet. This debris eventually moves through the tropopause into the troposphere, from where it is removed by precipitation scavenging and by deposition.

1. *Transport in the stratosphere.*—The stratospheric levels in question are mainly in a region where relatively sparse synoptic data on the structure or air currents are available. However, they are mainly in a region of hydrostatically stable air and soundings indicate, in general a relative high degree of steadiness of stratospheric currents.

The winds in the stratosphere seem to have a predominant zonal component. The material injected at a certain locality will spread to other longitudes faster than to other latitudes. Material injected at a certain time in a vertical column may move more rapidly, or even in a different direction, at one level with respect to another. This shearing motion of the large-scale air currents represents a powerful factor for the spreading of an originally localized cloud to all longitudes within a few weeks.

All stratospheric circulation cells undergo more or less marked changes during the course of the seasons. Superimposed on the seasonal trend are day-to-day wind fluctuations caused by migrating or oscillating pressure systems. The present-day knowledge of independent stratospheric pressure systems is very limited. But it can be assumed that the stratosphere reacts, at least partly, to the migrating cyclones and anticyclones of the troposphere. Over periods of several weeks the net effect of the stratospheric wind variability will be similar to a process of large-scale eddy diffusion acting mainly in the horizontal directions.

2. *Diffusion in the stratosphere.*—One may approach the question of vertical diffusion in the stratosphere in three ways: first, using first principles; second, using natural gaseous tracers and third using man-made probes.

(*a*) *First principles.*—If asked for criteria to predict vertical mixing at the ground from meteorologically-observed parameters, one would point, in all likelihood, to three items: vertical temperature gradients, wind speed and wind shear. The greater the temperature stability the less the vertical mixing. It is primarily

on this ground that the stratosphere has been viewed as a region of quiescence in comparison with a turbulent troposphere below it.

With regard to wind speed, it seems fairly clear that an absence of horizontal kinetic energy will be associated with little or no vertical motions but, it is not evident that high wind speed necessarily will produce vertical turbulence. In any event, the lower stratosphere has a variety of speeds.

In the Richardson number, which under special conditions predicts the onset of turbulence, it is the shear rather than the wind speed which is significant. There is as large an assortment of wind shears in the stratosphere as in the troposphere, barring the layer adjacent to the jet streams in the troposphere.

One must conclude that on one count—probably the most important—stratospheric vertical mixing should be much smaller than tropospheric and that on the other two scores, it need not be.

(*b*) *Gaseous tracers.*—Ozone is the first such atmosphere property which comes to mind. It has been established that the ozone concentrations below the ozone maximum (about 25 km) are often in excess of the photochemical equilibrium amounts. It appears that the day-to-day variations and much of the seasonal variation of total ozone reflects changes in the non-equilibrium ozone in the "protected" region below the maximum. It is generally accepted that exchange processes transport ozone downwards from the region of ozone maximum. Three types of exchange process have been considered. The first involves large-scale meridional circulations in the stratosphere. There are some reasons for accepting such a meridional circulation involving both hemispheres but the evidence is not very impressive. A second exchange process is turbulent mixing. This is difficult to evaluate because of the lack of information on the magnitude of the mixing coefficient. It does seem, however, that the mixing coefficient required to provide the needed flux of ozone is not unreasonable. The third exchange process may be called "Gross austausch" since it involves the vertical motions associated with travelling cyclones and anticyclones. There is good evidence for this effect in the correlations between total ozone and the pressure field. It also provides a qualitative explanation for the annual variation of total ozone.

With the possible exception of the large-scale meridional circulation, the exchange processes described above will operate to bring ozone into the troposphere where it is destroyed at lower levels by particulate matter. The study and measurement of the ozone exchange should be applicable to the exchange of nuclear weapon debris.

Water vapor probably has no marked sink (due to cloud formations or precipitation) near the tropopause. Thus, changes in the gradient of water vapor mixing ratio should be a clue to the comparative upper tropospheric-lower stratospheric mixing intensities. The use of moisture as a tracer suggests but does not clearly indicate little vertical mixing in the lower stratosphere.

(*c*) *Man-made probes.*—Both parachutes and balloons have been used regularly to measure small-scale vertical motions in the stratosphere and the results generally reveal the stratosphere to have greater vertical motions than the troposphere. Also, aircraft report turbulence in the stratosphere. This evidence for comparatively short period vertical motions is clouded by the question of the role of the platform. The growth of the rising balloon, for example, alters the flow around it which may be the cause for the apparent vertical motions deduced from its ascent rate. Further, as with any measure of vertical motions, the probe does not distinguish between non-dispersive vertical motions like gravity waves, and true diffusing elements.

3. *Mixing through the tropopause.*—In a practical definition the tropopause is the level of minimum temperature of a high-altitude sounding, or the layer of maximum change of vertical lapse rate of temperature when no minimum temperature is encountered. Mean height-latitude cross sections of the atmosphere show that the tropopause is quasi-horizontal only in equatorial and polar regions, at approximately 18 and 9 km, respectively. The break occurs normally between 30 and 60 deg. latitude where the mean tropopause has either a significant slope or lacks uniqueness of definition so that multiple tropopauses are assumed by some authors even for mean conditions. Individual soundings may show considerably day-to-day fluctuations of the tropopause level, in connection with the passage of cyclones and anticyclones. Therefore, the tropopause is far from being a well defined geometrical surface and can hardly be considered an internal boundary which separates two distinct kinds of air masses. Air may move vertically through the mean tropopause level, or horizontally through the tropopause breaks. However, net radiation and convection processes are assumed to exist which result in a marked tendency towards re-establishment

of the tropopause at preferred levels just above the atmospheric layer in which the content of liquid and vaporous water is significant and condensation-precipitation cycles are dominant.

Four main types of exchange of air, or air properties through the tropopause may be distinguished: (i) small-scale vertical exchange, or vertical eddy diffusion—(ii) medium-scale penetration of tropospheric air into the stratosphere above extremely intense convective cells (heavy squall lines, frequently connected with tornadoes)—(iii) large-scale entrainment of stratospheric air into tropospheric systems such as cyclones, jet streams, hurricanes—and (iv) mean transport by vertical branches of large-scale to world-wide circulation cells.

4. *Tropospheric removal.*—The very small particles which are originally in the stratosphere and reach the troposphere weeks, months and even years after the detonation of a thermonuclear weapon, must eventually be deposited in the biosphere. However, the mechanisms by which these small particles are finally removed from the troposphere are not clear and the data concerning this problem is inconclusive.

Investigations of the rate of removal of natural radioactivity from the lower troposphere, both in the United States and in Germany, indicate that about half the activity is removed in a period of about one or two weeks. However, the particles involved are extremely small (probably less than 0.01) and are concentrated near the ground, so that the results may not be applicable to the fallout problem. On the other hand, Langmuir has shown that the collection efficiency of precipitation for very small droplets (less than 1) is small, but again the results may not be applicable to the fallout problem, where electrostatic and surface tension phenomena are different. Agglomeration between natural cloud elements and radioactive particles is operative for small particles.

Conflicting evidence on the rapidity of tropospheric removal is also found in studies of the actual fallout. Stewart, Crooks and Fisher, in Britain, estimate from indirect reasoning that deposition in rain exceeds dry deposition by a factor of twenty for thermonuclear explosions, a study of gummed film results in the United States does not bear this out—average monthly deposition at 40 monitoring stations during September and October, 1954, shows no correlation with either total rainfall during the month or the number of days with rain at the station. Again, using the British data, it is seen that the specific activity of the lower atmosphere showed a more than fourfold increase during the interval from 10 weeks after the Pacific tests to 50 weeks after if the data is corrected for decay. Similar increases were found by the Naval Research Laboratory. It is hard to reconcile this increase in tropospheric concentration with the rapid cleansing of the troposphere.

E. Analysis of stratospheric storage from radiostrontium fallout data

1. *Statement of the problem.*—The fission product of greatest interest in terms of long-term hazard from nuclear detonations appears to be Sr^{90}, and estimates of the rate of deposition of this isotope in the biosphere are needed. Unfortunately, our knowledge of the physical mechanisms involved is too meagre to deal with this problem on a theoretical basis. Although it has been established that a considerable amount of debris is injected into the stratosphere and that this debris slowly mixes downward into the troposphere and is eventually deposited on the ground, the average storage times in the stratosphere, and even in the troposphere, are uncertain. Among the many unknowns in attempting a theoretical analysis are the initial distribution in the stratosphere and the physical mechanism of stratospheric removal. Even if the latter were known, we are at present unable to make quantitative estimates of the rates or intensities of these physical processes. However, due to the biological uncertainties in estimating the hazard from Sr^{90}, a precise answer is not needed, and even a gross estimate would be useful.

2. *Analysis by W. F. Libby.*—Dr. W. F. Libby of the Atomic Energy Commission has published an estimate of the stratospheric storage time based on the estimated stratospheric content and on the observed deposition, with little or no reference necessary to the physical mechanisms involved. Essentially, the annual deposition is divided by the amount in the stratosphere, yielding the fractional removal during the year. If the fractional removal rate is assumed constant (i. e., the stratospheric content is assumed to decrease exponentially) the mean residence time of the debris is given by the ratio of the stratospheric content to the deposition.

The basic data used by Dr. Libby are the stratospheric content immediately after the completion of the Castle (Spring 1954) tests in the Pacific and the deposition of Sr^{90} during the following year or so as measured in three ways, a

world-wide gummed film fallout network, the Sr^{90} content of Chicago rainfall and air filter measurements at Washington, D. C. From these results, Libby concludes that the mean storage time for debris in the stratosphere is approximately 10 ± 5 years.

3. *Conclusion.*—Stratospheric storage not only serves to delay the fallout of debris, but also to disperse it over the globe, minimizing the chance of locally high concentrations of debris. At present, the amount of Sr^{90} in the stratosphere from nuclear weapon tests is far too small to approach maximum permissible concentration even if it were to be all deposited now. However, if the testing programs of the several countries producing thermonuclear weapone were to intensify, stratospheric storage time may become a critical item in terms of hazard to mankind. For this reason, a continuing program to investigate this phenomenon is needed, including actual measurements of the radioactivity in the stratosphere and improved and more representative methods of observing fallout.

CHAPTER II. ATMOSPHERIC RADIOACTIVITY FROM CIVILIAN APPLICATION OF NUCLEAR ENERGY

A. Sources of contamination

The hazards of atmospheric contamination from the military uses of atomic energy have tended to overshadow other possible sources of contamination, principally because, to date, relatively insignificant contamination has occurred from non-military sources. Certainly, the near future will see a tremendous increase in the utilization of nuclear energy for peaceful purposes, including the production of electric power, medical, industrial and agricultural applications, and nuclear propulsion of air, sea and land vehicles.

As far as can be seen today, the largest potential use of nuclear energy will be in the production of electric power and the discussion is based on this aspect of the problem, however, other applications could conceivably double the values used in the estimates given here. A consensus of estimates of global power requirements and of the proportion of this energy which will be supplied by nuclear sources indicates that by 1975 there will be a nuclear heat energy production of 10^8 to 10^9 kilowatts and by the year 2000 this will increase to 10^9 to 10^{10} kilowatts.

These rates of production will produce enormous amounts of fission products. However, most of these will be in solid or liquid form at present day processing temperatures and it can be expected that such material will not be intentionally released into the atmosphere. Of the remaining volatile fission products, storage and "cooling" of the fuel before processing can reduce the activity materially. The two volatile isotopes of most interest are 10-year krypton 85 and 8-day iodine 131. Only the 10-year krypton is sufficiently long lived to be relatively insensitive to the cooling time of the fuel before processing. There are two aspects to the problem of radioactive hazard from these sources, large-scale contamination on a global or hemispheric basis and local or regional contamination in the areas of processing plants.

B. Large-scale contamination

1. *Krypton 85.*—The long half-life of Kr^{85} results in the accumulation of this isotope in the atmosphere. If by the year 2000 nuclear thermal power has risen to 10^{10} kilowatts, the world inventory of radiokrypton would be of the order of 10^{10} curies. Mixed uniformly through the mass of the troposphere (4×10^{21} grams of air), the resulting sea-level concentrations would be less than 10^{-8} curies/meter^3. Since most of the activity is likely to be released in the middle latitudes of the northern hemisphere, large scale concentrations of 3 to 5 times the global average could be experienced in these latitudes.

No value for the maximum permissible concentration of Kr^{85} is presently available. If, from the chemical and radiological similarity, we assume that it is analogous to radioxenon, then the estimated worldwide concentration in the year 2000 is about two orders of magnitude less than the maximum permissible concentration. However, such comparisons are extremely questionable and it is important that maximum permissible concentration levels be established for Kr^{85}.

2. *Iodine 131.*—The problem of I^{131} in the atmosphere is largely dependent on the fuel recharging interval and the cooling time. For each combination of fuel cycle and cooling time it is possible to calculate the total amount of I^{131} in the atmosphere. This is an equilibrium value assuming no removal at the source or after release. Total amounts of I^{131} in the atmosphere based on the estimated nuclear energy production in the year 2000 are given in the following table.

Total I^{131} (curies) in the atmosphere per 10^{10} kilowatts of nuclear energy

	Decay time before release		
	none	10 days	100 days
Fuel recharging frequency:			
Once a year	6 x 10^9	3 x 10^9	10^6
10 times a year	6 x 10^{10}	3 x 10^{10}	10^7
Continuous	2 x 10^{11}	10^{11}	4 x 10^7

The present maximum permissible concentration of I^{131} is 3×10^{-9} curies/meter^3. If the I^{131} is mixed with the whole mass of the troposphere, then 10^{10} curies would produce the maximum permissible concentration. However, the assumption of world-wide tropospheric mixing is unwarranted for an isotope with a half-life of 8 days. Assuming the term large-scale contamination in the case of I^{131} can at most involve a 20° or 30° band of latitude in the northern hemisphere, and that vertical mixing may be incomplete, then even for large-scale considerations an atmospheric burden of 10^8 or 10^9 curies of I^{131} may approach the maximum permissible concentration, and appropriate cooling or decontamination measures must be used.

C. Local contamination

It is evident that consideration of the average contamination over major portions of the globe cannot approach the hazard to be found in local areas downwind from sources of contamination. Locally, higher concentrations that would exist 10 to 100 miles from fuel processing plants (assuming something of the order of 1% of the world's fuel to be processed at any single site) could add an additional factor of 10 to 100 in the case of Kr^{85} and several thousand in the case of I^{131}. Also, transitory excess concentrations due to unfavorable meteorological conditions could raise local concentration by an additional one to two orders of magnitude.

The above effects are cumulative so that concentrations of I^{131} about 10^4 times the global average could occur regularly near fuel processing plants in the northern temperate latitudes, rising occasionally to 10^5–10^6 times the global average during unfavorable meteorological conditions. Deposition by precipitation could increase the possibilities of harmful effects. Further detailed analysis would be required in order to indicate under what conditions the concentrations of krypton, iodine, or other isotopes would exceed permissible limits. In any case, it seems that a combination of reasonably conservative fuel cooling periods, some progress in off-gas cleaning, and a judicious choice of fuel processing locations, is indicated to minimize the adverse effects of unfavorable meteorological conditions. At the larger plants, meteorological scheduling of gas releases may be required. These principles are applied today, and will become increasingly important.

D. Accidental releases

There is the possibility, even if remote, that a large high-power reactor or fuel processing facility could be damaged or destroyed by accident and release part or all of the contained fission products to the atmosphere. The results of such an event could well be catastrophic, and extend over great distances. Estimates of areas of damage range upwards of thousands of square miles for very large reactors. By the year 2000 the release of only about 1% of the world-wide Sr^{90} inventory that could then exist, even if mixed uniformly throughout the global troposphere, could produce concentrations on the order of 5×10^{-10} curies m^{-3} or about twice the currently recommended maximum permissible concentration. This same 1%, if deposited on the surface, could seriously contaminate the entire area of the earth. It is more likely, in the event of such a catastrophe, that the activity would remain concentrated in a much smaller area near the source. Still, the operation of any significant fraction of the earth's nuclear reactors without proper safeguards would be of concern to all.

E. Conclusions

The solution to radioactive air pollution problem is the same as in other air pollution problems, prevention of the escape of pollutants to the atmosphere. Thus, primary consideration must be given to engineering features limiting the

escape of hazardous gases either during normal operations or accidents. As additional safety factors meteorological research to locate plants in areas where unexpected releases will do the least damage is desirable. Finally, it should be pointed out that the release of a hazardous substance by any country may affect other countries—particularly in the same latitude belt. International control to establish and maintain high standards of safe plant operation is essential.

CHAPTER III. USE OF RADIOACTIVITY IN ATMOSPHERIC STUDIES

A. Natural radioactivity

There exist two important sources of naturally occurring radioactivity in the atmosphere: (1) cosmic ray interactions in the stratosphere and (2) the rock and soil of the earth's outer crust. The study of the cosmic ray induced products entails considerable difficulties because of the low level of activity. On the other hand, the radioactive substances which originate in the earth can be detected and measured with relative ease.

Radon and thoron are released as gases in the radioactive decay of radium and thorium which are found in all rock and soil. The concentration of these gases and their distribution in the atmosphere is determined by their half-lives and meteorological conditions. Although it is considered generally that the relative amounts of the various natural activities are dependent on meteorology, very few correlations with specific meteorological parameters have been made, in spite of the fact that measurements have been carried out over a period of many years. At the present time, insufficient data are available to make reliable estimates of the global distribution of radioactivity in the air over land, although it is known that at some distance from large land masses the radioactivity concentration is exceedingly low. Measurements indicate that the amount of radon decreases rapidly with altitude to about one half the surface value at one kilometer.

Radon and thoron and their daughter products would seem to provide an easily detectable tracer for the study of the vertical "Austausch." Ground level measurements indicate that exchange phenomena within even a few feet of the surface have marked effects on the concentration of radioactivity. Such measurements might well be carried on in conjunction with micro-meteorological observations. From consideration of the lifetimes of the radioactive isotopes which are involved, it is obvious that even for relatively low wind velocities, horizontal transport of these radioactivities over distances of several hundred miles is entirely possible. The study of simultaneous variations in concentration over these distances should be valuable if the locations were carefully selected to avoid the effects of terrain. Land to sea measurements should be especially interesting.

Instances of increases in radon concentration coincident with air pollution have been reported. Since atmospheric radioactivity and pollution are strongly affected by the stability of the lower atmosphere this effect is not surprising. For the same reason it is quite possible that a relationship could be established with the tropospheric scattering of electromagnetic radiation.

Experiments have shown that the radon and thoron decay products are attached to submicron particulates. The details of the attachment process are not well understood; for example the relationship between various ionic species or the number and kind of nuclei. These radioactivities exist in the form of a readily detectable submicron aerosol which generally follows the surface wind pattern. These small particles, and incidentally other pollution, appear to be removed from the lower atmosphere in a matter of days, principally through precipitation. Further study of this removal process, carried out at different locations and for a variety of climatological conditions would perhaps shed some light on the scavenging efficiency of precipitation.

The natural radioactivity of precipitation is considerable and is easily measurable. The mechanism for the entrainment of the radioactive particles in rain droplets is not certain. From theoretical considerations, the probability for attachment of these very small particles in rain is quite low. It has been suggested that the radioactive ions could themselves act as condensation nuclei. On the other hand, there is the possibility that clouds of charged radioactive particles could act as a sort of "trigger" for electrical phenomena leading to cloud electrification and precipitation. Experimentally, the difficulties of working with large volumes of rainwater are partially offset by the large activities encountered. The actual air volumes swept out by precipitation is very great

and it would seem that there are possibilities for tracing air masses by using natural radioactivity.

Traditionally atmospheric radioactivity has been associated with atmospheric electricity and might well supplement studies in this field. The radon and thoron decay products are charged and can be collected by electrical means. They are estimated to cause about one half of the ionization in the lower atmosphere. Certain of the theories of atmospheric and cloud electrification are quite sensitive to changes in the ion concentration. Since large changes in the radioactivity concentration are the rule, further studies carried out in conjunction with atmospheric electrical measurements should be valuable.

The most extensively studied of the cosmic ray induced isotopes found in the atmosphere have been C^{14}, H^3 and Be^7. Probably both short term increases in fossil CO_2 from industrial sources and the long term global distribution could be detected using sensitive techniques. H^3 is present in the air principally in the form of tritiated water and will probably find its most useful applications to hydrology, although more extensive sampling of precipitation is no doubt desirable. Because of its relatively short half-life, Be^7 may be of very great importance in the study of the rate of mixing between the stratosphere and troposphere. Unfortunately, there is a great lack of experimental information suitable for correlation with meteorological phenomena.

B. Debris from weapons tests

The debris injected into the atmosphere from the testing of nuclear weapons can provide a useful tool for investigating atmosphere phenomena. However, two basic limitations on the usefulness of the approach must be recognized:

1. The source strength and distribution in space is largely unknown. Such important information as the distribution of particles with altitude, the exact configuration of the stabilized cloud, the relation of particle size to activity, the fractionation of elements within the cloud, etc., is not available.
2. Sampling techniques are imperfect. Air concentration measurements are difficult because of the low concentrations and small particle sizes involved. Ground collections result from either deposition of the particles themselves or by precipitation scavenging.

Using the gummed paper collection system described in Chapter I, it has been possible to obtain certain valuable meteorological information on such items as: a measure of the cross-equatorial transport and some feature of the general circulation from U. S. Pacific tests, scavenging by the upper portions of rain clouds of the particulate fission products, an estimate of rapidity of the removal of particulates from the troposphere, an estimate of the rate of transport from the stratosphere to troposphere.

Using aircraft sampling procedure, it has been possible to obtain estimates of the rate of lateral spread of an atmospheric contaminant and vertifications of meteorological trajectories.

By following the Tritium released by the CASTLE series of weapons tests it has been possible to estimate the removal time for atmospheric water molecules.

It is likely that the potential of even the existing unclassified information on radioactivity released by weapons tests has not been exhausted. This potential would be enhanced by disclosure of additional information on weapons, debris measurements, and source strengths. For example, the weapons tests offer an opportunity to determine storage and transit time parameters for surface water sheds of almost any size. By comparing the amount and level of radioactivity in rainfall and runoff as a function of time following a weapons test, it would be possible to measure those parameters which are vital to studies of ground water, river runoff, and flood forecasting.

C. Artifically introduced radioactive tracers

Artifically introduced radioactive tracers can serve meteorology in at least three fields: first, through the delineation of the airflow and rates of diffusion; second, in hydrometeorology, including studies of condensation, precipitation, evaporation and hydrology; and third, in atmospheric electricity.

As a tracer of air motions, radioactive substances are in competition with fluorescent dye particles, sulfur dioxide and other non-radioactive substances. Their advantages lie in the possibility of being able to treat large-scale atmospheric phenomena which otherwise require too large amounts of source material, in being able to utilize tracers which partake in the particular process under

investigation and, in certain cases, in our ability to detect the presence of the tracer instantaneously in the field. In any specific experiment it will be necessary to weigh economic, safety and scientific factors in the use of radioactive tracers over non-radioactive tracers.

Regions in which it would be highly desirable to further knowledge concerning air trajectories are in the neighborhood of jet streams, in cols, in hurricanes to measure both the three dimensional airflow and define the air comprising the eye, and in the Antarctic. In the field of dffusion, the use of radioactive tracer material can further knowledge of diffusion near the ground for air pollution studies, etc., and of diffusion in the stratosphere and tropospheric and stratospheric mixing.

The radioactive tracer material which appears to be most promising for the above meteorological studies is tritium. Tritiated water would be washed out, thus making for additional complications. Tritium in the form of ordinary hydrogen is acceptable although costs of analysis of the sample might be high. For the large-scale experiment to establish the tropospheric-stratospheric exchange tritiated methane has been suggested. Tritium has the advantageous properties of emitting a weak beta particle, of being available without difficulty, and of having a reasonably long half-life.

Water molecules are readily marked by tritium so that in any experiment in which the travel of water vapour is desired it becomes feasible to introduce tritiated water as a tracer. If sufficient amounts of tritium were available, a large-scale experiment to study the hydrologic cycle could be devised. Even on smaller scales, tritiated water could be used to study such features as the evaporation from a ponded lake, water sources for dew, contributions of local transportation or evaporation from local bodies of water to precipitation elements, etc.

Activation analysis techniques extend the possibilities for studying very small particles (such as sodium chloride) that play an important role in condensation and ice formation. Radiosilver can be introduced in a preparation of silver iodide to be able to determine the presence of silver iodide in the precipitation which was alleged to be stimulated by it. By releasing another tracer which would be scavenged with equal efficiency by precipitation it might be able to determine whether the silver iodide has played a role in the formation of the precipitation.

Finally, the ionizing properties of radioactive substances can be used to make local changes in the electrical fields of the atmosphere, to determine if such changes affect weather processes.

CHAPTER IV. THE EFFECT OF ATOMIC EXPLOSIONS ON WEATHER

A. Introduction

From the beginning of time, man has looked beyond the field of meteorology in the hope of finding some explanation for the vagaries of weather. Many inventions of man—gunpowder, radio, airplanes, and television—have been blamed for changes in weather and climate. It is only natural that atomic and thermonuclear explosions, being among the most dramatic achievements of mankind, would come in for their share of the blame.

There seems to have been an increase in unusual and undesirable weather in the past decade. When submitted to rigorous statistical tests, these apparent abnormalities do not exceed the limits that can be expected by chance and are consistent with accepted meteorological principles involving large-scale (hemispheric) weather patterns which could not be directly affected by the explosions. The failure to detect statistically significant changes in the weather during the first ten years of the atomic age is no proof that physically significant changes have not been produced by the explosions, but it does show that a careful physical analysis of the effects of atomic and thermonuclear explosions on the atmosphere must be made.

The energy of even a thermonuclear explosion is small when compared to most large-scale weather processes. Moreover, it is known that much of this energy is expended in ways that cannot directly affect the atmosphere. Even the fraction of the energy which is directly added to the atmosphere is added in a rather inefficient manner from the standpoint of affecting the weather. Meteorologists and others acquainted with the problem are readily willing to dismiss the possibility that the energy released by the explosions can have any important direct effect on the weather processes. However, there remains the possibility that

the explosion will serve as a trigger mechanism to divert some much larger natural store of energy from the path it would otherwise have followed.

Three general means by which this might be accomplished have been considered:

1. The debris thrown into the air by the explosion may have some catalytic effect on the behavior of clouds and thereby change the regime of cloudiness or precipitation over wide areas.

2. The radioactive nature of the debris will change the electrical conductivity of the air, and this may have some effect on more directly observable meteorological phenomena.

3. The debris thrown into the stratosphere by the explosion may interfere with the passage of solar radiation and thereby serve to decrease the temperature of the earth.

Our present knowledge of atmospheric physics makes difficult a final authoritative evaluation of any of these possibilities.

The results of studies and experiments conducted by various organizations show the following:

1. The debris which has been thrown up into the atmosphere by past detonations was found to be ineffective as a cloud-seeding agent. Since the techniques for testing nucleating efficiency are not entirely satisfactory, the condensation and freezing nuclei produced by nuclear explosions and their effect on the formation of clouds and the precipitation process must be continually investigated.

2. The amount of ionization produced by the radioactive material is insignificant in affecting general atmospheric conditions. Various theories on the possible connection between the electrical properties of the atmosphere and the precipitation process are still in the developmental stage.

3. Dust thrown into the air by past volcano eruptions decreased the direct solar radiation received at the ground by as much as 10–20%. The contamination of the atmosphere by past nuclear tests has not produced any measurable decrease in the amount of direct sunlight received at the earth's surface. There is a possibility that a series of explosions designed for the maximum efficiency in throwing debris into the upper atmosphere might significantly affect the radiation received at the ground.

4. Much of the increase in severe storms reported in recent years can be traced directly to the improved methods of reporting severe storms that normally occur.

No statistically significant changes in the weather during the first ten years of the atomic age have been found, yet careful physical analysis of the effects of nuclear explosions on the atmosphere must be made if we are to obtain a definite evalution of this problem. Although it is not possible to prove that nuclear explosions have or have not influenced the weather, it is believed that such an effect is unlikely.

HARRY WEXLER, *Chairman*
LESTER MACHTA, *Rapporteur*
CHARLES E. ANDERSON
R. R. BRAHAM, Jr.
MERRIL EISENBUD
D. LEE HARRIS
B. G. HOLZMAN
H. G. HOUGHTON
W. W. KELLOGG
HEINZ LETTAU
N. M. LULEJIAN
R. J. LIST
WILLIAM K. WIDGER

SUMMARY REPORT OF THE COMMITTEE ON EFFECTS OF ATOMIC RADIATION ON OCEANOGRAPHY AND FISHERIES

1. To Whom Is This Report Addressed?

In writing this report we have had four groups in mind—research administrators, statesmen, scientists and the public. For those who have responsibility for the support of research, we have attempted to outline the scientific questions that need to be answered as a basis for intelligent policy, the means by which they can be attacked by classical research methods at the outset, and the broader problems of the oceans that can be hopefully attacked by the use of radioactive tracers. For the statesmen who have responsibility for national and international policy, we have attempted to formulate recommendations, based on our present small body of knowledge and our awareness of our larger area of ignorance, concerning the national and international actions and agreements that are necessary for the happy exploitation of the oceans in the new atomic age. For the scientists, we have attempted to summarize what is known about the actual and potential effects of radioactive materials in the oceanic realm and the interest of marine

scientists in these substances. For the public, to which we all belong when we are outside our own specialties, we have summarized the levels of calculated risk that must be balanced against the wonderful promise of atomic energy for the welfare of mankind.

2. How Does the Atomic Energy Program Affect the Oceans?

We have considered three aspects of the atomic energy program that directly involve the oceans and, therefore, the marine sciences: weapons tests over or in the sea, disposal of radioactive wastes from nuclear power plants, and the use of radioactive substances in increasing our understanding of the oceans and of the creatures that live in the sea. These different aspects cannot easily be separated. Weapons tests and the disposal of radioactive wastes present great opportunities for studying the oceans. On the other hand, increased knowledge of the oceans is essential to avoid or minimize the destruction of marine resources in the development of atomic energy.

The continuing development of atomic energy will produce progressively greater amounts of radioisotopes, and with them greater amounts of radioactive waste material. Since the oceans cover 71% of the earth, and ultimately receive the drainage from the land, they are the principal reservoir where radioisotopes will finally accumulate. Relatively small quantities are now being added to the surface waters of the ocean as fallout from weapons testing programs, and in a few places as waste materials.

When nuclear reactors for the production of power are put into large-scale operation, as they certainly will be in the foreseeable future, the oceans will be seriously considered for the disposal of large quantities of wastes. Even if direct and intentional disposal at sea is not practiced, reactors may be built along seacoasts or on rivers near large population centers and accidental pollution may occur.

The problem of disposal of radioactive wastes is similar in character to, though potentially far greater in scope than other problems of pollution. An object lesson can be drawn from our experience with the disposal of human and industrial wastes in inland water bodies and coastal waters and with the smog problem that afflicts many of our large cities. During the early stages of the growth of industries and populations in cities, wastes were added to nearby lakes or bays, and to the air, in what seemed at the time to be innocuous quantities. As a matter of fact, the quantities were small enough to be purified by natural processes. In the course of time, however, the quantities increased insidiously so that today many natural waters cannot purify themselves and without expensive treatment are dangerous to humans.

In almost every case the problem was ignored until it had become formidable in magnitude. Short-range solutions were employed, based on inadequate knowledge, special interest, and what we now know was an unfounded confidence in the capacity of the atmosphere and the waters to absorb noxious substances. As a result, unnecessary damage was done to human beings and their environment. Much of this could have been avoided if an adequate program of scientific investigation had been started sufficiently far in advance and if scientifically based policies had been followed.

It is imperative that the nature of the wastes associated with the development of atomic energy be evaluated in advance. We know that purification of waters receiving radioisotope waste will proceed only by dilution, by precipitation and settling on the bottoms, and by the decay of radioactivity. Nothing could be done to reverse an undesirable accumulation that might result from ill-considered disposal of this type of waste.

There is no question of trying to keep all of this material out of the sea. It is certain that some of it can be safely added. Tolerability of materials must be determined, and the locations where they should be put must be wisely selected in terms of the quantity and character of the radioactivity. It is not possible today to see clearly the problems of the future; we can only define the studies that must be made to provide a scientific basis for wise evaluation, and urge that these studies be begun without delay. The costs of such studies may seem large, but they are actually negligible in terms of the potential benefits. They are also very small when compared to the total present expenditures for the development of atomic energy. We cannot wait to begin these studies until radioisotope pollution becomes serious, for it is irreversible.

3. Is There Naturally Occurring Radioactivity in the Sea?

Yes, but one of the remarkable characteristics of the ocean is the extremely low level of the natural radioactivity. Marine animals and plants living more

than a few hundred feet beneath the surface are bombarded by much less natural radiation (radioactivity plus cosmic rays) than is received by terrestrial plants and animals.

For example, although radio potassium accounts for about 90 percent of the activity in the sea, it is present in most igneous rocks at about 100 times the concentration found in the ocean. Uranium, radium, and thorium are 3,000 to a million times more concentrated in rocks than in the sea. This raises an interesting scientific question concerning the character of genetic change and evolution in many marine creatures. It emphasizes the need for basic biological studies on marine organisms. Because of their experimental difficulty, such studies have been comparatively neglected during the past few decades.

4. Have Weapons Tests Added Measurable Amounts of Radioactivity to the Sea?

Yes, though in terms of the total radioactivity of the sea the amount is negligible. Radioactivity in the waters of the test area is of course very greatly increased at the time of tests, and even after diffusion over thousands of miles concentrations remain that are readily detectable. Two days after the 1954 tests in the Pacific the radioactivity of the surface waters near Bikini was observed to be a million times greater than the naturally occurring radioactivity. This material was transported and diluted by ocean currents, and four months later concentrations three times the natural radiation were found 1,500 miles from the test area; thirteen months later the contaminated water mass had spread over a million square miles. Artificial activity had been reduced to about one-fifth the natural activity, but could be detected 3,500 miles from the source.

5. In What Other Ways Will Radioactive Materials be Added to the Oceans?

In England radioactive wastes are being piped into the Irish Sea from an atomic installation. In the United States, wastes from laboratories and hospitals are being carried to sea in containers and dumped. At Oak Ridge, some of the fission products are discharged into the Tennessee River system. At Hanford, water from the Columbia River is used for cooling and returned to the river with some induced short-lived radioactivity. Waste products from the uranium fuel processing plants are now being confined, some in containers, others in pits in the ground. When the power reactors and fuel processing plants reach their expected development many rivers will have to be used. It will not everywhere be practical to confine the wastes locally. Transporting them to sea in barges or by other means may then be necessary in many cases. Although we may be sure the atomic installations will be carefully engineered and maintained, accidental discharge of waste may occasionally occur. On those occasions intense radioactivity may reach the sea.

6. Has the Atomic Energy Program as Yet Resulted in Serious Damage to Marine Life?

Probably no. We know that radioactive radiation is damaging to living things and that marine organisms tend to concentrate many fission product elements. But there is no evidence that any lasting damage has been done to the animal or plant populations of the sea or large inland water bodies by the release of radioactive substances.

Certainly in the weapons test area terrestrial forms were killed or injured by the tests. The evidence concerning marine life is not conclusive, but biologists feel certain that deleterious effects occurred in the near vicinity. There is, however, no evidence that populations have been affected after the dilution and transport mentioned above. This is a subject on which intensive studies are essential before a definite answer can be given. We know that "high" levels are lethal, and that "low" levels may have no direct effect, but we cannot give quantitative values for "high" and "low" except in a few cases. Low levels, which produce no measurable effect in the organism itself, may produce genetic effects and thus influence the marine populations in the future, but there is no conclusive evidence that this will be undesirable.

7. Do Living Things Take Up Radioactive Materials into Their Bodies?

Yes. Radioactive materials added to the sea can remain in solution, precipitate and settle on the bottom, or be taken up by the plants and animals that live in the water. The plants of the sea are mainly microscopic in size, but they can concentrate many thousand-fold those elements that are necessary to them. Radioactive substances are also absorbed on the body surfaces of living things. Small plants and animals serve as food for the larger forms and the radioactive materials are passed on from one to another. The amount of each element accumu-

lated in each form depends upon the rate at which it is taken up, either directly or as food, and the rate it is excreted. Some of the radioactive materials remain in the body for relatively long periods of time and may accumulate to a considerable degree. Others may be lost rapidly and very little will accumulate.

This statement is a great over-simplification. Different plants and animals require and accumulate different elements. Shell fish, for example, concentrate calcium and strontium in their calcareous shells; fish concentrate zinc. It will be necessary to know among other things both the composition of the waste, and the populations in the area, before any particular disposal operation can be evaluated.

8. Are All the Radioactive Elements Equally Harmful?

No. Those elements that living organisms naturally accumulate and that have long radioactive half-lives are more harmful than others. Radioactive strontium, and to a lesser extent, cesium and its daughter barium, cerium, praesodymium and promethium represent particular hazards to human beings from ocean disposal.

9. How Much Radioactive Waste Will be Produced by Nuclear Power Reactors in the Future?

The answer to this depends upon how optimistic one is concerning the development of nuclear power. One estimate assumes that within about 50 years nuclear fission will be producing about half as much power annually as the peoples of the world are using today from all sources.

Accumulations year after year will eventually result in a constant quantity of radioactivity, such that the rate of radioactive decay will balance the rate of production of fission products to give what has been called the steady state. This should be approached within a few decades after full production is reached. The waste radioisotopes at this point would equal between one and two times the total natural radioactivity in the world oceans. This is roughly a thousand times the amount produced so far in weapons tests.

10. What Means Are Being Considered for Disposing of Radioactive Wastes?

The methods being considered fall into two categories, isolation and dispersal. It is probable that a judicious combination of the two methods for different types of wastes or for different countries will be essential. Chemical treatment of the wastes to isolate usable fractions, or those, like strontium and caesium, that decay most slowly, offers promise in simplifying the problem. For isolation, permanent storage in tanks or introduction into geological structures such as salt domes are being studied by other committees. The only place on earth where dispersal can be considered practical is in the ocean. Because it is large and fluid, the ocean could provide immense dilution. Because of its depth, and the stratification of water-masses with differing densities, various degrees of isolation may be possible. It is a prime purpose of this report to emphasize the need for investigation as to whether this possible isolation is adequate.

11. Will it be Safe to Introduce Very Large Quantities of Radioactive Wastes from Atomic Power Indiscriminately into the Sea?

The answer is certainly no, but the strongest negative must be given for coastal waters and for the upper water layers everywhere that are the home of commercially important fishes. These surface waters interconnect and are in continuous motion. Anything added in one spot will, in the course of a few decades at most, be carried to all parts of the world. There is no place in the sea where very large amounts of radioactive materials can be introduced into the surface waters without the probability of their eventually appearing in another region where human activities might be endangered.

It should not be forgotten that the coastal waters enter the harbors and estuaries and would carry any waste materials there with them; and that many of the major fishery resources of the world are concentrated over banks and near coasts, and would become contaminated.

We must also remember that all plants and animals in the sea, from the smallest bacteria to the largest whale, play a part in concentrating, transporting, and dispersing radioactive and other dissolved and suspended materials.

12. Does This Mean that Large Quantities of Radioactive Wastes Should Never be Dumped in the Sea?

No, not necessarily, but it does mean that the length of life of the radioactive material, its role in biological processes, and the mixing rate of the ocean should be carefully studied before large quantities of wastes are introduced into the

sea. Unfortunately, although we know the decay time of most radioactive substances, we know very little about the exchange processes in organisms and in the water. We do know that even the bottom waters of the deep ocean basins slowly exchange with those of the surface, but the rate of this exchange is uncertain.

13. From What Is Known, Where Would be the Safest Place to Dump Radioactive Wastes in the Sea?

At the present time it is only possible to give rough engineering estimates based on order-of-magnitude calculations.

Remembering the importance both of isolation (to allow time for radioactive decay) and dispersal (to reduce the amount of radioactivity per unit volume) the problem is to find places in the ocean where the rate of transfer of radioactive materials to the surface waters would be slow, or where great dilution would occur before radioactive materials came in contact with marine food products or human beings, and preferably where both conditions would prevail.

There are some places where a contaminant could be isolated for long periods. For example, it is estimated that in the deepest parts of the Black Sea the "flushing time" is about 2500 years. This is the time required for most of the deep water to move near to the surface and be replaced with new water mixing downward. In this respect the Black Sea is unique. Elsewhere the "age" of the deep water indicates that exchange with the near surface waters goes on less slowly. Thus in the deeps of the Atlantic and Caribbean the time required for replacement of the water with new water from near the surface is probably only a few hundred years. Some oceanographers believe that the Atlantic deep water sank from the surface in high northern latitudes about 150 years ago.

We are fairly certain that substantial amounts of long-lived radioactive materials, dumped on the bottom in the deep sea, would remain isolated for more than 100 years and that during this period they would become diluted by mixing through an enormous volume of deep water. We do not understand the nature of the physical and biological exchange processes between the deep and surface waters well enough to be able to say whether in the steady state, after decades of nuclear power production, deep sea disposal would give adequate protection of the commercial fisheries from long-lived fission products such as strontium. Large quantities of short-lived fission products could certainly be disposed of safely in this way.

14. Can Radioactive Materials be Used to Learn About the Oceans and to Increase the Harvest from the Sea?

Yes. For example, an understanding of the flow of material through food chains is essential to the effective use and conservation of the food resources of the sea. The natural elements used by the marine plants and their transfer to the commercially valuable fish and shellfish can be studied on a large scale, using radioactive isotopes. As these readily detectable substances are traced through the various steps of the food chain—plants, animal plankton, small fish, large fish—the efficiencies and inter-relationship of the various levels should become much better known. This knowledge is of fundamental importance for the evaluation of the potential of the living resources of the sea as a source of food and other marine products, and as a basis for their full utilization and conservation.

Radioactive materials, both natural and man-made, can also be used in the study of oceanic mixing processes and circulation. These processes serve to supply marine plants with the fertilizers they need from deeper waters, as well as to dilute and disperse radioactive wastes dumped in the sea. At present we cannot measure, but can only estimate the mixing rates. The ability to trace radioactive materials, even though present in great dilution, will permit us to obtain quantitative information. Improved knowledge of the mixing processes and of currents will help man to locate and evaluate unexploited resources of fish and other food organisms.

For example, thirteen months after radioactive materials were introduced into the seat fallout from weapons tests in the Marshall Islands, a research vessel traced their distribution in the Western Pacific. The extent to which radioactivity was taken up by plankton and fish was measured, as well as the extent to which activity was mixed downward and transported westward in the western limb of the great North Pacific eddy. These measurements showed the average speed at which materials were carried away from the test area, giving convincing proof of the transport and mixing of material over a vast region.

Large amounts of radioactive tracers ranging in magnitude from curies to megacuries can be used at sea in studying oceanographic problems, including the problems of fisheries, and thus laying the ground work for increasing our harvest from the ocean. Smaller amounts are needed in the laboratory. We are here concerned not with the general problems of physiology and biochemistry but with specific ecological studies, including investigations of the efficiency of transfer of energy along the food chain, rates of filtration, concentration of elements and compounds in various tissues, the rates of accumulation and excretion of elements and compounds, the passage of substances across biological membranes, the concentration and role of biotic and antibiotic substances in the sea, the dynamics of marine populations, including the mass of living material in a given volume of water, the flux of organic substances from one organism to the other and between the organism and the sea water, and the inter-relations of animal and plant communities. In both field and laboratory experiments fission products are useful but some problems require the use of artificially radioactive substances produced by other means. An outstanding example is the use of carbon 14 to study the efficiency of various steps in the food chain. Large quantities of this material are needed for field studies in restricted water bodies. Though the cost would be high, the value of the results would more than justify the expenditure.

CONCLUSIONS AND RECOMMENDATIONS

1. Tests of atomic weapons can be carried out over or in the sea in selected localities without serious loss to fisheries if the planning and execution of the tests is based on adequate knowledge of the biological regime. The same thing is true of experimental introduction of fission products into the sea for scientific and engineering purposes.

2. Within the foreseeable future the problem of disposal of atomic wastes from nuclear fission power plants will greatly overshadow the present problems posed by the dispersal of radioactive materials from weapon tests. It may be convenient and perhaps necessary to dispose of some of these industrial wastes in the oceans. Sufficient knowledge is not now available to predict the effects of such disposal on man's use of other resources of the sea.

3. We are confident that the necessary knowledge can be obtained through an adequate and long-range program of research on the physics, chemistry, and geology of the sea and on the biology of marine organisms. Such a program would involve both field and laboratory experiments with radioactive material as well as the use of other techniques for oceanographic research. Although some research is already underway, the level of effort is too low. Far more important, much of the present research is too short-range in character, directed towards *ad hoc* solutions of immediate engineering problems, and as a result produces limited knowledge rather than the broad understanding upon which lasting solutions can be based.

4. We recommend that in future weapons tests there should be a serious effort to obtain the maximum of purely scientific information about the ocean, the atmosphere, and marine organisms. This requires, in our opinion, the following steps: (1) In the planning stage committees of disinterested scientists should be consulted and their recommendations followed, (2) funds should be made available for scientific studies unrelated to the character of the weapons themselves, and (3) the recommended scientific program should be supported and carried out independently of the military program rather than on a "not to interfere" basis.

5. Ignorance and emotionalism characterize much of the discussion of the effects of large amounts of radioactivity on the oceans and the fisheries. Our present knowledge should be sufficient to dispel much of the over-confidence on the one hand and the fear on the other that have characterized discussion both within the Government and among the general public. In our opinion, benefits would result from a considerable relaxation of secrecy in a serious attempt to spread knowledge and understanding throughout the population.

6. Sea disposal of radioactive waste materials, if carried out in a limited, experimental, controlled fashion, can provide some of the information required to evaluate the possibilities of, and limitations on, this method of disposal. Very careful regulation and evaluation of such operations will, however, be required. We, therefore, recommend that a national agency, with adequate authority, financial support, and technical staff, regulate and maintain records of such disposal, and that continuing scientific and engineering studies be made of the resulting effects in the sea.

7. We recommend that a National Academy of Sciences-National Research Council committee on atomic radiation in relation to oceanography and fisheries be established on a continuing basis to collect and evaluate information and to plan and coordinate scientific research.

8. Studies of the ocean and the atmosphere are more costly in time than in money and time is already late to begin certain important studies. The problems involved cannot be attacked quickly or even in many cases, directly. The pollution problems of the past and present, though serious, are not irremediable. The atomic waste problem, if allowed to get out of hand, might result in a profound, irrecoverable loss. We, therefore, plead with all urgency for immediate intensification and redirection of scientific effort on a world-wide basis towards building the structure of understanding that will be necessary in the future. This structure cannot be completed in a few years; decades of effort will be necessary and mankind will be fortunate if the required knowledge is available at the time when the practical engineering problems haye to be faced.

9. The world-girdling oceans cannot be separated into isolated parts. What happens at any one point in the sea ultimately affects the waters everywhere. Moreover, the oceans are international. No man and no nation can claim the exclusive ownership of the resources of the sea. The problem of the disposal of radioactive wastes, with its potential hazard to human use of marine resources, is thus an international one. In certain countries with small land areas and large populations, marine disposal of fission products may be essential to the economic development of atomic energy. We, therefore, recommend: (1) that cognizant international agencies formulate as soon as possible conventions for the safe disposal of atomic wastes at sea, based on existing scientific knowledge; and (2) that the nations be urged to collaborate in studies of the oceans and their contained organisms, with the objective of developing comparatively safe means of oceanic disposal of the very large quantities of radioactive wastes that may be expected in the future.

10. Because of the increasing radioactive contamination of the sea and the atmosphere, many of the necessary experiments will not be possible after another ten or twenty years. The recommended international scientific effort should be developed on an urgent basis.

11. The broader problems concerned with full utilization of the food and other resources of the sea for the benefit of mankind also require intensive international collaboration in the scientific use of radioactive material.

Roger Revelle, *Chairman*
Howard Boroughs
Dayton E. Carritt
Walter A. Chipman
Harmon Craig
Lauren R. Donaldson
Richard H. Fleming
Richard F. Foster
Edward D. Goldberg
John H. Harley
Bostwick Ketchum
Louis A. Krumholz
Charles R. Renn
M. B. Schaeffer
Allyn C. Vine
Lionel A. Walford
Warren S. Wooster

Report of the Committee on the Effects of Atomic Radiation on Agriculture and Food Supplies

I. General

The Committee interpreted its task as requiring its members to survey the scientific aspects of that great sequence of events which precedes the delivery of food items to the ultimate consumer, and to do so from two separate viewpoints. These were (1) the beneficial effects that may result from the deliberate involvement of radiation of any sort with constructive intention, or what has been spoken of so frequently as the "peaceful uses of atomic energy," and (2) the harmful or disadvantageous effects of radiation of any sort due to nuclear warfare, to accidents involving atomic power plants, or even to a slowly rising background of radiation that conceivably may follow as a result of atomic technological developments in industry.

Public and private funds are currently being expended in the United States for research in agriculture and food processing at a rate in the vicinity of 300 million dollars annually. And undeterminable but not insignificant fraction of this considerable body of research involves radiation or radioisotopes. Members

of the Committee did not believe it to be incumbent upon them to defend or justify, to criticize or to challenge applications of atomic radiation to agriculture that have been developed or are under discussion. They did not wish to evaluate the programs of particular agencies or groups, but instead with judicial mind to examine the accomplishments and the potentialities, the implications and the limitations of radiation as related to the production and processing of agricultural products.

One broad conclusion is that there is not imminent any drastic change in *agricultural production* as a result of the application of radiation. However, radiation techniques provide new tools for research and may aid agricultural production by improving and enhancing the efficiency of production methods.

The Committee is strongly of the view that the applications of radiation will be of far greater immediate consequence to agricultural research than directly to agriculture, and that most of the benefits that may arise to agriculture, as manifest in the availability of an adequate and varied supply of wholesome food for man, wherever he may be, will come as a summation of many improvements, small and large, in materials, in plants and animals and in the technology of husbandry and processing developed through programs in agriculture and food processing research.

Changes therefore may be expected to come in a series of little steps, none of which in themselves may be of great impact, but which, through the years, are likely to be impressive in their total.

Another broad conclusion is that the slowly rising background of radiation caused by weapons testing in peacetime at the present rate is not likely to impair or interfere with food production. Levels of radiation considered tolerable by man are below those believed to have effects in plants or animals that would place food production in jeopardy. However, the high levels of radiation which might develop in small or large areas as a result of atomic or thermonuclear weapons in wartime, or from mishaps with nuclear power plants in peacetime could have catastrophic effects on agricultural production that might be of long duration, because of injury to personnel and animals, disruption of services, and contamination of soil, vegetation and water supplies.

II. TRACER STUDIES IN AGRICULTURAL RESEARCH

In the consideration of the beneficial effects of radiation the Committee endeavored, not wholly successfully, to separate in its thinking those benefits that may arise from additions to the pool of basic knowledge about plants and animals and their welfare, from those more direct effects that may specifically result from the exposure of plants, animals or agricultural products to radiation. Tracer studies in the biological sciences have already been enormously fruitful in aiding the elucidation of essential metabolic processes in plants and animals, and may be expected to be increasingly so as the number and diversity of such experiments increases. When there is knowledge and understanding of a process then comes the opportunity to control it for a desired end; in this way the art of agriculture is transformed to the science of agriculture.

They endeavored to make the separation mentioned above because of the conviction that there is nothing unique about radioisotopic studies as applied to agricultural research. Tracer techniques, however, frequently permit answers to be obtained to questions which seemed previously unanswerable by conventional experimentation. The involvement of isotopes puts a new dimension into metabolic studies, and areas, formerly dark, may now stand out in relief.

It is worthy of comment that many of the applied problems involved in the arts or technology of agriculture are as susceptible to study by procedures involving radioisotopes as are those more basic questions of plant and animal physiology or nutrition. Excellent examples of this type of employment of isotopes are to be found in work on the placement and recovery of phosphorous fertilizers in soils, the efficiency of various methods of application of insecticides, fungicides and herbicides, the determination of post-harvest residues of such chemicals, the extent of utilization of feed components by animals, etc. It is to be anticipated that there will be greatly increased use of tracer radioisotopes in the solution of such applied problems, and that the immediate dividends from such research may be considerable. Further, it is likely that new methods of employing isotopes advantageously will be developed; the ingenuity of investigators in this field should not be underestimated.

Because of the unanimity of their views as to the enormous potentialities of isotope tracers as a research tool in agricultural science and biology generally, the Committee gave some consideration as to whether there are limitations in

facilities for training or funds for specialized equipment for such studies. The consensus seemed to be that motivation for the use of such techniques must come from individual investigators themselves, that the necessary know-how is to be found in almost all research institutions, and that progress in agricultural research is not at the moment limited by inadequacies in dissemination of knowledge and techniques. There was, however, a feeling that much of the graduate training in this field is rather informal, that more universities might consider establishing courses in which the methodology, techniques and principles of this new and powerful science are expounded, and that there is an additional need for an advanced training program for specialists in radiochemistry and radiobiology who may be developers of new techniques or interpreters of new applications of potential value in agricultural research.

III. EFFECTS OF RADIATION ON CROP PRODUCTION

It is abundantly established that mutations can be induced in many plant species by exposure to x-radiation, gamma radiation and other forms of radiation. The changes which result are possibly due to chromosome deletions or abberations. There is some difference of opinion as to whether radiation-induced mutants intentionally obtained are qualitatively identical with those which occur spontaneously from naturally occurring mutagenic agents, but there is no doubt that their frequency is increased. Even so the mutation rate in most species is still very small, and furthermore most mutations are disadvantageous. The investigator seeking to exploit this phenomenon must expect to have to handle very large populations, and so far has been able to look only for desirable changes that are reflected in morphology or appearance and therefore can readily be seen, or for changes which can be recognized by some blanket method such as inoculating all irradiated plants with disease organisms in the hope of finding one or more exhibiting resistance to infection.

It is likely that characters at present unrecognized also undergo change and that there are unexplored potentialities for effecting improvement in quality that may alter the demand for the plant, or in physiological properties that may alter the relationships of the plant with its environment.

It would be a mistake to imply that this new development has greatly simplified the tasks of those involved in crop improvement. On the contrary, it has made them more complex, but, by extending the boundaries, offers many new possibilities. It is not to be expected that acceptable new agronomic varieties can be obtained by simple irradiation of present varieties, though this is possible if large enough populations are examined. In general, however, back-crossing and recombination are needed to add the new characteristic to a crop plant acceptable in other respects.

As yet relatively few new varieties of economic plants, developed from radiation-induced mutants, have actually been introduced and widely planted. These, however, do attest to the potentialities of the procedure. Much of the research effort in this field has properly been devoted to the investigation of techniques, to such vital questions as the determination of the particular stage of development at which radiation exposure may be most effective, and the comparative mutability of crop species. It appears that different species cannot be expected to respond in an identical manner. More perhaps is known about this aspect of corn genetics than of any other major crop plant.

Mutations in micro-organisms may similarly be induced by exposure to various types of radiation, though at considerably higher radiation levels than with crop plants. The changes induced have been shown to include the degree of virulence and host range of certain pathogenic fungi. The suggestion has repeatedly been made that the plant pathologist should examine this phenomenon so as to anticipate disease-resistance requirements in a breeding program. As yet, however, there have been no significant results along these lines. Considerable success has been achieved in the development of greatly enhanced antibiotic production by some molds through radiation-induced mutation and selection. Similar genetic changes in the case of other micro-organisms have produced information about the likelihood of genetic control of metabolic processes.

There is considerable evidence that bud mutations or somatic mutations can be induced by radiation, and that this phenomenon can be exploited in the development of new strains of crop plants that are normally propagated by cuttings and grafting. This may be of special value in the improvement of some such crops, but as yet there have been no striking accomplishments in this direction. Progress in such studies is however inevitably slow because of the

nature of the materials, the length of time necessary to recognize a desirable change, and to produce the stocks necessary for field evaluation.

Since the mutation rate of plants may be enhanced by radiation, presumably there is some possibility of the appearance of undesirable mutants in areas where the background radiation becomes higher than normal for any reason. This may be of some significance in connection with waste disposal practices or atomic accidents. There is, however, no evidence of such changes in areas containing radioactive springs or ores. This may be due to lack of intensive examination of the vegetation of such areas, and such surveys are to be encouraged. However, the likelihood of appearance of undesirable lines under radiation levels that would be tolerated on other grounds seems small.

There is no evidence that plant growth is stimulated or crop yields increased by exposure to low levels of radiation, despite earlier well-publicized claims to this effect. Radioactive fertilizers, used in a conventional manner, produce yield increments no greater than expected from ordinary fertilizers.

Plants accumulate nutrient elements present in the root zone in solution or absorbed onto soil colloids, but non-nutrient elements are not excluded and may similarly be taken up. The availability of radioisotopes has greatly improved the understanding of plant nutrition and soil-plant relationships, and may be expected to aid substantially in the improvement of cultural practices, as indicated earlier. Through the use of isotopes it has been demonstrated unequivocably that certain elements can enter the plant through the leaves. This is of some consequence in relation to fall-out. Radioisotopes of long life or high activity if deposited in fall-out from an atomic or thermonuclear incident are likely to be accumulated in crop plants by root uptake from the soil and entry through the foliage. Some of the products deposited may be initially quite insoluble, but may become soluble through weathering. Others, initially soluble, may be irreversibly fixed by many soils in a form not readily available to crops. It appears at present that Sr^{90} and I^{131} are the chief radioactive elements which are of concern in such circumstances. The subsequent use of such crops presents a great diversity of problems depending on the level of radioactivity, its nature and the specific use of the crop. The Committee was interested to learn that the Department of Agriculture is preparing for farmers some informational material relating to these problems.

The Committee desires to examine further the available information on the inter-actions of fall-out components with soil their entry and accumulation in crop plants in order to determine whether there is available the necessary basic information from which appropriate agronomic recommendations could be formulated for agricultural operations in areas that may have undergone any likely level of contamination.

IV. EFFECTS OF RADIATION ON ANIMAL PRODUCTION

Whereas it appears that crop improvement programs may be considerably aided by the availability of radiation-induced mutants that may have certain desirable characteristics capable of incorporation into an agronomically acceptable variety, currently available evidence does not suggest that a similar approach with animals would be so rewarding. This statement is made not from a belief that farm animals are inherently less responsive to radiation than plants but because physical differences of size, cost, generation time, etc., militate against extensive studies with animals, and act as obstacles that cannot readily be overcome. Probably only with poultry and to a lesser degree with swine would it be possible to handle large enough populations, and even here, if one extrapolates from the smaller laboratory animals, the chances of improvement seem slim. At present one such study, with chickens, is known to be underway.

Limited whole-body exposure studies with farm animals have primarily been carried out to investigate physiological and pathological changes, often with the intention of transferring the information by analogy to problems of responses in man. The sequence of changes induced in most farm animals by heavy radiation exposures has been well defined. There are one or two examples however of the use of radiation exposure as a research tool for inhibiting certain functions in animals. For example various functions in the oviduct of poultry can be blocked by proper radiation techniques thereby permitting a study of the contribution made by the parts of this organ.

Much of the work with radioisotopes in the animal field centers around problems of animal nutrition and metabolism, and substantial progress has been made both in the elucidation of fundamental problems of animal physiology as

well as in those of a more applied character, such as the utilization of feed constituents, and the incorporation in animal tissues of inorganic constituents of forages. The experimenters in this field at present encounter one serious difficulty, which in the case of the larger farm animals greatly limits the scale of activity. This is the problem of the salvage or disposal of animals after use in experiments involving radioisotopes or radiation exposure. Even in the case of short half-life isotopes and at tracer levels only, the animals cannot be marketed through the usual outlets. This problem is of course much more serious with dairy or beef cattle than with hogs or poultry because the cost to the program is so much greater. Moreover, this limitation tends to restrict undesirably the scale and scope of such experiments, with the result that the conclusions may be less surely established than if the numbers of animals used were larger.

It appeared to the Committee, therefore, that essential research on farm animals using radioisotopes or radiation is being discouraged by the high costs involved because animals must be destroyed at the termination of experiments. It recommends that a special committee be appointed to study this problem and to develop procedures and standards that, if followed and enforced, would adequately protect the consumer, but permit the marketing of animals that in experimentation have been brought into contact with radioactive substances or exposed to radiation.

The welfare of the livestock population is enhanced if troublesome insect pests can be controlled or eradicated. As mentioned earlier, insecticide studies have been greatly aided by the availability of radioisotopes as tracers, but in addition there may be certain opportunities for control of insect pests by taking advantage of radiation-vulnerable stages in their life cycles. Eradication of the screw worn fly from the southeastern United States is to be attempted, based on the virtual elimination of this fly from the island of Curacao by the release of males rendered sterile by radiation exposure. This technique may not be generally applicable to all insects pests.

V. RADIOISOTOPES IN AGRICULTURAL PRODUCTS AND FOODS

The Committee discussed in detail some of the difficult problems that may arise because of the presence of a radioisotope burden in agricultural products and foods higher than that "naturally occurring." The applicable legislation in this area is clouded with uncertainties, because the very possibility was not envisaged by those who enacted the laws and defined the responsibilities of the agencies that protect the public food supply. There are no permissible limits for radioisotopes in foods; any burden above the "natural" is regarded as undesirable. The current interpretation of the law places isotopes in the same category as poisonous additives. It is difficult, however, to be wholly consistent in this, inasmuch as the normal radioisotope burden varies considerably in different agricultural products, and in the same product from different locations. Moreover, the testing of atomic and nuclear weapons is placing in soil, water, and air, the world over, radioisotopes not formerly present, though at extremely low levels. Th "natural content" of foods now consumed by animals and man is not the same as in the pre-atomic age. Though extremely small, the increment is measurable, and inescapable.

It is to be anticipated that there will be in the years ahead a slowly rising background of radiation manifest in agricultural and food products by the presence of the isotopes of elements not previously found therein or of "unnatural" levels of radioactivity. Atomic warfare might greatly increase the rate of this development. As pointed out earlier in this report, radiostrontium is particularly the element which would cause concern in the latter event. Forage directly contaminated with fall-out, if consumed by farm animals soon after deposition might cause radiation injury from the presence of insoluble radioactive products. Strontium is metabolically similar to calcium and moves into bone and other calcium-accumulating tissues or fluids. Much is known of the relative behaviors of calcium and strontium but there appears to be no way of wholly preventing strontium retention. There is some evidence that poultry may "decontaminate" or "detoxify" themselves by reason of a continued dilution through transfer to eggshell. In meat animals certain tissues might be consumable if boned out, but such an expedient would be beyond the ordinary scope of meat inspection. Dairy products would contain radiostrontium for some considerable time after ingestion of strontium-containing forage. Moreover, all avail-

able feeds, in heavily contaminated aras, might contain significant levels of radiostrontium, perhaps for years.

At present it is not possible to say at what level a food, otherwise wholesome, becomes unwholesome or deleterious by reason of the presence of an unnatural burden of radioactivity. There is a great deficiency of requisite data on the long-term biological effects that may follow the ingestion of such foods by animals and man. Situations in which such information might be of great public importance are not inconceivable and possibly inevitable.

The Committee therefore urgently recommends that appropriate experimentation be immediately activated to provide specific information about possible total or cumulative biological effects that might follow the ingestion of such foods. It further urges that the planning of such experiments be broadly based, and that the development of the experimental designs and details of their subsequent execution be most carefully considered in order that the emerging data will be acceptable as a basis for the crucial decisions that ultimately will have to be taken, and directly of value to the regulatory agencies charged with the protection of the public interest.

VI. ENVIRONMENTAL CHANGES AND ECOLOGICAL STUDIES

In the decades ahead there is a strong possibility that the general background of radioactivity in agricultural areas will rise. Contributing to this would be fall-out, if weapons-testing continues, and wastes from nuclear power plants or isotope processing plants. As indicated in the report of another Committee every effort will have to be made to contain radioactive wastes. Atomic warfare, or accidents involving nuclear power sources could of course greatly augment the background and pose difficult problems of land-use for agricultural purposes. Limited ecological studies are in progress in the vicinity of certain A. E. C. installations, but it may be wise to consider this general problem somewhat more widely and to attempt to establish, through careful sampling, the present background in representative agricultural areas, and in their chief crop and livestock products.

Research activities might appropriately be carried out on areas near weapons test sites where substantially greater changes in background would be anticipated. The distribution in the environment, in the soil at various depths, in the vegetation, in the wildlife, in the streams, etc. would all be pertinent. The rate of accumulation in soil as affected by land use ought to be studied. Forested land, rangeland, rotation grassland, and plowland, irrigated and non-irrigated, may each present a different situation. It is possible that certain of the State Agricultural Experiment Stations might be in a position to undertake limited surveys of this type on areas likely to be under their control for some considerable time in the future.

The Committee recognized clearly that sustained monitoring and ecological research activities of this type are expensive and are not apt to be professionally rewarding to the individuals participating therein, because trends and conclusions would emerge only slowly. However, to be able to recognize changes in the levels of radioactivity in the environment and in products removed therefrom, and to follow movements in the system, may well be in the public interest from a long-range viewpoint.

VII. EFFECTS OF RADIATION ON PLANT OR ANIMAL PRODUCTS (FOOD PROCESSING)

A recent development in food technology, potentially of considerable and possibly dramatic significance, is the recognition of the fact that radiation can be used as a means of preserving certain foodstuffs or of lengthening shelf life, either unrefrigerated or refrigerated. The radiation source may be gamma rays or high energy electron beams. No radioactivity is induced in the irradiated material. Feeding experiments to date indicate that foods so irradiated will prove to be suitable and safe for consumption by man. Parasites in meat and meat products can be killed by exposure to penetrating radiation; and undesirable post-harvest changes in plant products, such as the sprouting of potatoes, can be delayed.

The prime objective in radiation processing is to destroy microorganisms, or so greatly to reduce the microbial population (radiation pasteurization) that spoilage is long delayed. To accomplish this, very heavy radiation exposures are necessary because microorganisms are much less sensitive to radiation than are animals and higher plants. The food processor is particularly attracted by

the fact that the radiation exposure can and should be carried out after packaging.

The acceptability of some radiation sterilized foods is open to doubt because of the development of off-flavors, and changes in odor or in the texture of the tissues. Much of the developmental work in this field however has been of a rather empirical nature, and it is possible that through research means may be found to repress some of these undesirable changes.

Although the feasibility of radiation sterilization has been amply demonstrated, the economics of the various processes have not yet been established. This development has largely been financed by the military with the Army Quartermaster Corps as the primary agency involved, but there has been a broad basis of cooperation in industry and elsewhere, with some technical guidance and evaluation by Advisory Committees of the National Academy of Sciences. Having in mind the magnitude and coherence of the current broad programs in this area the Committee was of the opinion that the potentialities of this use of radiation are being thoroughly explored, and that the interests of the food consumer will be adequately protected. At a later date the Committee expects to review particularly the evidence of wholesomeness and acceptability of irradiated foods.

VIII. COMMITTEE MEMBERSHIP

The names and institutional affiliations of members subscribing to this report are listed below. In their deliberations they were aided by Douglas M. Whittaker of the Rockefeller Institute and Charles I. Campbell of the staff of the National Academy of Sciences-National Research Council. As Consultants the Committee is indebted to A. J. Lehmann, Food and Drug Administration, Robert Somers, Meat Inspection Service, U. S. D. A., J. Wolff, Atomic Energy Commission.

A. G. NORMAN, *Chairman*
C. L. COMAR
GEORGE W. IRVING, Jr.
JAMES H. JENSEN
J. K. LOOSLI
ROY L. LOVVORN
RALPH B. MARCH
GEORGE L. MCNEW
ROY OVERSTREET
KENNETH B. RAPER
H. A. RODENHISER
W. RALPH SINGLETON
RALPH G. H. SIU
G. FRED SOMERS
GEORGE F. STEWART

SUMMARY REPORT OF THE COMMITTEE ON DISPOSAL AND DISPERSAL OF RADIOACTIVE WASTES

INTRODUCTION

Experience in handling the waste disposal problems to date is mostly limited to conditions as they exist in the areas of the national atomic energy establishments. The determination of hazards from the disposal of wastes in these areas, most of which are in remote and somewhat isolated regions, involving relatively short periods of time, has to date revealed no deleterious effect on the public or its environment.

This does not provide, however, a completely adequate basis for projecting the magnitude of the hazard into the vastly expanded realm of industrial atomic power production. Not only does the problem itself take on new significance with the projected amount of wastes, but environmental factors which may lie dormant under conditions existing in the remote areas take on full blown importance when viewed under the more stringent requirement for highly populated areas.

Many such problems immediately come to the surface as a result of consideration of the long-term legal and insurance aspects. These problems reflect first of all a need for deeper understanding of the basic issues and for more refined measurements, and not merely for greater but still unknown factors of safety. Long-term responsibilities, moral, legal, and financial, stemming from the ownership of atomic wastes simply come into sharp focus when it is emphasized that the radioactive life of the wastes would probably exceed by several centuries the official life of the organization itself. Legal and insurance requirements, therefore, will undoubtedly have a great deal to do with the shaping of rigid administrative policies with respect to these long range aspects of the atomic waste disposal problem. It may be difficult to maintain an adequate balance between objectives which primarily must emphasize the legal requirements and those

which in the broad biological sense must establish the foundations for a truly preventive approach to this problem.

PRESENT STATUS OF PROBLEM

The following listing summarizes the conclusions regarding the status of waste dispersal and disposal operations:

1. The safe handling and ultimate disposal of radioactive wastes is an important technical, economic and administrative aspect of the nuclear energy industry. Waste operations must be thoroughly integrated with all other phases of nuclear energy operation.
2. From a technological standpoint the highly radioactive wastes resulting from the processing of reactor fuels constitute the bulk of the problem. To date essentially none of those wastes has been disposed of, i. e., returned to the environment. Tank storage is presently utilized as an interim answer to this problem.
3. Wastes resulting from normal reactor operations are an important consideration, but technically represent a problem for which solutions are generally available.
4. Research and development have indicated possible feasible systems for ultimate controlled disposal of highly radioactive wastes, but considerably more work is required to bring these systems to the point of economic operating reality.
5. Major technical and economic considerations underlying the waste problem are:
   a. Characteristics of nuclear fuels and chemical (or other) processing associated with them.
   b. Separation of specific isotopes from the wastes and use of these materials to economic advantages.
   c. The proper selection of the site for nuclear facilities—especially reactor and fuel processing plants.
   d. The detailed quantitative evaluation of the environment in order to assess its capacity to receive radioactive materials without creating deleterious effects on the environment.
   e. Systems for the physical handling and transportation of highly radioactive materials.
6. Major policy and administrative considerations relevant to the regulation of the waste problem are:
   a. The establishment, perhaps through private enterprise, of suitable waste disposal services.
   b. The regulation and control of waste disposal practices through existing and traditional state, interstate and local channels where feasible.
   c. Continuation and strengthening of established practices in relations with the public and its agencies.

RELATION TO NUCLEAR INDUSTRY GROWTH

Based on the best estimates available (which vary over rather wide ranges) and, to a substantial extent on technical judgment, the indications are that the principal source of fission products from nuclear reactors in the next decade will arise from the generation of electricity at nuclear powered central stations. On the basis of present developments, the second most important source probably will be reactors for naval service. Compared with these, other sources are comparatively small and amount to substantially less than the uncertainty in the estimates of the principal uses.

By 1965 the average rate of reactor heat release is estimated to be about 11,000,000 kilowatts. Naval service will account probably for 20 percent of this output in 1965. This rate of heat release will result in the production of somewhat over 10 kilograms of fission products per day in 1965.

In addition, the presence of radioactive wastes in quantity will have a profound effect on certain non-nuclear industries which may be damaged by air or water contaminated with radioactive wastes. Numerous wet-processing industries are likely to be detrimentally affected by radioactive wastes even in trace concentrations. Among this vulnerable group are those requiring water of the highest purity, such as for the manufacture of photographic film. Other industries which should be alerted to the problem are pharmaceutical manufacturers and food processing companies. It is not possible, at this time, to enumerate with assurance the industrial processes which can be completely

eliminated as subjects of this potential hazard, without the assembly of extensive research and statistical data applicable to specific operations.

RELATION TO FUEL PROCESSING AND TYPES OF REACTORS

Neither the type of fuel nor the length of irradiation time greatly influence the accumulated total radioactivity of fission products. Atfer approximately three years decay the residual radioactivity is essentially the same for various irradiation times, assuming constant heat generation during the irradiation period.

Essentially all of the radioactive material from fuel separations processes must be kept from the environs to maintain human exposures within maximum permissible limits. An important problem which possibly limits storage volume is the rate of heat removal from the containers. After solvent extraction wastes are concentrated by supplied heat to about 2000 gallons per ton of irradiated uranium, the heat of radioactive decay will continue the concentration to 100–500 gallons per ton. Practical heat removal mechanisms may require that more concentrated waste produced by other separations processes be diluted to the same volume range. More concentrated fluid wastes also need stronger, less economical containers. The volumes of stored waste accumulated by 1980 are estimated at 20 x 10^7 gallons, by 1990 at 60 x 10^7 gallons and by 2000 at 240 x 10^7 gallons.

The future possibility of high burn-up of reactor fuels might ultimately result in a situation where processing may be unwarranted. This would not change accumulation of fission products, but would have a profound effect on waste storage and disposal considerations. Similarly, the development of non-aqueous chemical processing methods would be important in modifying the waste management problem.

ISOTOPES PROBLEMS

The technical and administrative problems associated with the transport, use and disposal of radioactive materials in medicine, biology, and industry will undoubtedly grow in complexity and quantity as the demand for the use of these radioactive materials increases. The expanding demand is already apparent in the rapidly increasing number of individual isotope users as evidenced by the expansion of the isotope distribution program. The program for the distribution of reactor-produced radioisotopes is nearing one decade, having been initiated on August 2, 1946. During this period more than 100,000 shipments of radioisotopes have been made from AEC facilities to some 3,200 institutions throughout the United States. These materials are being applied in science, agriculture, medicine and industry. The Oak Ridge National Laboratory, the principal radioisotope production facility in the United States, has shipped approximately 130,000 curies to date.

All indices of radioisotope utilization reveal continued rapid growth. A look at the last three years of the program shows a growth in the number of using institutions from 1,400 to 3,200. This is an increase of approximately 125%. There has been a 100% increase in annual numbers of shipments made since January 1, 1953. The principal growth during the period has been in the industrial use of radioisotopes.

However, of even greater significance in connection with environmental and hazard control problems is the ever increasing desire for larger and larger individual sources of radioactivity. Requirements for intense radiation sources are obviously at their earliest stages. Such uses as food and pharmaceutical sterilization, promotion of chemical reaction, and other yet unknown applications will undoubtedly result in a much more extensive use of mobile and more widespread sources of intense radiation.

Increased use, especially of highly active materials and the increase in the production of by-product materials at widely scattered geographical locations will result in ever increasing new technical and especially administrative problems in both the transport of the material and the disposal of the wastes, in order to protect the environment against normal and potential emergency hazards.

Compliance with existing transportation regulations present few significant problems in the shipment of by-product material even through certain specific limitations exist. However, consideration should be given to a complete critical review of existing ICC, Civil Air, Coast Guard and Postal regulations to bring them in line with current requirements and radiation safety knowledge.

The radiological health and safety record in the nation-wide use of radioisotopes is excellent. Incidents which have come to the Atomic Energy Commission attention involving significant overexposure of personnel are exceedingly small; fewer than 10. In large measure this may be attributed to active educational efforts in radiological protection through a field advisory service to isotope users and through effective and practical licensing practices.

At present activity levels of use of radioisotopes and with the wide dispersal of users substantial environment health problems do not exist due to waste disposal or other practices resulting in the introduction of radioisotopes into the environment.

ITEMS REQUIRING FURTHER STUDY

The following listing summarizes conclusions in this area:

1. Geophysical and geochemical aspects of ultimate disposal of highly radioactive wastes.
2. Site selection for various nuclear facilities, particularly chemical processing plants and their location with respect to suitable waste disposal areas.
3. Transportation of highly radioactive materials.
4. Relationship of introduction and development of nuclear facilities to basic public health, social and economic situations extant or resulting from such development.

PROBLEMS OF ACCIDENTAL HAZARDS

The following conclusions in respect to the consequences of accidents involving radioactive materials appear warranted:

1. The problems of waste disposal could be international in character and must be solved technically so that the total environment is maintained at a low level of radioactivity in order that accidents that are bound to occur will not be disastrous.
2. The type of accident that could result in a catastrophic spread of radioactive materials is the complete vaporization of the core of a reactor and its release to the surroundings. The probability of a catastrophic accident with a properly designed nuclear reactor is extremely small.
3. Reactor waste processing plants or storage facilities offer a greater hazard on a long-term basis than any single reactor.
4. Accidents in handling, transport, and chemical separation of radioactive materials, while locally severe, should not affect a wide public area and, in all cases, the contaminated areas can be cleaned up.
5. The probability of accidents in handling radioactive isotopes and low-level radioactive materials is similar to that in handling other types of lethal substances.
6. Use of nuclear reactors to drive ships appears feasible from a consideration of the consequences of possible accidents provided uranium-233 and plutonium are kept to a minimum. The technology of the use of nuclear reactors to drive locomotives and commercial airplanes has not developed to the point where the committee can form a judgment as to the consequences of possible accidents.
7. Development of improved methods to limit the volumes of wastes produced in nuclear power reactors is justified from the viewpoint of the hazards due to possible accidents.
8. Continuous and vigorous appraisal of reactor and fuel processing plants design and operation and waste storage will be required in all nations using atomic energy in order to keep the radioactivity level of the world environment at tolerable levels.
9. Improved safety devices for control of transients in nuclear reactors should continue to be vigorously developed.
10. Further tests are required of reactors to evaluate their ability safety to withstand power excursions which may occur as a result of unusual operating circumstances.
11. Until such time as advances in the technology of reactors lessen potentially hazards substantially, sealed buildings properly designed, constructed, and tested should be required for all nuclear reactors to be built in or near populated areas.
12. All operations involving radioactive materials in sufficient amounts to create possible health hazards should be supervised by trained and responsible people.

FALL-OUT CONSIDERATIONS

It is apparent that as of the present time the dispersal of radioactive material resulting from weapons testing has not been an environmental contaminant of substantial public health significance. However, because of various unknown factors regarding distribution and ultimate fate of this material, plus the potentials of possible wider spread and more frequent weapons testing it is also apparent that the subject in all of its aspects merits meticulous and continuing attention. The problem of fall-out is one of international significance and should be studied and evaluated on that basis, perhaps looking forward to international cooperation in control.

ABEL WOLMAN, *Chairman*	H. M. PARKER
F. L. CULLER	W. A. PATRICK
ARTHUR E. GORMAN	SHEPPARD T. POWELL
L. P. HATCH	LESLIE SILVERMAN
H. H. HESS	PHILIP SPORN
CLARENCE W. KLASSEN	CONRAD P. STRAUB
SIDNEY KRASIK	CHARLES V. THEIS
JOSEPH A. LIEBERMAN	FOREST WESTERN

THE NATIONAL ACADEMY OF SCIENCES—NATIONAL RESEARCH COUNCIL

The National Academy of Sciences—National Research Council is a private non-profit organization of scientists, dedicated to the furtherance of science and to its use for the general welfare.

The Academy itself was established in 1863 under a Congressional charter signed by President Lincoln. Empowered to provide for all activities appropriate to academies of science, it was also required by its charter to act as an adviser to the Federal Government in scientific matters. This provision accounts for the close ties that have always existed between the Academy and the Government, although the Academy is not a governmental agency.

The National Research Council was established by the Academy in 1916, at the request of President Wilson, to enable scientists generally to associate their efforts with those of the limited membership of the Academy in service to the nation, to society, and to science at home and abroad. Members of the National Research Council receive their appointments from the President of the Academy. They include representatives nominated by the major scientific and technical societies, representatives of the Federal Government, and a number of members-at-large. More than 3000 of the foremost scientists of the country cooperate in the work of the Academy-Research Council through service on its many boards and committees in the various fields of the natural sciences, including physics, astronomy, mathematics, chemistry, geology, engineering, biology, agriculture, the medical sciences, psychology, and anthropology.

Receiving funds from both public and private sources by contribution, grant, or contract, the Academy and its Research Council thus work to stimulate research and its applications, to survey the broad possibilities of science, to promote effective utilization of the scientific and technical resources of the country, to serve the Government, and to further the general interests of science.

EXCERPTS FROM PATHOLOGIC EFFECTS OF ATOMIC RADIATION, STUDY BY THE NATIONAL ACADEMY OF SCIENCES, NATIONAL RESEARCH COUNCIL

APPENDIX I

REPORT OF THE SUBCOMMITTEE ON ACUTE AND LONG TERM HEMATOLOGICAL EFFECTS OF ATOMIC RADIATION

Eugene P. Cronkite, Brookhaven National Laboratory, Chairman
Carl V. Moore, Washington University School of Medicine
William N. Valentine, University of California Medical Center
Victor P. Bond, Brookhaven National Laboratory
William Moloney, Boston City Hospital
George V. LeRoy, Billings Hospital, University of Chicago
George Brecher, National Institutes of Health
James S. Nickson, Memorial Hospital, New York

Consultants:
James Hartgering, Lt. Col. (MC) USA
Karl Tessmer, Lt. Col. (MC) USA, Walter Reed Army Medical Research Institute

ACUTE AND LONG TERM HEMATOLOGICAL EFFECTS

On January 7 and 8, 1956, the Subcommittee met at the National Academy of Sciences in Washington, D. C. with all members present. A preliminary report was written by Drs. Bond and Cronkite and distributed to Subcommittee members. The comments of the members have been incorporated into this report.

INTRODUCTORY COMMENTS BY THE CHAIRMAN

Mankind has always lived in an environment suffused with radioactivity from natural and unavoidable sources such as radioactive minerals and cosmic rays. Natural radioactivity varies greatly in degree throughout the world. Intensities tend to be much lower at sea level and on most small islands, with the exception of Baltic islands, throughout the world. At high altitudes cosmic ray activity increases significantly. Similarly natural radioactivity from minerals increases significantly in some mines, and in the water supplies of some areas. For example, water in the Joliet area in Illinois contains relatively large quantities of radium and its daughter products. Since the discovery of x-rays by Konrad Roentgen and natural radioisotopes by Becquerel and the Curies, there has been a steady increase in the amounts of radiation to which segments of mankind are being exposed. With the development of nuclear weapons and the spread of atomic energy by industrial activities, the levels of world wide radiation will unquestionably continue to increase.

At the present time there is confidence that the increment to the naturally existing radioactivity is but a small fraction of that believed to exist prior to the testing of atomic weapons and the presently developed atomic energy industry. However, when one specifies the diverse sources of radiation to which large numbers of mankind are being exposed, it is quite evident that serious concern must be felt by physicians and scientists for the possible influence of such radiation upon individuals, selected groups, and whole populations. In the course of the deliberation of this panel attention was called to the existence of the following types of exposure to radiation to which human beings were exposed, voluntarily and involuntarily.

1. Natural sources
2. World wide low-level fallout
3. Roentgenographic surveys of large segments of the population for tuberculosis and cancer
4. Dental x-rays
5. Industrial fluoroscopy and radiography
6. Fluoroscopy of infants
7. Fluoroscopy for shoe fitting
8. Diagnostic x-rays
9. Medical and scientific use of tracers in human beings
10. Therapeutic use of radioisotopes and x-ray
11. Tracer radioisotopes in agriculture and industry
12. Research and power reactors
13. Ionizing radiations for food sterilization
14. Experimental accelerators

During the early years when the diagnostic and therapuetic uses of x-ray were being developed, heavy and repeated exposure to physicians, physicists, nurses, and technicians resulted in serious injuries. Historically, the occurrence of leukemia and aplastic anemia as late sequelae of exposure to x-ray and radioactive substances was well documented in the medical literature prior to 1937. Presumably Madame Curie died from an aplastic anemia. In recent years, while great care has been taken to avoid heavy radiation exposure, there is little knowledge on the hazards of repeated smaller does, especially in regard to late effects on the blood forming organs.

The record of the atomic energy development, which involved the handling of large amounts of dangerously radioactive material is an example of the effectiveness of the controlled environment.

Dose dependence and correlation of other effects with hematologic effects

In order to set the acute and chronic hermopoietic effects of radiation into the proper perspective with regard to overall radiation effects, the whole body radiation syndromes as a function of dose of radiation are summarized:

After very large doses of radiation delivered in a short time, a typical clinical syndrome is produced in animals. On the basis of observed symptomatology, it has been useful to name this symptom complex the central nerous syndrome (CNS). In animals, doses in excess of many thousands of r, are necessary to produce this complex. There are species variations. The "threshold" for this syndrome in man is not known and this syndrome has not been observed in man. Symptoms referable to the nervous system and GI tract appear promptly. Death may occur "under the beam" or within a few hours. In laboratory animals this syndrome invariably results in death either promptly, or later as a result of the next clinical syndrome which results in death at a later time. If the nervous symptom complex subsides, or if the dose has been smaller (in the region of 900–5,000 r for laboratory animals; dose for man not known), a symptom complex termed the gastrointestinal syndrome (GIS) appears. Nausea and vomiting appear shortly after exposure. Diarrhea and tenesmus become severe. The GI symptoms may be intractable or may subside for a variable period. Fluid and electrolyte loss from the GI tract progressively produce dehydration and eventual vascular collapse, and death that may occur during the 1st and 2nd weeks. This picture was observed in the Japanese casualties and has been well studied in laboratory animals. Death from this syndrome has been prevented experimentally in some dogs by adequate fluid and electrolyte replacement; in addition spontaneous recovery may occur after the lower doses in this range. However, the survivors then experienced another symptom complex that may result in death. It is this third symptom complex characterized by signs and symptoms referable to bone marrow depression which characterizes the lethal dose range.[1] By custom, the mortality from this syndrome has been tabulated as of 30 days after explosure. There are reasons, to be discussed later, why a 30 day tabulation may be too short for human beings.

A fourth phase of deaths was observed during the 2nd and 3rd months after exposure in the Japanese casualties in which in some instances the causes of death were not clear; however, pancytopenic sequelae were still present. Hemopoietic recovery was in progress but defects in proliferation and maturation were observed in the pathologic sections of marrow. Following the third month, deaths are infrequent and it becomes increasingly difficult to ascribe deaths to the effects of radiation since phenomena observed are those which may result in death in any non-irradiated population. In some instances cause of death was uncertain although pancsytopenia was prominent.

There is evidence that large single doses, or repeated small doses of radiation can produce diverse neoplasia, genetic defects, and shortened life span in select controlled *animal* populations. However, attempts to ascribe a specific role to irradiation in neoplasia in *human* populations becomes an exceptionally complex biometric study because of the increasing contamination of the atmosphere by industry with potential carcinogens, and the introduction and widespread use of an array of clinically useful drugs, whose long term effects in man are imperfectly understood, but which in some cases have produced severe blood dyscrasias. Accordingly, in all of the discussions on the long term effects of single doses of radiation on *man*, and the effect of repeated or low level exposure, one must be especially cognizant of the fact that the "effects" are *deduced* by statistical *correlations*, and cannot be proved by controlled experimentation, nor can other causative factors be eliminated. In this era of awakened public interest to the hazards of radiation, it is especially important that preoccupation with the hazards of ionizing radiation does not becloud the searching mind of the scientist or the responsible citizen to the presence of other hazards of equal importance. This is not an attempt to minimize the hazards of ionizing radiation with respect to the development of blood dyscrasias and other late effects. It is most

[1] Sublethal refers to the lower doses of radiation that will produce no deaths within a given period of time, usually taken as 30 days in animals. The lethal range extends from the threshold dose at which only rare deaths occur in this time interval, to the level at which virtually all exposed will die (the LD 1 to LD 99 range). Doses above the LD 99 level are termed supralethal. In all ranges, however, ultimate longevity is reduced to some degree.

important to bear in mind that the incidence of bone marrow failure[2] and leukemia has increased significantly in the United States in groups in whom there is no known overexposure to ionizing radiations. Today no informed physician believes that exposure to ionizing radiation has either a beneficial or stimulating effect on the blood.

In the course of the deliberation of this Subcommittee, attention was focused upon the known effects of nuclear explosions, the immediate and long term effects of single exposures from all causes, and the long term effects of intermittent and continuous explosure to radiation of diverse types. In the latter category, the Subcommittee felt that a reasoned judgment could not be made because of the paucity of realistic quantitative data on the degree of exposure.

ACUTE HEMATOLOGICAL RESPONSE TO SINGLE DOSES OF PENETRATING RADIATION

Although the available sources of hematological data on human beings exposed to total body external radiation have serious limitations, they were considered to be reasonably consistent among themselves to allow characterization of the time course of change in peripheral elements following exposure. The sources of data included the reports of the Japanese exposed to immediate radiation from atomic weapons, the account of the human beings accidentally exposed to fallout radiations at the Pacific Proving Grounds in March, 1954, the reports of human beings exposed to reactor accidents in the laboratory, and data on patients with incurable neoplastic disease exposed to therapeutic total body irradiation. The pattern of response of the peripheral blood elements changes with increasing radiation dose. In the following description, changes are divided into those that occur in the sublethal range, and those that occur in the lethal range (doses that result in some mortality within 60 days of exposure). This division is arbitrary, since the patterns of change merge imperceptibly, and each category covers a range of doses and thus degree of effect. When the dose is increased from sublethal levels to lethal levels, the lag period between exposure and depression is progressively shortened.

Response after sublethal doses

The *neutrophil* count shows an initial use in the first 12 to 24 hours followed by a sharp drop, to or below, the pre-exposure level. The count then fluctuates around or slightly below the pre-exposure level until the 3d or 4th week, following which definite depression is observed. The time of maximum depression occurs during the 5th or 6th week or even later, and is followed by a gradual return to pre-exposure levels. Complete recovery may require several months or more.

The drop in *lymphocytes* is early and profound. Little or no evidence of recovery in the high sublethal range may be apparent several months after exposure, and return to former levels may not occur for months or years. The total white count parallels closely the change in neutrophil count.

The *platelet* count shows little or no change over the first three weeks following exposure. At approximately the end of the 3rd week the platelet count falls. The time of maximum depression is remarkably constant at sublethal dose levels, and occurs on the 28th to the 32nd day of post-exposure.

No trend in eosinophile, basophile, or monocyte counts can be definitely ascertained. This may result in part from the larger errors inherent in counts of these cells. In the absence of hemorrhage, the hematocrit may show slight depression. This effect is probably due to a combination of inhibition of erythropoiesis and shortened life span of the red cell.

Response after doses in the lethal range

The *neutrophil* count may rise during the first two days following exposure. The count then falls steadily to reach values below 1,000/mm^3 by the 5th to the 10th post-exposure day, depending on dose. In survivors, recovery begins during the 5th week, but many not be complete for several months.

The *lymphocyte* count drops to vanishing levels within 12 to 24 hours of exposure; recovery is not apparent for several weeks; and it may not be complete for several months, or for a year. The total white count parallels the neutrophil count.

---

[2] Synonymous with aplastic anemia, refactory anemia, hypoplastic anemia. Aplastic anemia has been observed to terminate in leukemia. The occurrence of aplastic anemia after use of diverse drugs is common clinical knowledge.

The *platelet* count in the lethal range, in marked contrast to that at lower doses, may drop precipitously, starting approximately on the 4th day, and platelets may virtually disappear from the peripheral blood by the 10th day.

Changes in the eosinophiles, basophiles, and monocytes counts cannot be characterized definitely at this time. The hematocrit [3] is not appreciably affected until hemorrhage occurs. severe gross external or occult internal bleeding may occur as early as the 9th day, depending primarily on the time at which the platelet count reaches dangerously low levels. This may occur from the second to the fifth week, with peak incidence in the 4th week in the low and mid-lethal dose ranges. The degree of response as a function of dose varies for the several blood elements. The platelet and lymphocyte counts are affected by very small doses of radiation, and are reduced to minimal levels before the lethal dose range is reached. The neutrophil count, however, does not reach minimal values until the lethal range is reached.

Comparison of man and other mammals

The time course of changes in the leucocyte and platelet counts in human beings is definitely different from that observed in lower animals. In man, severe depression of these elements occurs later, and recovery is more delayed. Similarly, the time of deaths in man resulting principally from hematological depression differs from that of laboratory animals. In most laboratory species, essentially all animals alive on the 30th post-exposure day will remain alive for several months, although the life span is shortened. In man, however, the peak incidence of death from marrow depression occurs during the 4th and 5th post-exposure week (Hiroshima and Nagasaki data). Thus an LD 50, 30 day consideration is inadequate to characterize the acute lethal dose response of man, and an LD 50, 60 days would be preferable.[4] The extensive serial blood counts obtained in human beings exposed to fallout gamma radiations were relied on heavily in characterizing the hematological responses of human beings exposed to external radiation. Admittedly the dose rate with fallout was much lower than with prompt radiation and may have reduced the effectiveness somewhat. These individuals received, in addition to gamma radiations, beta radiations of the skin, and probably a minimal degree of internal contamination. It was the consensus of the Subcommittee that neither the beta lesions nor the low level of internal contamination significantly contributed to the pattern of change observed. This view was supported by the general agreement of these data with other less extensive data on human beings who did not receive additional skin lesions or internal contamination; and the lack of correlation between the severity of hematological change and the extent of beta lesions in those exposed to fallout radiation. The reservation was held, however, that data are inadequate to establish this view with certainty, and that synergistic effects cannot be ruled out.

Mortality and morbidity from whole-body radiation

The pan-hemopoietic depression contributes in large measure to morbidity and mortality following total body irradiation. In the sub-lethal and low lethal ranges, the response observed is consistent with other clinical pancytopenic states. Neutrophil depression increases the susceptibility to infection and platelet depression contributes to the bleeding tendency. Correction or treatment of these defects during the first few weeks may permit survival in some individuals who might otherwise have succumbed. The concept of total body x-radiation as primarily a pancytopenic state, while useful, is probably an oversimplification, particularly in low and high lethal ranges.

Susceptibility to infection is well established and the pathogenesis may well involve interference with specific immune mechanisms, phagocytosis, and migration of leukocytes in addition to simple neutrophil depression.

Susceptibility to bleeding is well correlated with platelet depression. However, additional factors may be involved such as lipid antithromboplastins (Tocantins) or disturbances in the β lipoprotein transport mechanism (Nickson and Bane).

---

[3] Admittedly the hematocrit can be misleading since it represents both changes in plasma volume and red cell mass. However, in general decreases in hematocrit represent a diminution in red cell mass for one reason or another (loss, hemolysis, or no new production).

[4] The reservation must be made here that the exposed Japanese population were heterogeneous with respect to age, sex, physical condition and degree of added trauma from burns or blast. The extent to which these factors affected survival time has not been determined. In studies on laboratory animals the converse is true—homogeneous populations are studied.

In some instances, the latter changes are similar to changes induced by heparin administration to rabbits. The relation of these alterations to the bleeding tendency has not been established. It was the consensus that frank heparinemia is not a contributing cause of bleeding.

After *higher doses*, death ensues even if hemorrhage and infection are corrected. Germ free rats die at dose levels moderately higher than the lethal dose for rats in the natural state. These animals die later with severe hemorrhage and anemia. At dose levels in excess of the LD 100, 60 day level, individuals die within the first week presumably from fluid imbalance and vascular collapse correlated with marked damage to the intestinal epithelium. It is clear that at all dose levels, poorly known and little understood biochemical changes[5] occur which may contribute to mortality in the exposed individual. Our knowledge is inadequate to determine at the present time to what extent such biochemical changes may prove lethal in themselves even when infection and hemorrhage can be treated adequately.

Lethal dose for man

No data are available to allow adequate characterization of the LD 50 value for man. The degree of hematological depression observed in patients receiving total body x-radiation indicates that the current estimate of 450 r is a reasonable estimate for x-radiation as employed in the clinic. A recent re-evaluation of the data from Hiroshima and Nagasaki indicated a value higher than this for immediate gamma radiation from the bomb. Geometrical and depth-dose considerations can be interpreted to indicate that the LD 50 for man exposed to immediate gamma radiation and fallout gamma radiation from the atomic bomb may be lower than this figure. A large degree of uncertainty exists in both approaches, and more biological and physical data are required to settle the issue. The situation is complex, and it became evident that it is not possible to extrapolate with confidence from one condition of radiation exposure to another, or from animal data to man.

Threshold dose for detectible effects

There appears to be no threshold[6] dose for changes in the peripheral platelet count and possibily for other elements of the blood. Changes at very low dose levels, however, can be detected only in a relatively large population. Nothing is known about subtle changes in the blood forming organs at dose levels so small that changes in the blood picture cannot be detected.

Diagnosis of radiation exposure and its severity

The diagnosis of exposure to radiation and its severity is made on the basis of the history and physical and laboratory examinations, as with any disease. Available estimates of air roentgen dose received obtained by physical means should be considered in evaluating the degree of exposure, but should never in themselves be taken as an index for disposition or treatment since *tissue dose* and *distribution* of absorbed energy ultimately determines effect not dose in air. Any degree of radiation exposure should be avoided if possible. If exposure is necessary under emergency conditions, severe hematological depression may be expected at doses of 100 r or more measured in air, from immediate radiation from the bomb or from the gamma radiation from fallout material. With human exposure a wide spectrum of ages and of state of health is likely to be involved. Thus it is not possible to predict acccurately the severity of response that might be expected for a population exposed at various dose levels. There is evidence from the human beings exposed to fallout radiation that children may be more severely affected than are young adults. Whether this is due to inherently greater sensitivity or to an increased depth dose due to smaller size is not known. From animal data, it has been postulated that elderly individuals may be more seriously affected than young adults.

Therapy of radiation injury

Recommendations for therapy are given for 1) conditions where exposed individuals can be carefully and individually handled because they are limited in number and adequate facilities exist for taking care of them, and 2) ex-

---

[5] Apparently decreased respiratory quotient (RQ) in animals, increased excretion of amino acids in irradiated human beings, etc.

[6] The threshold concept may be incorrect since inability to detect effects at lower doses may only be a manifestation of inadequate criteria for effect. For practical purposes a threshold might be classified as that dose where statistically sifinificant differences are detected. For many effects this may necessitate extremely large samples.

posures at the catastrophic level where adequate medical observation and care are impossible. In the first category, of cardinal importance in therapy is careful observation, good nursing care, and treatment on an individual basis of any condition that may arise. Antibiotics in general should not be given prophylactically, and should be administered only if infectious processes develop that would be treated with antibiotics in the absence of radiation. Prophylactic use of antibiotics may be considered if the neutrophil count drops below 1,000/mm^3. Prophylactic use of antibiotics may be considered particularly where severe wounds or other complications may be present. If antibiotics are used, they should be given in large doses and the broad-spectrum drugs should be employed. Fluids should be given as indicated clinically. Blood should not be given prophylactically, but only as indicated from clinical and laboratory findings. Fresh whole blood by direct silicone multiple syringes without anticoagulant or collected in plastic bags or platelet transfusions may be of some value in controlling purpura and other hemorrhagic manifestations. The use of drugs without clear indication is discouraged because of their unknown and possibly harmful effects on the irradiated individual whose metabolism is deranged. Parenteral administration of drugs should be held to a minimum because of the added trauma in an individual susceptible to purpura and infection. At present there are no specific prophylactic or therapeutic agents [7] that should be stock-piled for us ein the hematological depression and the resulting disease state following exposure to total body irradiation.

Under catastrophic conditions it of course will not be possible to adhere to the above regimen. The principles of therapy remain the same, except that here there is a much better potential case for widespread and empirical antibiotic dispensation, particularly to individuals in which burns, mechanical wounds or other added trauma exist.

LONG TERM EFFECTS ON THE BLOOD OF A SINGLE EXPOSURE TO IONIZING RADIATION

The best single source of information on this subject is the Japanese survivors exposed at Hiroshima and Nagasaki in August, 1945. Of necessity, data on the immediate radiation effects are fragmentary and data on the exposed individuals between the 16th week and the 2nd year are not available. In 1954 a statistical analysis of the hematological data obtained by studies on Hiroshima survivors and a control population, carried out from 1950–1953 (ABCC Program ME SS) showed that there was no evidence for an increase in leukopenia, leukocytosis or anemia in the exposed as compared to the control population. During this period several cases of aplastic anemia were encountered among the Nagasaki survivors. However, it cannot be definitely stated that these cases were due to atomic radiation. Up to late 1953, no cases of aplastic anemia had been found in the Hiroshima survivors. It should be noted that "aplastic" anemia is not an uncommon blood dyscrasia in the Japanese.

Incidence of leukemia

In contrast, an increased incidence of leukemia among survivors has been clearly established. It has long been known that repeated or single doses of radiation could increase the incidence of leukemia under controlled experimental conditions in laboratory animals. Accordingly, an intensive investigation of the incidence of leukemia in the irradiated survivors in Hiroshima has been carried out by the ABCC. This study is continuing and a statistical analysis, based on the verified cases of leukemia occurring in the Hiroshima survivors, establishes beyond reasonable doubt that the incidence of leukemia was significantly increased in exposed individuals.

The following graphs and tables are based on the 1947–1953 incidence of leukemia in Japan and have been supplied by the Committee on Atomic Casualties of the National Research Council.

[7] Antibiotics of great value in the therapy of infection in the exposed individual are not considered here as specific drugs. Such prophylactic agents known to this panel such as sulfhydryl compounds, hypoxia inducing drugs, spleen or bone marrow preparations, etc., claimed or shown to favorably modify acute radiation injury in animals, have no place as yet in the treatment of human radiation injury.

Leukemia in exposed persons—Number and rate by presence of radiation symptoms and distance from hypocenter

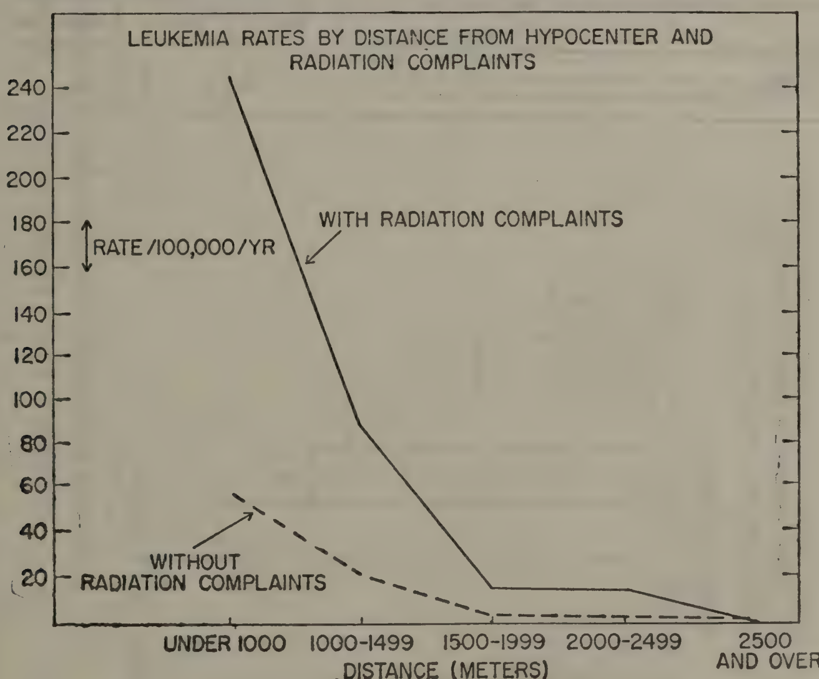
Distance from hypocenter—meters	Hiroshima population [1]			Number of cases of leukemia [2]			Incidence		
	SRC [3]	NRC [4]	Total	SRC	NRC	Total	SRC	NRC	Total
Under 1,000	750	450	1, 200	14	2	16	246. 2	58. 6	175. 8
1,000 to 1,499	2, 250	8, 250	10, 500	15	13	28	87. 9	20. 8	35. 2
1,500 to 1,999	1, 750	16, 950	18, 700	2	4	6	15. 1	3. 1	4. 2
2,000 to 2,499	950	16, 250	17, 200	1	1	2	13. 9	0. 8	1. 5
2,500 over	850	49, 650	50, 500	0	8	8	--------	2. 1	2. 1
Total	6, 550	91, 550	98, 100	32	28	60	64. 4	4. 0	8. 1

[1] Source: Population estimated and rounded off to the nearest 50 persons. These population figures were based on the Commission's 1949 Radiation census and the Japanese national census (1950). Numbers of survivors with severe radiation complaints were estimated from observations made by the Commission's genetics department on 19,675 Hiroshima survivors of childbearing age.

[2] Source: Listing of Leukemia Cases in Hiroshima and Nagasaki, Sept. 1955. Cases are restricted to those in persons resident in Hiroshima at the time of diagnosis, and described in the listing under the heading, Diagnosis Acceptable.

[3] SRC: Significant radiation complaints—Epilation or purpura on history not confirmed by competent physical examination or medical records.

[4] NRC: No radiation complaints.



Leukemia in persons exposed within 1,500 meters of the hypocenter—Number and rate, by sex and age ATB [1]

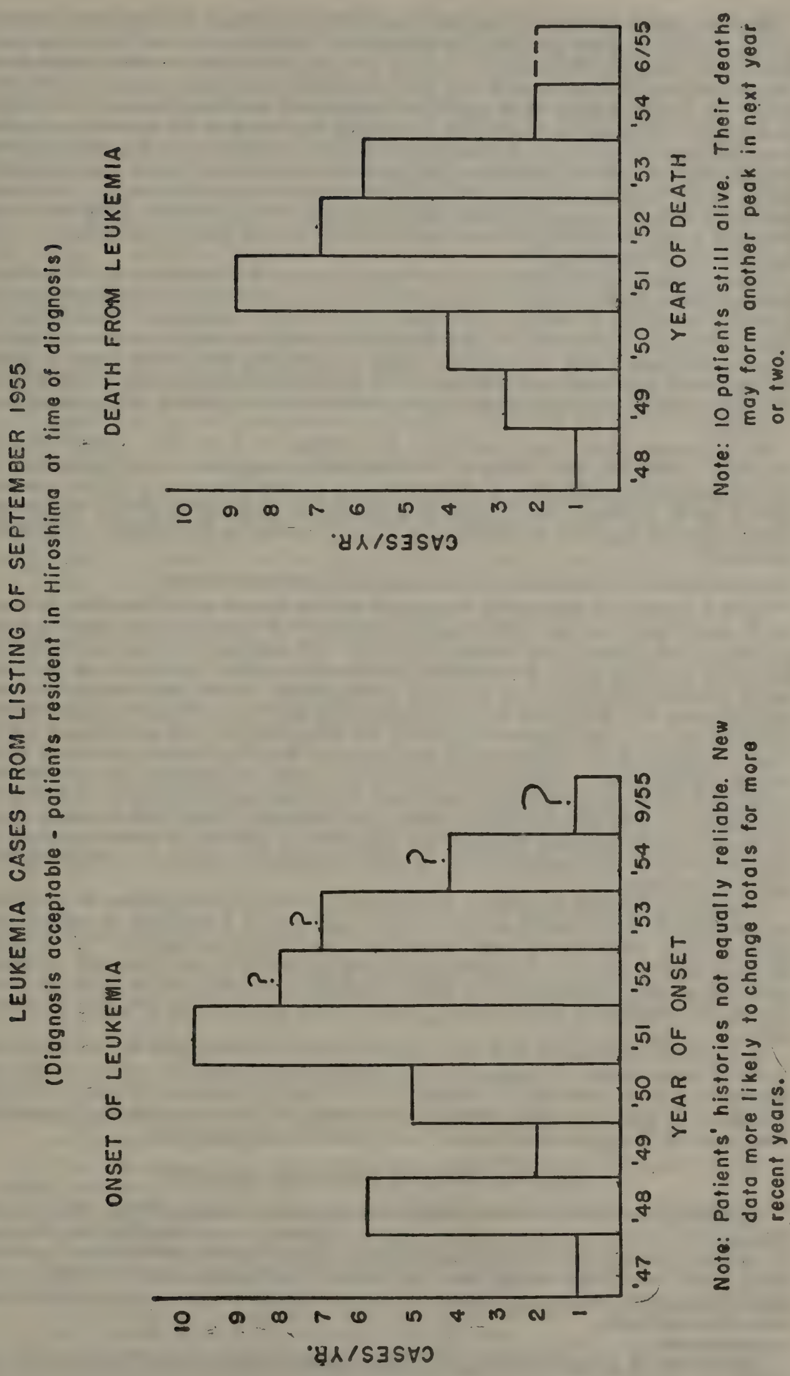
Age ATB	Hiroshima population, 1950		Number of cases of leukemia [2]		Incidence, annual rate per 100,000	
	Male	Female	Male	Female	Male	Female
0 to 9	839	878	6	6	94.3	90.1
10 to 19	995	1,490	7	2	92.8	17.7
20 to 29	458	1,352	3	6	86.4	58.5
30 to 39	713	1,118	3	2	55.5	23.6
40 to 49	902	1,016	3	2	43.9	26.0
50 to 59	606	572	1	2	21.8	46.1
60 to 69	236	278	----------	1	----------	47.4
Total	4,749	6,704	23	21	63.8	41.3

[1] Source: "Estimated Number of Survivors in Hiroshima City in 1950," Preliminary Report, Death Certificate Survey.

[2] Source: Listing of Leukemia Cases in Hiroshima and Nagasaki, September 1955. Cases are restricted to those in persons resident in Hiroshima at the time of diagnosis, and described in the listing under the heading, Diagnosis Acceptable.

Leukemia rates in USA as listed on the 1951 record of Vital Statistics

	Male	Female	Overall rate
White	7.6	5.3	6.1
Nonwhite	4.0	2.7	-------------


LEUKEMIA CASES FROM LISTING OF SEPTEMBER 1955
(Diagnosis acceptable - patients resident in Hiroshima at time of diagnosis)
ONSET OF LEUKEMIA
CASES/YR.
10
9
8
7
6
5
4
3
2
1
'47
'48
'49
'50
'51
'52
'53
'54
9/55
?
YEAR OF ONSET
Note: Patients' histories not equally reliable. New data more likely to change totals for more recent years.
DEATH FROM LEUKEMIA
CASES/YR.
'48
'49
'50
'51
'52
'53
'54
6/55
YEAR OF DEATH
Note: 10 patients still alive. Their deaths may form another peak in next year or two.

The preceding tables and graphs clearly indicate that the incidence of leukemia increases with the dose of radiation. The incidence of radiation complaints at 2000 to 2500 meters is open to question since the dose received at these distances was between 8 and 35 r according to the best source of information.[8] It is noteworthy that radiation complaints are based on late interrogation and not on medical records by competent observers at the time of the bombing and as such are prone to subjective error in memory by the individuals interrogated. At closer distances this possible error is probably negligible because of the unquestioned and recorded histories of radiation injury. The peak incidence of onset appears to have been passed in 1951; however, scattered cases are still being observed, but relationship to confirmed previous exposure to radiation injury is obscure.

Since precise data on the radiation doses received at Hiroshima and Nagasaki have not been made available to the ABCC it is not possible to set a "Dose threshold" for induction of leukemia in man by whole body radiation. Such information is vital to the future welfare of human beings who may be forced to live in contaminated areas. In addition, with the growing belief that there may not be threshold levels for radiation effects it becomes absolutely essential to obtain the most reliable estimate of the exposure wherever osbervable effects have been detected.

In Japanese atom bomb survivors the leukemias have been predominantly myeloid. However, Dr. Furth of the committee emphasizes that radiation induced *lymphatic* leukemias in Europeans have been observed. In this connection it is of interest that lymphatic leukemia is a rarity in Japan suggesting that radiation is prone to induce the type of leukemia that might occur spontaneously.

Hematologic changes preceding development of obvious leukemia

In the course of studies on Japanese atomic bomb survivors, observations on hematologic changes preceding development of overt chronic myelogenous leukemia were made in a number of cases. In routine surveys, blood studies revealed evidence of a generalized proliferative effort by the bone marrow, many months before obvious evidence of leukemia. These manifestations were the presence of a small per cent of myelocytes and metamyelocytes, and a very striking increase in absolute numbers of basophils, in the peripheral blood. These changes were accompanied by increaed numbers of platelets and occasional normoblasts. Beginning in November, 1952, biochemical studies on the separated leukocytes demonstrated that in these early pre-clinical cases of leukemia, the polymorphonuclear leukocytes contained very little alkaline phosphatase. These alkaline phosphatase values were similar to those reported by Valentine et al. for neutrophiles in well established cases of chronic myelogenous leukemia.

Subsequent studies in chronic myelogenous leukemia, employing histo-chemical as well as biochemical methods, have shown that 2 per cent or less of segmented polys contain even small amounts of alkaline phosphatase. In contrast, in other conditions with increased polymorphonuclear leukocytes such as infection and myeloid metaplasia, the alkaline phosphatase values are high and practically all segmented neutrophiles contain large amounts of alkaline phosphatase.

It has been postulated that the leukemic cell is deficient and in the precursor stage of development of leukemia, two populations of cells are present with the leukemic type increasing in number until a typical leukemic blood picture is evident. It has been the experience of some of the Subcommittee members that cases of "bone marrow failure" may terminate in leukemia.

EFFECTS OF REPEATED LOW LEVEL EXPOSURE

Increasing numbers of human beings are exposed to repeated does of radiation frequently at very low dose levels. Thus in industry and in AEC installations, in radiologists and radiological technicians, public health surveys and particularly those using fluoroscopy, and in repeated roentgenograms in medical and dental diagnosis, large populations are exposed to radiation at levels well in excess of background.

[8] The Effects of Atomic Weapons, U. S. Government Printing Office, Washington, D. C., 1950.

There are several studies on radiologists and radiological technicians, indicating that statistically the blood counts of such individuals may be altered. Similarly from the vast number of counts on individuals exposed to low level radiations at AEC installations, there is evidence that the so-called maximum permissible dose may result in statistical alteration in the blood count. The possible significance of these small changes is not clear. The slight decrease in neutrophiles or lymphocytes count has little or no significance in itself. It would appear that its significance in relation to the later development of leukemia or other disease that shortens life span should be investigated. Recommendations to this effect are given below. Data are available on the hematological effects of exposure to radiation up to 10 times background. The drinking water of prisoners at Joliet prison in Illinois contain 20 times the radium content of the drinking water of neighboring communities. Extensive study has failed to detect differences ascribable to the increased radium content of the drinking water. In the course of radiotherapy for relatively benign conditions, it seems clear that serious late effects can result from a single exposure or a series of exposures to x- or isotopic radiations. Thus thyroid cancer has resulted in children given x-radiation for thymic enlargement. Similarly, leukemia has been reported in individuals receiving repeated x-radiation therapy for spondylitis, and in patients receiving repeated I–131 for thyroid cancer.

USEFULNESS OF HEMATOLOGIC STUDIES IN CONTROL OF RADIATION INJURY

A large effort at great expense was made by the AEC during the development of atomic energy to determine if routine hematologic studies would detect low level exposure to radiation. It was the consensus that frequent routine studies on personnel exposed to low levels of radiation have a limited value that does not justify the expense. Physical control of the environment by radiation monitoring is an effective means of maintaining a safe environment, and nothing is gained by widespread hematologic studies on personnel. However, it would not be wise to dispense completely with hematologic studies since it is important to have pre-exposure levels in the individuals who may be exposed to radiation such as with those accidentally exposed to fallout radiation. Had base line studies been available relative depression and recovery time as a function of dose could have been more precisely determined. Accordingly it is believed that periodic, perhaps annual hematologic studies should continue on limited groups of individuals who run a greater risk of accidental over-exposure. Certainly, all individuals who have been exposed so accidentally at dose levels of 25 r or more of essentially whole body radiation (single exposure) should have periodic systematic studies to determine the degree of hematologic depression and the recovery rate. These individuals should remain away from an environment where further overexposure is likely until the dose received has been amortized at the rate of 0.3 r/week. The latter is suggested because clinical radiation therapy experience indicates that individuals who have been exposed previously as a result of local therapy or whole body exposure show greater hematologic depression following further whole body radiation. A more fruitful field of hematologic study in relation to chronic radiation exposure would appear to be the periodic study of phosphatase content of the neutrophils and number of basophils, on limited populations, who are known to be exposed chronically, such as radiologists, urologists, orthopedists, x-ray technicians, and dentists.

RECOMMENDATIONS FOR RESEARCH

A. Recommendations With Respect to the Acute Hematological Response of Human Beings to Radiation

(1) The Japanese data from Hiroshima and Nagasaki bombings should be further analyzed in respect to:

(a) Duration of depression of leukocytes as a function of distance and shielding (dose).

(b) Leukocyte counts at various intervals in relation to ultimate survival.

(c) Survival time as a function of distance and shielding (dose), and of age.

(d) The degree of initial blood count depression in relation to the later development of leukemia and other late disease.

(2) Additional and intensive studies should be initiated on human beings receiving radiation to the whole body or large portions of the body in the therapy of malignant disease. Particular attention should be given to the time course of

peripheral blood counts for several weeks following exposure to different doses, and the nature of the clotting defect.[9]

(3) Initiate studies on the cause of death in animals in which death from hemorrhage and infection have been prevented. This refers to deaths within the first few weeks, as opposed to the much later deaths from nephritis, neoplasia, etc.

(4) Although nothing of practical value is now available for the specific therapy of acute radiation injury, it is urged that further research be pursued on the fundamental defects produced by ionizing radiation on mammalian systems. *Medical experience has shown that rational therapy is only developed when the basic physiologic defects are understood.* With this in mind further research is needed. However, it would be unfair to the public to imply that effective therapy can be expected in the near future or indeed that overwhelming doses are ever likely to yield to therapy.

B. Recommendations With Respect to Long Term Effects of a Single Exposure to Ionizing Radiation

(1) Periodic hematologic surveys should be performed on the Marshallese and Americans exposed to fallout radiation in March, 1954. Careful study for cytological changes mentioned above, especially basophilocytosis and immature leukocytes, and routine bistochemical studies for alkaline phosphatase (using peripheral blood smears and either Gomori's Cobalt technique or the azo dye method) should be carried out. In suspicious cases, biochemical determinations for alkaline phosphatase on separated leukocytes should be done. In view of the long "latent period", studies for many years after exposure if not for life, are essential.

(2) The cytologic and histochemical-biochemical studies might well be employed in surveys of radiologists and other chronically exposed groups.

(3) The present concepts of leukemoid reactions and myeloid metaplasia, and the relationship of these disorders to leukemia are obscure. Further studies on the enzyme and metabolic activities of leukocytes in these disorders may lead to a better understanding of radiation effects on myeloid cells and the role of irradiation in leukemogenesis.

(4) It is generally recognized that routine hematologic studies of potentially exposed individuals are wasteful and unproductive. However, studies on select groups by routine and newer techniques are highly desirable, e. g., radiologists, physicist.

(5) It was the consensus that it would be desirable to know the incidence of leukemia in WW I soldiers who were exposed significantly to mustard gas.

(6) Pediatricians have fluoroscoped newborn babies, and a considerable dose of whole body radiation may have been received. A long term follow-up on these exposed children is needed.

C. Recommendations With Respect to Effects of Repeated Low Level Exposure

(1) Since there are geographical locations in which the known radiation intensities vary considerably, it is felt that the incidence of leukemia should be established in:

(a) Island populations (low background except Baltic Islands)

(b) Andes (high background)

(c) Prison and civil population in Joliet (radium content in water higher than normal)

(2) It was the consensus that a ceaseless search should be continuously made for other harmful agents in the atmosphere and our modern diet. It is genuinely felt that preoccupation with radiation may obscure other equally hazardous factors in man's environment.

CONCLUSIONS

At the commencement of the deliberations there was some question in the minds of the Subcommittee as to the objectives and the reasons for establishing it. However, in the course of the discussions it became apparent that in addition to the confusion in the minds of the public there also exists some large gaps in knowledge essential for the understanding and quantification of radiation hazards in the world of today let alone the world of the future. The immediate effects of direct exposure to high intensity radiation are well documented and the relation

[9] One member of the panel reactivated the heparinemia concept of radiation hemorrhage by describing the cessation of bleeding in an irradiated individual with thrombopenia following injection of protamine, an antiheparin agent.

of dose to effect is known with some degree of confidence, even though certain hiatuses exist that are listed in the general discussion and recommendations. In the realm of chronic exposure it was recognized that the unavoidable background level of radiation was known to vary with seasons of the year, geographical location, and altitude above sea level. However, world-wide levels do not seem to be known with sufficient accuracy to determine when a rise in atmospheric level of radiation is definitely occurring. Since there is little quantitative information on the relation of dose to effect under conditions where harmful effects were observed it becomes vital to ascertain natural levels of radioactivity and to try to establish the level of atmospheric radioactivity at which detectable chronic effects might conceivably occur. However, with all of the recommendations contained in this report, it is believed that a "crash-type" research program to obtain needed information is not indicated.

Bibliography—Radiation Effects on Blood and Blood-Forming Tissues of Man—1900—March 1956

Prepared by Marjorie Comstock, Research Library, Brookhaven National Laboratory

INTRODUCTION

The literature was searched for references relating to the effects of radiation on blood and blood-forming tissues of man. In this bibliography, radiation includes radiation from neutrons, alpha, beta, gamma rays, A-bomb, x-rays and internally deposited radioisotopes. References relating to ultraviolet, infra-red, microwaves, thermal radiation and visible light have been omitted.

The following sources were checked:—

- Chemical Abstracts (CA) v. 1 (1907)–v. 48 (1954)
- Chemische Zentralblatt (CZ) 1897–1906
- Current List of Medical Literature (CLML) v. 19 (1950)–v. 29 (March 1956)
- Nuclear Science Abstracts (NSA) v. 1 (1948)–v. 10 (March 15, 1956)
- Quarterly Cumulative Index Medicus (QCIM) v. 1 (1927)–v. 53 (1953)
- Quarterly Cumulative Index to Current Medical Literature (QCI) v. 1 (1916)–v. 12 (1926)
- Library Files

In addition to the above sources, Zentralblatt für die gesamte Radiologie was also used to check references. References are available in the Research Library unless otherwise indicated. The secondary source, abbreviated as above in parenthesis, is given when possible for references not available at the Laboratory. Journals have been abbreviated according to the Chemical Abstracts list as far as possible. In some cases where the authors initials were known, they have been added in parenthesis although they did not appear either in the original article or in the abstract.

An effort has been made to check references for pertinency. This could not be done for some of the earlier references due to the lack of abstract journals covering the subject. It was necessary to rely solely on the title for many of these entries.

Every effort has been made to make this bibliography complete. However, there is no way of knowing whether or not this has been accomplished.

BIBLIOGRAPHY

EFFECTS OF RADIATION ON THE BLOOD AND BLOOD FORMING TISSUES OF MAN

1956

Irene Joliot-Curie dies from radiation induced leukemia. New York Herald Tribune, Sect. I, p. 58 (March 18, 1956)

1955

Bloom, P. S., A. Querido and C. H. W. Leeksma. Acute leukaemia following x-ray and radioiodine treatment of thyroid carcinoma. Brit. J. Radiol. *28*, 165–6 (1955).

Brown, W. M. C. and J. D. Abbatt. The effect of a single dose of x-rays on the peripheral blood count of man. Brit. J. Haematol, *1*, 75–85 (1955). In Med. Library.

Cronkite, E. P., V. P. Bond, R. A. Conrad, N. R. Shulman, R. S. Farr, S. H. Cohn, C. L Dunham and L E Browning Response of human beings accidentally exposed to significant fall-out radiation. J. Am. Med. Assoc, *159*, 430–4 (1955).
Dienstbier, Z., F. Hermansky and J. Camsky. Mechanism of leukocyte modification immediately following fatal dose of irradiation. Le Sang *26*, 321–5 (1955). (CLML *28*, #47522).
Dowben, R. M. and J. K. Walker. Methemoglobinemia induced by x-irradiation. Proc. Soc. Exptl. Biol. Med. *90*, 398–400 (1955).
Effects of exposure to nuclear radiation. Lancet *268*, 707–8 (1955). In Med. Library.
Egorov, A. P. and V. V. Bochkarev. Blood formation and ionizing radiation. Moscow Medgiz. 1955 *9*, #7621).
Henn, O. and F. Leibetseder. Injury to the hematopoietic system of man by small doses of radiation. Strahlentherapie *97*, 435–443 (1955).
Hutaff, L. W. and H. W. Belding. The effects of irradiation of the pelvis in patients with carcinoma of the cervix uteri on the iliac and sternal marrow and on the peripheral blood. Am. J. Roentgenol. Radium Therapy Nuclear Med. *73*, 251–8 (1955).
Kohn, H. I. Changes in the human leukocyte count during x-ray therapy for cancer and their dependence upon the integral dose. Radiology *64*, 382–391 (1955).
Koslowski, L., H. Popper and E. Walther. Blood histamine determination and skin erythema measurement in constant single dose roentgen irradiation. Strahlentherapie *97*, 266–271 (1955).
Lange, R. D., S. W. Wright, M. Tomonaga, H. Kurasaki, S. Matsuoke and H. Matsunaga. Refractory anemia occurring in survivors of the atomic bombing, Nagasaki, Japan. Blood *10*, 312–324 (1955).
Lavedan, J. Cytologic modifications of the blood during therapeutic irradiations of cancer. Le Sang *26*, 457–476 (1955). (CLML *28*, #58256).
Looney, W. B., R. J. Hasterlik, A. M. Brues and E. Skirmont. A clinical investigation of the chronic effects of radium salts administered therapeutically. (1915–1931). Am. J. Roentgenol. Radium Therapy Nuclear Med *73*, 1006–1037 (1955).
Loutit, J. F. Radiation exposure of staff in diagnostic procedures. I. Blood counts; research or routine? Brit. J. Radiol. *28*, 647–650 (1955).
Maurer, H. J. Biological and biochemical effects of ionizing radiations. III. Problem of the magnesium blood level during deep roentgen therapy. Strahlentherapie *96*, 629–631 (1955).
Moloney, W. C. and M. A. Kastenbaum. Leukemogenic effects of ionizing radiation on atomic bomb survivors in Hiroshima City. Science *121*, 308–9 (1955).
Morczek, A. An investigation of the effects of irradiation on sodium-potassium exchange in erythrocytes. Strahlentherapie *96*, 618–624 (1955).
Pisani, G., A. Romanini and A. Malaspina. Cytopochemistry in leukopenia caused by roentgen irradiation. II. Polysaccharide fractions in polynuclear structures in circulating blood. Boll. soc. ital. biol. sper. *31*, 526–8 (1955). (CLML *29*, #21525).
Reynolds, M. L. and F. X. Lynch. Atomic bomb injuries among survivors in Hiroshima. Public Health Repts. (U. S.) *70*, 261–270 (1955). In Med. Library.
Schattmann, K., W. Schlungbaum and P. Kuhne. Influence of ionizing radiations on 17-ketosteroid elimination and eosinophil count. Arztl. Forsch. *9*, p. I/342–6 (1955). (CLML *29*, #534).
Silson, J. E., M. Kleinfeld and S. J. Harris. External radiation exposure in the radium-dial painting industry. Arch. Ind. Health *12*, 503–8 (1955).
Stefanini, M. Hematological aspects of atomic warfare. Minerva med. *46*, 928–936 (1955). (CLML *29*, #24674).
Tripodo, C. and G. Del Buono. Blood coagulation factor VII and experimental hepatopathy. IV. Behavior of factor VII after x-irradiation of the liver. Radioterapia, Bologna *10*, 228–232 (1955). (CLML *28*, #46727).
Tsuzuki, M. Results of radioactive injury of Japanese fishermen from Bikini ash. Münch. med. Wochschr. *97*, 988–994 (1955). (CLML *29*, #17581).
Upton, A. C. The pathogenesis of the hemorrhagic state in radiation sickness; a review. Blood *10*, 1156–1163 (1955).

1954

Avezzu', G. and O. Zardi. Behavior of Quick and Howell Time of the alexinic power of the blood during roentgenotherapy. Giron. clin. med. (Parma) *35*, 837–845 (1954). (CLML *27*, #12480).

Benthaus, R. The reaction of the leukocytes and lymphocytes to fractional roentgen irradiation. Strahlentherapie *93*, 124–9 (1954).
Brown, W. M. C. and R. F. Mahler. Integral dose, body size, and the site of irradiation as factors determining the severity of radiation sickness. J. Fac. Radiologists, London *5*, 200–9 (1954). (CLML *28*, #43439).
Burdon, K. L. and R. K. Guthrie. Changes in the proteolytic and antiproteolytic activity of the blood serum in man and animals following exposure to x-rays. Baylor Univ. and School of Aviation Med. 1954. 28p. NP 5257 (Microcard).
De Plaen, P., G. Lion and R. van Male de Ghorain. Blood modifications caused by radiotherapy. J. belge radiol. *37*, 41–57 (1954).
Dienstbier, Z., J. Camsky and V. Maly. Mechanism of early modification of leukocyte count following lethal dose of roentgen rays. II. Effect of anesthesia and of mercaptoethylamine; neurohumoral regulation of white blood picture. Casopis Lekarus Ceskych *93*, 1364–7 (1954). (CLML *28*, #2920).
Fletcher, N. R. The blood count in radiographers; a group survey. Radiography *20*, 19–24 (1954). (NSA *8*, #1782).
Foti, M. and I. Deak. The effect of roentgen rays on eosinophils. Z. ges. inn. Med. u. ihre Grenzgebiete *9*, 564–7 (1954). Also in Magyar radiol. *6*, 129–132 (1954). (CLML *27*, #17835, #34966).
Helske, E. Injuries by atomic weapons; review. Sotilaslaak aikak *29*, 154–168 (1954). (CLML *27*, #37908).
Hornykiewytsch, T., G. Seydl and O. W. Thiele. Histochemical and serological studies on the effect of x-rays. IV. Studies on plasmalogen and acetalphosphatidies in blood in vitro and in vivo following irradiation with varying high doses of roentgen rays. Strahlentherapie *95*, 523–6 (1954).
Jasinski, W. Postirradiation reaction of the blood in occupational exposure to ionizing radiations. Polski przeglad. radiol. *18*, 51–66 (1954). (CLML *26*, #48719).
Keyser, J. W. Effect of nitrogen mustard or x- or radium-irradiation treatment on the level of blood serum protein-bound carbohydrate (polysaccharide) in patients with neoplastic diseases. Brit. J. Cancer *8*, 238–250 (1954).
Krajewski, F. Biernacki's reaction as a test of changes in the blood following irradiation with massive doses of roentgen rays. Polski Tygodnik Lekarski *9*, 1091–9 (1954). (CLML *27*, #48793).
Krajewski, F. Effect of small doses of roentgen rays on erythrocyte sedimentation rate. Polski Tygodnik Lekarski *9*, 1314–8 (1954). (CLML *27*, #48862).
Lange, R. D., W. C. Moloney and T. Yamawaki. Leukemia in atomic bomb survivors. I. General observations. II. Observations on early phases of leukemia. W. C. Moloney and R. D. Lange. Blood *9*, 574–585 (1954); ibid 663–685 (1954).
Lasser, E. C. and K. W. Stenstrom. Radiation sickness. A study of its relation to adrenal cortical function and the absolute eosinophil count. Am. J. Roentgenol. Radium Therapy Nuclear Med. *72*, 474–487 (1954).
Levitt, W. M. Leukaemia as a radiation hazard. Med. Illust. (Lond.) *8*, 188–9 (1954). (CLML *26*, #16261).
Lorenz, E. and C. C. Congdon. Radioactivity; biologic effects of ionizing radiation. Ann. Rev. Med. *5*, 323–338 (1954).
Maurer, H. J. Effect of ionizing rays; clarification of biochemical processes following depth roentgen irradiation. Klin. Wochschr. *32*, 37–9 (1954).
Moloney, W. C. and R. D. Lange. Cytologic and biochemical studies on the granulocytes in early leukemia among atomic bomb survivors. Texas Rept. Biol. and Med. *12*, 887–897 (1954).
Muth, H. Radiation injuries caused by ionizing rays. Strahlentherapie *94*, 527–538 (1954).
Patt, H. M. and A. M. Brues. The pathological physiology of radiation injury in the mammal. II. Specific aspects of the physiology of radiation injury. (In Radiation Biology, volume I, high energy radiation, A. Hollaender, ed. New York. McGraw-Hill Book Co., Inc. 1954, pt. II, p. 959–1028).
Piazzi, M. Decrease of eosinophils following diencephalo-pitiuitary irradiation with small doses. Folia Endocrinol. (Pisa) *7*, 599–603 (1954). (CLML *27*, #44145).
Pisani, G. and O. Andreoni. Statistical method of evaluation of electrophoretic behavior of serum proteins, of chloruremia, and of hematocrit value in cancerous patients exposed to roentgentherapy. Radiol. med., Tor. *40*, 385–392 (1954). (CLML *26*, #40760).

Raubitschek, H. V. Pathologic findings in victims of the A-bomb attack on Nagasaki and Hiroshima. Wien klin. Wochschr. *66*, 143–5 (1954). (CLML *26*, #19944).

Ravina, A. The first known effects of hydrogen bomb on man. Presse med. *62*, 881 (1954). (CLML *26*, #40384).

Shirabe, R. Medical survey of atomic bomb casualties. Atomic Bomb Casualty Commission. 1954. 29p. NYO 4456.

Stelling, M. D. Effect of radiation on the blood. X-ray Technician *25*, 415–432 (1954). (CLML *26*, #20022).

Stoliarova, L. B. and R. D. Nikitenko. Effect of intravenous administration of indicator doses of artificially radioactive iron and phosphorus on the composition of peripheral blood. Med. Zhurnal (Ukraine) *24*, 42–7 (#5) (1954). (CLML *28*, #55954).

Verstraete, M. Hemorrhagic syndrome due to irradiation. Ann. Belges med. militaire *107*, 67–73 (1954). (CLML *26*, #34426).

Vetter, H. and H. Vinazzer. Proaccelerin deficiency following irradiation with therapeutic doses of radiophosphorus. Blood *9*, 163–172 (1954).

1953

Astaldi, G., E. G. Rondanelli, P. Gorini and C. Zelaschi. Studies on proliferation of erythroblasts in culture in the blood plasma irradiated with roentgen rays. Rev. hematol. *8*, 477–488 (1953). (CLML *26*, #30519).

Balmukhanov, S. B. and F. A. Okun. Effect of X-rays on the blood picture in goiter patients. Vestnik Akad. Nauk Kazakh. S. S. R. *10*, no. 12, (whole no. 105) 83–7 (1953). (CA *48*, p. 6598).

De Bernardi, E. and A. Lendini. Early reaction of the blood to radiations. J. radiol. et electrol. *34*, 165–8 (1953).

Decowski, M. Effect of radium on bacteriostatic properties of the blood in a case of cancer of the cervix uteri. Polski Tygodnik Lekarski 8, 1606–8 (1953). (CLML *26*, #7807).

De Vido, G. and A. Ancetti. The behavior of the proteinemia, hemometry and hematocrit before and during radium and roentgentherapy. Oto-Rino-Laringologia Italiana *21*, 358–369 (1953). (CLML *26*, #17504).

Hecht, H., A. Neumayr and B. Thurnher. Indirect effect of x-irradiation of pituitary-diencephalic region on capillary permeability in man. Strahlentherapie *91*, 261–9 (1953).

Helde, M. and T. Wahlberg. Some observations on the relation between radiation dose measures and blood changes. Acta Radiol. *40*, 435–442 (1953).

Hempelmann, L. H. and J. G. Hoffman. Practical aspects of radiation injury. Ann. Rev. Nucl. Sci. *3*, 369–392 (1953).

Kusano, N., ed. Atomic bomb injuries. Edited by Japanese Preparatory Committee. Tokyo. Tsukiji Shokan Co. 1953. 112p.

Loeffler, R. K., V. P. Collins and G. A. Hyman. Comparative effects of total body radiation, nitrogen mustard, and triethylene melamine on the hematopoietic system of terminal cancer patients. Science *118*, 161–3 (1953).

Matej, H. Effect of explosion of atomic bomb on the human organism. Postepy hig. med. dosw. 7, 235–283 (1953). (CLML 27, #6010).

Maurer, H. J. and K. H. Pfeffer. Changes of adenosinetriphosphoric acid in the blood and urinary 17-ketosteroids excretion during irradiations with deep roentgen rays. Strahlen-therapie *92*, 19–24 (1953).

Morczek, A. Experimental studies on the behavior of potassium ions during ionizing radiations of the erythrocytes. Z. ges. inn. Med. u. ihre Grenzgebiete *8*, 684–7 (1953). (CLML *26*, #32864).

Pagani, C. Serum protein picture and its variations during roentgen therapy of carcinoma of the uterus. Ann. ostet. ginencol. *75*, 1189–1220 (1953). (CLML *25*, #46563).

Pape, R. Effects of low x-ray doses on hemopoietic organs. Strahlentherapie *91*, 108–114 (1953).

Pasqualini, R. Q. Clinical effects of the atomic bomb. Rev. san. mil. Agrentina *52*, 329–339 (1953). (CLML *25*, #55208).

Pavlovsky, A. Repercussion of radiations on hemopoietic organs. Medicina (Buenos Aires) *13*, 58–63 (1953). (CLML *24*, #33645).

Spear, F. G. Radiation and living cells . . . New York. John Wiley & Sons. 1953, p. 94–118.

Verhagen, A. and H. Suren. Behavior of prothrombin level during radium therapy of gynecological diseases. Strahlentherapie *91*, 282–6 (1953).

Wagner, H. Irradiation of the spleen and blood morphology. Strahlentherapie *91*, 115–9 (1953).
Wagner, K. Roentgen and leukemia. Krebsarzt. *8*, 330–4 (1953). (CLML *25*, #52012).
Willbold, O. Blood and sternal marrow in roentgen irradiation of lymphogranulomatosis. Strahlentherapie *91*, 103–7 (1953).
Yamasowa, Y. Hematologic studies of irradiated survivors in Hiroshima, Japan. Arch. Internal Med. *91*, 310–4 (1953). In Med. Library.

1952

Birkner, R., J. G. Frey and J. Trautmann. Effect of roentgen rays on prothrombin potential of human blood. Strahlentherapie *88*, 44–52 (1952).
Bjerkelund, C. J. Polycythemia vera-radioactive phosphorus-acute leukemia. Nord. Med. *48*, 1037–8 (1952). In Med. Library.
Bourdeau, R. and H. Laufman. Local hemostasis in the hemorrhagic syndrome of irradiation. Arch. Surg. *64*, 154–8 (1952). In Med. Library.
Bugher, J. C. Delayed radiation effects at Hiroshima and Nagasaki. Nucleonics *10*, #9, 18–21 (1952).
Chamberlain, A. C., F. M. Turner and E. K. Williams. The evaluation of white-cell counting in radiation protection. Brit. J. Radiol. *25*, 169–176 (1952).
Clark, M. L. and F. X. Lynch. Clinical symptoms of radiation sickness, time to onset and duration of symptoms among Hiroshima survivors in the lethal and median lethal ranges of radiation. Military Surgeon *111*, 360–8 (1952). (CLML *23*, #26702).
Cliffton, E. E. Variations in the proteolytic and antiproteolytic reactions of serum: effect of disease, trauma, x-ray, anaphylactic shock, ACTH and cortisone. J. Lab. Clin. Med. *39*, 105–121 (1952).
Crisalli, M. and C. Marenzana. Effect of splenic irradiation; experimental study. Minerva pediat. *4*, 1049–1051 (1952). (QCIM *53*, p. 286).
Cronkite, E. P. and G. Brecher. Effects of whole body irradiation. Ann. Rev. Med. *3*, 193–214 (1952).
Cronkite, E. P., G. J. Jacobs, G. Brecher and G. Dillard. The hemorrhagic phase of the acute radiation syndrome due to exposure of the whole body to penetrating ionizing radiation. Am. J. Roentgenol. Radium Therapy Nuclear Med. *67*, 796–804 (1952).
Cutler, S. S., B. Burbank and E. R. Marzullo. Hypocoagulability of irradiated plasmas. J. Am. Med. Assoc. *148*, 863–4 (1952). In Med. Library.
Di Bella, S. Effect of x-rays on the content of pyruvate and lactate of the blood and on the glycolysis of the bone marrow. Boll. soc. ital. biol. sper. *28*, 1974–6 (1952). (CLML *24*, #29796).
di Rienzo, S. Effects of irradiation on blood. Rev. med. Cordoba *40*, 306–312 (1952). (QCIM *52*, p. 1295).
Donner, M. Hyperprothrombinemia due to roentgen irradiation and therapeutic sequels. Deutsche med. Wochschr. 77, 1326–8 (1952). (QCIM *52*, p. 288).
Effects of radiations on the hemopoietic system. Le Sang *23*, 796–7 (1952). (CLML *24*, #18998).
Folley, J. H., W. Borges and T. Yamawaki. Incidence of leukemia in survivors of the atomic bomb in Hiroshima and Nagasaki, Japan. Am. J. Med. *13*, 311–321 (1952). In Med. Library.
Gottlob, R. Radiation effects on large blood vessels of extremeties. Wien, klin. Wochschr. *64*, 361–2 (1952). (QCIM *52*, p. 1297).
Grassi, G. Protein modifications in the serum of patients with uterine and vaginal neoplasms during radium and roentgen therapy. Quaderni clin. ostet. e ginecol. 7, 349–368 (1952). (NSA 7, #730).
Hewitt, J. E., T. L. Hayes, J. W. Gofman, H. B. Jones and F. T. Pierce. Effects of total body irradiation upon lipoprotein metabolism. Cardiologia *21*, 353–365 (1952). (CLML *23*, #44697).
Ingram, M., M. Adams, L. Coonan, J. Jespersen, G. Nielsen, D. Piatt and G. Yettewich. The occurrence of lymphocytes with bilobed nuclei in cyclotron personnel. Science *116*, 706–8 (1952).
Kahn, R. L. and F. J. Hodges. Final report on universal serologic reaction following irradiation for period July 1, 1949–June 30, 1952. Univ. Mich. 66p. AECU 2266. (Microcard).
Kikuchi, T. and G. Wakisaka. Hematological investigation of the atomic bomb sufferers in Hiroshima and Nagasaki cities. Acta Schol. Med., Univ. Kioto *30*, 205–237 (1952). (CA 47, p. 9378).

Leibetseder, F. Monocytic leukosis following chronic roentgen-radium injury. Med. Klin. (Munich) *47*, 46–8 (1952). (NSA *6*, #3901).

Pape, R. and G. Koller. Hourly reaction of leucocytes. Strahlentherapie *87*, 399–414 (1952).

Patt, H. M. Some aspects of the biological action of high energy radiations. Ann. Rev. Nucl. Sci. *1*, 495–524 (1952).

Peller, S. and P. Pick. Leukemia and other malignancies in physicians. Am. J. Med. Sci. *224*, 154–9 (1952).

Seegars, W. H. Blood clotting mechanisms and acute radiation injuries. J. Michigan State Med. Soc. *51*, 344 (1952). (CLML *21*, #39099).

Stelling, M. D. Effect of radiation on the blood. Radiography *18*, 221–231 (1952). In Med. Library.

Vetter, H. and H. Vinazzer. Coagulation defect following therapeutic doses of radioactive isotopes. Wien. Z. inn. Med. *33*, 455–7 (1952). (QCIM *53*, p. 1346).

1951

Beller, F. K. Blood prothrombin level in diagnosis of x-ray and radium injuries in radiological personnel. Fortschr. Gebiete Röntgenstrahlen *75*, 95–8 (1951).

Brüda, B. Modifications of blood morphology by injuries of Roentgen irradiations. Artzl. Wochschr. *6*, 402–4 (1951). (CLML *20*, #61818).

Chamberlain, A. C. and F. M. Turner. Statistical review of haematological techniques and records. Atomic Energy Res. Estbl. 1951. 36 p. AERE MED/R 708.

Furth, J., G. A. Andrews, R. H. Storey and L. Wish. The effect of x-irradiation on erythrogenesis, plasma and cell volumes. Southern Med. J. *44*, 85–92 (1951). (NSA *5*, #1743).

Grünberger, V., F. Pakesch and W. Rankl. Effect of roentgen ovarian castration on the blood picture. Wien. klin. Wochschr. *63*, 525–7 (1951). (CLML *20*, #100987).

Hartl, F. Cholesterol content of cadaveric blood in neoplastic malignancy; its relation to liver function and the effects of radiotherapy. Naunyn-Schmiedeberg's Arch. eptl. Pathol. u. Pharmakol. *213*, 235–248 (1951).

Henstell, H. H., I. S. Henstell and E. M. Ornitz. Electrolytic resistance of blood clot in polycythemia vera before and after radiation and its relationship to clot retraction. Am. J. Clin. Pathol. *21*, 820–7 (1951). In Med. Library.

Ingram, M. and S. W. Barnes. Experimental confirmation of a previously reported unusual finding in the blood of cyclotron workers. Science *113*, 32–4 (1951).

Ishibashi, K. Clinical observations of atomic bomb radiation sickness. Pt. IV. Nagoya J. Med. Sci. *14*, 193–205 (1951). (NSA *8*, #3218).

Jacobson, L. O., E. K. Marks and E. Lorenz. Hematological effects of ionizing radiations. (In Industrial medicine of the plutonium project. R. S. Stone, ed. New York. McGraw-Hill Book Co. 1951. p. 140–196).

Lindemann, B. Roentgen hemolysis; a contribution on the biologic effects of ionizing radiations. Fortschr. Gebiete Röntgenstrahlen *75*, 523–559 (1951).

Lynch, F. W. Leukemia as possible complication of radiodermatitis. Arch. Dermatol. and Syphilol. *63*, 503 (1951). In Med. Library.

Lyttleton, J. W. and L. Vallet. Coagulability of irradiated plasma. Lancet *260*, 1102–3 (1951). In Med. Library.

Maloney, M. A. Anemia in thermal and radiation injury. Am. J. Med. Technol. *17*, 238–241 (1951). (CLML *21*, #8064).

Manzanilla, M. A. and M. A. Manzanilla, Jr. Eosinophilic variations after irradiation of the adrenal glands. J. Intern. Coll. Surgeons *15*, 628–632 (1951). (CLML *20*, #74310).

Manzanilla, M. A. and M. A. Manzanilla Jr. Lymphocytic variations after irradiation of the adrenal glands. J. Intern. Coll. Surgeons *15*, 281–5 (1951). (CLML *20*, #56786).

Moses, C. and M. Platt. Changes in the total circulating eosinophile count in cyclotron workers. Science *113*, 676 (1951).

Moshman, J. Hemotological effects of chronic low-level irradiation. J. Appl. Physiol. *4*, 145–155 (1951).

Neumayr, A. and B. Thurnher. Effect of local roentgen irradiation on permeability of human capillaries. Strahlentherapie *84*, 297–305 (1951).

Oughterson, A. W. et al. Medical effects of atomic bombs; the report of the Joint Commission for the Investigation of the Effects of the Atomic Bomb in Japan. Army Inst. Pathol. 1951. Pts. II–V. NP 3037–NP 3040.

Paper, R. and R. Seyss. The importance of central nervous regulation and reaction site for the hour-by-hour reaction of the blood after roentgen irradiation. Wien. Z. inn. Med. *32*, 214–223 1951). (CLML *20*, #69454).
Picco, A. Pyruvic acid content of blood in cancerous patients and its behavior during radioactive treatment. Tumori *25*, 270–282 (1951). (CA *46*, p. 4107).
Roiz, N. M., J. A. de Argumosa Valdes. Research on glutathionemia in irradiated patients, Medicamenta Madr. *9*, 210–3 (1951). (CLML *20*, #75328).
Sarian, J. N. Irradiated human plasma and phagocytosis. Am. J. Roentgenol. Radium Therapy *65*, 940–6 (1951).
Sarian, J. N. Effect of physical and chemical factors on phagocytosis in presence of irradiated human blood and plasma. Am. J. Roentgenol. Radium Therapy *65*, 465–471 (1951).
Scholl, F. and R. Seyss. Effect of minimal x-ray doses on blood potassium-calcium balance. Wien. Z. inn. Med. *32*, 266–275 (1951). (CLML *20*, #89419).
Sheppard, C. W. and G. E. Beyl. Cation exchange in mammalian erythrocytes; prolytic effects of x-rays on human cells. J. Gen. Physiol. *34*, 691–704 (1951).
Wakabayashi, M. and F. Kawamura. Action of x-ray on the serum proteins. Monograph Ser. Research Inst. Applied Elec. (Japan) Research Biophys, no. 2, 1–10 (1951). In English. (C. A. *46*, p. 8690).
Wied, G. L. and G. Schmidt. Injuries to health following uncontrolled nuclear fission chain reactions. Arztl. Forsch. *5*, no. 12, I/455–465 (1951). (NSA *6*, #52).

1950

Astaldi, G., C. Mauri and L. di Guglielmo. On proliferative activity of erythroblasts of human bone marrow in culture. Haematologica *34*, 529–558 (1950). (QCIM *49*, p. 1224).
Bode, H. G. and H. Theismann. Radiation reaction and peripheral circulation. Dermatol. Worchschr. *121*, 218–222 (1950). (QCIM *47*, p. 237).
Bruda, B. E. Modification of the blood picture due to the injurious effects of roentgen rays. Prakt. Arzt. *4*, 538–540 (1950). (CLML *20*, #16524).
Davis, R. W., N. Dole, M. J. Izzo and L. E. Young. Hemolytic effect of radiation. Observations on renal bile fistula dogs subjected to total body radiation and on human blood irradiated in vitro. J. Lab. Clin. Med. *35*, 528–537 (1950).
Fonio, A. The quantitative effect and the qualitative injury to the functioning of thrombocytes relative to the retraction of blood clots. Bull. schweiz. Akad, med. Wiss. *6*, 115–120 (1950). (NSA *4*, #4437).
Frieden, J. and A. White. Studies of electrophoretic serum protein patterns in subjects treated with pituitary-adrenal cortical hormones, nitrogen mustard, or x-radiation. Yale J. Biol. and Med. *22*, 395–406 (1950).
Helde, M. The relation of irradiation risk to blood picture modifications in roentgen technicians. Strahlentherapie *83*, 283–290 (1950).
Kikuchi, T. et al. Studies on the atomic bomb injuries in Hiroshima City. Report to the Special Research Committee on the Atomic Bomb Disasters. 1950. Kyoto, Japan. Not in library.
Ingram, M. Hematological changes following exposure to radiation (in Medical aspects of civil defense against atomic weapons. J. N. Standard and H. A. Blair, ed. Univ. Rochester. 1950. p. 153–164. UR 112).
LeRoy, G. V. Hematology of atomic bomb casualties. Arch. Internal Med. *86*, 691–710 (1950). In Med. Library.
Lindemann, B. Roentgen hemolysis as structure problem. Fortschr. Gebiete Röntgenstrahlen *72*, 365–370 (1950).
Löw-Beer, B. V. A. and P. M. Aggeler. Hematological effects of ionizing radiations. VII. Progress report, April 1, 1949–February 28, 1950. Univ. Calif. 87p. UCRL 862.
March, H. C. Leukemia in radiologists in a 20-year period. Am. J. Med. Sci. *220*, 282–6 (1950).
Mossberg, H. Platelet values in personnel engaged in roentgen diagnostic work. Acta radiol. *34*, 186–192 (1950).
Ravina, A. Late hematologic effect of atomic bombardment. Presse med. *58*, 907–8 (1950). (QCIM *48*, p. 187).
Watts, W. E. and D. R. Mathieson. Studies on lymphocytes from persons treated with radioactive iodine. J. Lab. Clin. Med. *35*, 885–9 (1950).
Zacek, J. and R. Rosenberg. Action of x-rays on the membrane of red corpuscles. Compt. rend. soc. biol. *144*, 462–4 (1950).
Zeleny, A. Effect of x-irradiation on the level of histamine in blood. Casopis Lekaru Ceskych *89*, 998–1002 (1950). (CLML *20*, #2533).

Zimmer, E. A. Changes produced by x-ray in blood corpuscles. Pts. I–IV. Strahlentherapie *81*, 495–512; 599–616 (1950); ibid *82*, 129–142; 261–280 (1950).

1949

Allen, J. et al. Abnormal bleeding. J. Am. Med. Assoc. *139*, 1251–4 (1949). In Med. Library.

Andrews, G. A. Thrombocytopenic purpura complicating radioactive phosphorus treatment in a patient with polycythemia vera. Am. J. Med. 7, 564–8 (1949). In Med. Library.

Bourret, J. Blood changes in roentgen personnel. Deut. Med. Wochschr. *74*, 447 (1949). NSA *3*, #510).

Browning, E. Blood changes in luminizers using radioactive material. Brit. Med. J. *1*, 428–431 (1949). In Med. Library.

Buchsbaum, R. and R. E. Zirkle. Shrinking and swelling after alpha irradiation of various parts of large erythrocytes. Proc. Soc. Exptl. Biol. Med. 72, 27–9 (1949).

Cronkite, E. P. Ionizing radiation injury; diagnosis by physical examination and clinical laboratory procedures. J. Am. Med. Assoc. *139*, 366–9 (1949). In Med. Library.

Deho, G. G. Modifications of protidemia following roentgen irradiation of marrow. Rass. fisiopat, clin. *21*, 817–828 (1949). (QCIM *47*, p. 259).

Diethelm, L. and W. Helscher. Danger from radiation and the blood picture. Röntgen Blätter *2*, 145–163 (1949). (NSA *4*, #2529).

Drescher, H. Blood findings in cancer patients treated by roentgen irradiation. Zentralblatt Gynäkol. *71*, 1161–4 (1949). (QCIM *47*, p. 247).

Frank A. and W. Punin. Concerning information of a cold-agglutination in vitro after roentgen radiation of blood serum. Klin. Wochschr. *27*, 121–2 (1949).

Ingram, M. and S. W. Barnes. Observations on the blood of cyclotron workers. Phys. Rev. *75*, 1765 (1949).

Liebow, A. A., S. Warren and E. DeCoursey. Pathology of atomic bomb casualties. Am. J. Pathol. *25*, 853–1027 (1949).

Low-Beers, B. V. A. Hematological effects of total body irradiation from external and internal sources. (In Medical and Health Divisions Quarterly Report, Jan. 1949-March 1949. Univ. Calif., Rad. Lab. UCRL 332, p. 65–8).

Miyer, H. W. Effect of ionizing radiations on reproductive cells and white blood cells. X-Ray Technician *20*, 215–218 (1949). NSA *2*, #1057).

NEPA Medical Advisory Panel. Tabulation of available data relative to radiation biology. NEPA Project. 1949. 42p. NEPA 1019.

Ney, H. Can defense mechanism of body be influenced by roentgen irradiation? Report on preliminary clinical investigation of plasma or serum protein pattern in rheumatoid arthritis. Ann. Rheumatic Diseases 8, 191–204 (1949). (QCIM *46*, p. 275).

Paul, T. O. Blood groups and effects of roentgen irradiation in retrolental fibroplasia. Arch. Ophthalmol. (Chicago) *41*, 659–666 (1949). (QCIM *46*, p. 269).

Silverman, S. B. Changes in the coagulability of the blood after radiation therapy. The results of studies using the modified Waugh-Ruddick test for increased coagulability. Am. J. Roentgenol. Radium Therapy *62*, 541–6 (1949).

Snell, F. M., J. V. Neel and K. Ishibashi. Hematologic studies in Hiroshima and a control city two years after the atomic bombing. Arch. Internal Med. *84*, 569–604 (1949). In Med. Library.

Soykova-Pachnerova, E. and O. Soyka. Changes in prothrombin level of female cancer patients treated by radiation. Casopis Lekaru Ceskych *88*, 169–171 (1949). (QCIM *45*, p. 185).

Vogt, A. Concerning the treatment of leukemias with roentgen rays. Deut. Med. Wochschr. *74*, 42–5 (1949). (NSA *3*, #34).

Wilson, R. H. Ionizing radiations. Ohio State Med. J. *45*, 579–581 (1949). (NSA *3*, #221).

1948

Barnard, R. D. A note on the depression of blood cholinestearase level following x-ray therapy and its bearing on the mechanisms of radiation sickness. Med. Record *161*, 350–2 (1948). (CA *44*, p. 10000).

Brown, W. M. C. The value of platelet counts in radiotherapy. Brit. J. Radiology *21*, 221–5 (1948).

Bryan, F. A. Problems relative to possible physiologic effects caused by radiation. Ind. Med. *17*, 367–371 (1948). (QCIM *44*, p. 1017).

Capra, P. Hyperplasia of reticuloendothelium of lymphosarcomatous type with secondary probable atypical myeloid leukemic blood picture; late stage of lym-

phatic metaplasia due to irradiation; case. Haematologica *31*, 85–98 (1948). (QCIM *44*, p. 1054).
Copley, A. L. Hemorrhagic diathesis in Hiroshima, Nagasaki and at Bikini atomic bomb test. J. Am. Med. Assoc. *137*, 145 (1948). In Med. Library.
Cronkite, E. P. The diagnosis of ionizing radiation injury by physical examination and clinical laboratory procedures. Naval Med. Res. Inst. 1948. 15p. NP 573.
Dobson, R. L. and J. H. Lawrence. Physiologic effects of radiant energy. Ann. Rev. Physiol, *10*, 479–500 (1948).
Herrera, J. M. and M. Rognoni. Acute leukemia with tumor of thymic region after anti-asthmatic therapy by irradiation of theoracic region. Arch. Hosp. Santo Tomas *3*, 135–156 (April-June 1948). (QCIM *46*, p. 1239).
Howarth, F. Isotopes and radiation hazards. Lancet *255*, 51–3 (1948). In Med. Library.
Howland, J. W. and S. L. Warren. Effects of atomic bomb radiation on Japanese. Adv. Biol. & Med. Phys. *1*, 387–408 (1948).
Infantile blood system. Science News Letter *54*, 146 (Sept. 4, 1948). (NSA *1*, #1180).
Jacox, H. W. Thrombocytopenic purpura following therapeutic administration of radioactive sodium. Radiology *51*, 860–1 (1948).
Lawrence, J. S., A. H. Dowdy and W. N. Valentine. The effects of radiation on hemopoiesis. Radiology *51*, 400–413 (1948).
Low-Beers, B. V. A. Hematological effects of total body irradiation from external and internal sources. (In Medical and Health Divisions Quarterly Reports, Oct. 1947–Jan. 1948, Jan. 1948–March 1948, April 1948–June 1948, July 1948–September 1948.) UCRL 41, 98, 157, 193.
Price, F. L. Effects resulting from atomic bomb explosion. J. Florida Med. Assoc. *34*, 651–3 (1948). (NSA *2*, #402).
Setälä, K. Preliminary observations on effects of irradiation upon chylomicrons in human blood. Radiology *50*, 803–9 (1948).
Setälä, K. and P. Ermala. Irradiation sickness (relation to changes in lipids). Ann. Chir. et Gynaec. Fenniae (Helsinki) (suppl. 1) *37*, 1–60 (1948). (QCIM *44*, p. 204).
Tripodo, C. and G. de Buono. Hemopoietic function and irradiation of the pyloric duodenal tract. Pt. I. Behavior of erythropenia and of digestion. Leucocytosis in normal persons subjected to irradiation of the pyloric duodenal tract. Haematologica *31*, 693–710 (1948). (NSA *4*, #3596).
Warren, S. Nagasaki survivors as seen in 1947. Military *Surgeon 102*, 98 (1948). (NSA *1*, #410).

1947

Dickie, A. and L. H. Hempelmann. Morphologic changes in lymphocytes of persons exposed to ionizing radiation. J. Lab. Clin. Med. *32*, 1045–1059 (1947).
Feller, A. Influence of isolated cell nuclei on blood picture, with special consideration of leukopenia following roentgen and radium irradiation. Lekarske Listy *2*, 97; 113 (1947). (QCIM *41*, p. 933).
Halberstaedter, L. and J. Liebowitz. Haemolysis and fixation of erthyrocytes by x-rays. Biochem. J. (London) *41*, 235–9 (1947).
Jacobson, L. O. and E. K. Marks. The hematological effects of ionizing radiations in the tolerance range. Radiology *49*, 286–298 (1947).
March, H. C. Leukemia in radiologists. J. Am. Med. Assoc. *135*, 179 (1947). In Med. Library.
Marks, E. K. Effects of radiation exposure on the cells of the peripheral blood. Argonne Natl. Lab. 1947. 3p. MDDC 912. (Microcard).
Mossberg, H. The behaviour of the thrombocytes during and after local radiotherapy. Acta Radiol. *29*, 277–8 (1947). Abstract of paper appearing in Acta Radiol. suppl. 67, 1–146 (1947).
Mossberg, H. Radiation thrombocytopenia. Acta Radiol. *28*, 110–114 (1947).
Mossberg, H. Thrombocytopenia following local radiotherapy. Acta Radiol. suppl. 67, 1–146 (1947).
Setälä, K. Effects of radiation upon mobile visible lipids in human blood. Ann. Med. Exptl. et Biol. Fenniae (Helsinki) (suppl. 1) *25*, 1–58 (1947). (CA *43*, p. 4311).
Whitby, L. Effects of atomic energy on blood. Prensa med. argent. *34*, 648–650 (1947). (QCIM *42*, p. 162).
Whitby, L. Hematologic effects of radiation. Rev. d'hematol. *2*, 251–9 (1947). (QCIM *43*, p. 1244).

1946

Beck, J. S. P. and W. A. Meissner. Radiation effects of the atomic bomb among the natives of Nagasaki, Kyushu. Am. J. Clin. Pathol. *16*, 586–592 (1946). In Med. Library.

Benkwith, K. B. Retinal hemorrhage as seen in atomic bomb casualty. Am. J. Opthalmol. *29*, 799–800 (1946). (QCIM *40*, p. 127).

Borowskaja, D. Effect of roentgen irradiation on serum content of haemagglutinins in human blood. Nature *158*, 269 (1946).

Craver, L. F. Tolerance to whole-body irradiation of patients with advanced cancer. Argonne Natl. Lab. c. 1946. 14p. MDDC 993.

Fua, C. A constant blood protein curve in radium therapy of uterine carcinoma. Boll. soc. ital. biol. sper. *22*, 171 (1946). (CA *41*, p. 528).

Fukase, M., H. Fujiwara, S. Note, R. Ishigami and T. Yoda. Hematological observations in Tsu City war sufferers. 1946. 4p. NP 309.

Gasser, C. Chronic myelogenous leukemia in children, with special consideration of effects of roentgen irradiation and splenectomy on blood picture and bone marrow; 3 cases. Helvet. Paediat. Acta *1*, 299–344 (1946). (QCIM *39*, p. 171).

Helde, M. Connexion between roentgen ray risks for workers and changes in their blood pictures. Acta Radiol. *27*, 308–315 (1946).

Kaufmann, J. The coagulogram as critical indicator of effect; preliminary report based upon 7 year survey of personnel of Department of Radiology, Royal Victoria Hospital. Am. J. Roentgenol. Radium Therapy *55*, 464–472 (1946).

Maia de Mendonca, J. Blood picture of radiologists. Hospital, O (Rio de Janeiro) *29*, 537–562 (1946). (QCIM *40*, p. 177).

Mossberg, H. Two cases of allergy-like reaction with hemorrhagic diathesis in connection with roentgenotherapy. Acta Radiol. *27*, 400–8 (1946).

Nordenson, N. G. Blood changes in radiologic work. Acta Radiol. *27*, 416–432 (1946).

Paladino, T. Serum calcium after Röntgen inhibition of the hypophysis during pregnancy. Boll. soc. ital. biol. sper. *22*, 299–301 (1946). (CA *41*, p. 510).

Paracchi, P. and N. Paracchi. Determination of vitamin PP (nicotinic acid) in the blood and urine of patients with carcinomas of the female genital tract under treatment by radium and by Röntgen irradiation. Vitaminologica *1946*, 112–122 (CA *41*, p. 5613).

Pascher, F. and B. Kanee. Reactions of hemopoietic system to agents used in treatment of dermatoses; effects of low voltage therapy. Arch. Dermatol. and Syphilol. *53*, 1–5 (1946). (QCIM *39*, p. 861).

Schwartz, S., E. J. Katz, L. M. Porter, L. O. Jacobson and C. J. Watson. Studies of the hemolytic effect of radiation. Univ. Chicago Metallurgical Lab. 1946. 51p. MDDC 1342. (Microcard).

Ulrich, H. Incidence of leukemia in radiologists. New England J. Med. *234*, 45–6 (1946). (QCIM *39*, p. 861).

Warren, S. The pathological effects of an instantaneous dose of radiation. Cancer Research *6*, 449–453 (1946).

Warren, S. and R. H. Draeger. Pattern of injuries produced by atomic bombs at Hiroshima and Nagasaki. Cincinnati J. Med. *27*, 871–5 (1946). (QCIM *41*, p. 139). Also in U. S. Naval Med. Bull. *46*, 1349–1353 (1946). (CA *40*, p. 6339).

1945

Hall, B. E., C. H. Watkins, M. M. Hargraves and H. Z. Giffin. Radioactive phosphorus in the treatment of polycythemia vera; results and hematologic complications. Am. J. Med. Sci. *209*, 712–717 (1945).

Lidman, B. I. and C. Cohn. Effect of radar emanations (stray roentgen radiations) on hemopoietic system. Air Surgeon's Bull. *2*, 448–9 (1945). (QCIM *39*, p. 861).

1944

Halberstaedter, L. Effects of x-rays on erythrocytes irradiated in vitro. Nature *153*, 683 (1944).

Hempelmann, L. H., Jr., E. H. Reinhard, C. V. Moore, O. S. Bierbaum and S. Moore. Hematologic complications of therapy with radioactive phosphorus. J. Lab. Clin. Med. *29*, 1020–1041 (1944). Also in J. Am. Med. Assoc. *124*, 735 (1944).

Henshaw, P. S. and J. W. Hawkins. Leukemia incidence in physicians. J. Natl. Cancer Inst. *4*, 339–346 (1944).

March, H. C. Leukemia in radiologists. Radiology *43*, 275–8 (1944).

Schoen. Aleukemic myelosis following therapeutic roentgen irradiation of skin. Deut. med. Wochschr. *70*, 15 (1944). (Comment on Gerbis' article, see entry under 1943.) Reply by H. Gerbis ibid *70*, 15 (1944). (QCIM *37*, p. 486).

1943

Cantril, S. T., L. Jacobson and J. J. Nickson. The effects of irradiation on the blood and blood forming tissues. Argonne Natl. Lab. 1943. 33p. MDDC 991. (Microcard).

Gerbis, H. Aleukemic myelosis following therapeutic roentgen irradiation of skin. Deut. med. Wochschr. *69*, 540 (1943). (QCIM *35*, p. 520).

Keeser, E. Therapeutic activity of radium emanation. Med. Welt *17*, 817–18 (1943). (CA *40*, p. 2136).

Nuttall, J. R. The effects of occupational exposure to x-rays and radioactive substances. Clin. J. (London) *72*, 181–5 (1943). (CA *40*, p. 763).

1942

Adams, W. Prothrombin level in gynecological illness with special consideration of a combined radium-roentgen irradiation treatment of genital carcinoma. Strahlentherapie *71*, 248–254 (1942).

Borak, J. Radiation effects on blood vessels. Part I. erythema, edema. Part II. inflammation, degeneration, suppression of growth capacity, retrogression, necrosis. Part III. telangiectasis; effects on lymph vessels. Radiology *38*, 481–492 (1942); ibid *38*, 607–617 (1942); ibid *38*, 718–727 (1942).

Dunlap, C. E. Effects of radiation on normal tissues. III. Effects of radiation on the blood and the hemopoietic tissues including the spleen, the thymus, and the lymph nodes. Arch. Pathol. *34*, 562–608 (1942). In Med. Library.

Lacassagne, A. and G. Gricouroff. Action of radiation on tissues. Paris. Mason et Cie. 1941. P. 103–127. (In French).

Loeper, M. and J. Mallarme. Acute Leukosis in patient with chronic benzene intoxication treated with irradiation. Le Sang *15*, 406–7 (1942–43). (QCIM *38*, p. 664).

Low-Beer, B. V. A. and A. DeG. Treadwell. Clinical studies with the aid of radio-phosphorus. V. Early effects of small amounts of radio-phosphorus on blood cell levels, uptake and excretion. J. Lab. Clin. Med. *27*, 1294–1305 (1942).

Novacek, L. Changes in calcium-potassium index of blood and heart muscle following irradiation with gamma rays. Sbornik lekarsky *44*, 274, 277 (1942). (QCIM *40*, p. 815).

Osgood, E. E. Is the action of roentgen rays direct or indirect? An investigation of this question by method of human marrow culture. Am. J. Roentgenol. Radium Therapy *48*, 214–219 (1942).

Osgood, E. E., P. C. Aebersold, L. A. Erf and E. A. Packman. Effects of million volt roentgen rays, 200 kilovolt roentgen rays, radioactive phosphorus, and neutron rays by marrow culture technic. Am. J. Med. Sci. *204*, 372–381 (1942).

Sims, J. L. and M. L. Carns. Post-radiation panmyelophthisis clinically simulating agranulocytosis. Ann. Internal Med. *17*, 1021–8 (1942). In Med. Library.

Warren, S. Blood findings in cyclotron workers. Radiology *39*, 194–9 (1942).

Weiss. Occupational roentgen and radium injuries in German communal hospitals during last 20 years. Strahlentherapie *72*, 307–329 (1942).

1941

Ambrosi, F. Action of x-rays on blood prothrombin content. Boll. soc. ital. biol. sper. *16*, 478–480 (1941). (CA *40*, p. 6106).

Holmes, B. E. Serum cholesterol and irradiation sickness. Brit. Med. J. *1941*, I, 314. (CA *35*, p. 7424). In Med. Library.

Liechti, A. and W. Wilbrandt. Radiation hemolysis. I. Hemolysis by roentgen rays. Strahlentherapie *70*, 541–567 (1941). Pts. II and III not pertinent.

Macht, D. I. Pharmacology of human blood after its exposure to roentgen rays. Am. J. Roentgenol. Radium Therapy *45*, 446–9 (1941).

Roffo, A. H. and A. E. Roffo, Jr. pH of the blood after total applications of high-voltage deep x-ray therapy of 400 to 600 kv. Boll. inst. med. exptl. estud cancer *18*, no. 56, 137–148 (p. 148 in English) (1941). (CA *36*, p. 101).

Woodward, H. Q. and N. L. Higinbotham. Serum- and tissue-phosphatase determinations as an aid in evaluating the radiation therapy of bone tumors. J. Am. Med. Assoc. *116*, 1621–7 (1941). In Med. Library.

1940

Albers, D. The influence of ultraviolet light and x-rays on serum phosphatase. Fundamenta Radiol. (Berlin) *5*, 157–164 (1940). (CA *36*, p. 519).

Mogilnitski, B. N., M. V. Lepska and A. F. Goldberg. Changes in blood chemistry during processes of stimulation and inhibition of reticuloendothelial system under influence of roentgen irradiation. Radiol. med. (nos. 7–8) *5*, 55–9 (1940). (QCIM *29*, p. 179).
Roffo, A. E. Jr. The installation of an apparatus for super-deep teleröntgentherapy with 600,000 volts. Bol. inst. med. exptl. estud cancer *17*, 779–807 (1940). (CA *35*, p. 5139).
Ting, T. P. and R. E. Zirkle. Effects of x-rays on the vapor pressure of blood and its components. J. Cellular Comp. Physiol. *16*, 277–283 (1940).
Ting, T. P. and R. E. Zirkle. The kinetics of the diffusion of salts into and out of x-irradiated erythrocytes. J. Cellular Comp. Physiol. *16*, 197–206 (1940).
Ting, T. P. and R. E. Zirkle. The nature and cause of the hemolysis produced by x-rays. J. Cellular Comp. Physiol. *16*, 189–195 (1940).
Wakabayasi, M. and I. Sawada. Effect of ultrashort waves on potassium and calcium content of blood. Japan. J. Med. Sci. III Biophys. *6*, 104*–6* (1940). (QCIM *30*, p. 166).

1939

Bean, W. B., R. W. Vitler and T. D. Spies. The effect of roentgen rays on the blood codehydrogenase I and II, Ann. Internal Med. *13*, 783–6 (1939). In Med. Library.
Bignami, G. and F. Pizetti. The behavior of coagulation time and erthrocyte resistance after irradiation. Scr. ital. Radiobiol. *6*, 229–244 (1939). (Zentr. ges. Radiol. *31*, p. 549).
Bignami, G. Normal radiobiology (effect of radium and roentgen rays) of blood hematopoietic tissue and organs. Haematologica *20*, 561–616 (1939). (QCIM *26*, p. 1101).
Dell'Acqua, G. · Radiobiological observations. Strahlentherapie *65*, 189–192 (1939).
Dognon, A. and C. Piffault. Effect of various radiations on surface films of proteins. Compt. rend. *208*, 907–8 (1939).
Jovin, J. and N. Gingold. Effects of partial teleroentgentherapy on the hematologic picture. Bull. Acad. Med. Roumanie *4*, 279–283 (1939). (QCIM *26*, p. 709).
Lambert, O., J. Driessens and M. Cornillot. Humoral studies in the course of persistent vomiting secondary to irradiation therapy of cancers. Treatment of these accidents by a chloride-glucose-insulin combination. Bull. assoc. franc. etude cancer *32*, 274–281 (1939). (CA *33*, p. 5908).
Malczynski, S. Genesis of changes of blood cholesterol after irradiation. Nowinky Lek. *51*, 161–174 (1939). (QCIM *26*, p. 216).
Mardersteig, K. Roentgen injury and blood picture. Strahlentherapie *64*, 311–317 (1939).
Plasencia, H. T. Action of prolonged fractional irradiations on chemical balance of blood; general biologic action. Acta Unio intern. contra Cancrum *4*, 333–357 (1939). In Med. Library.
Sanigar, E. B., L. E. Krejci and E. O. Kraemer. Effect of ultraviolet radiation and soft x-rays on sedimentation behavior and light absorption of purified human serum albumin. Biochem. J. (London) *33*, 1–16 (1939).
Segal, G. Histamine of patients treated with x-rays. Compt. rend. soc. biol. *131*, 1079–1080 (1939).
Spindler, H. v. Question of leukemia as result of exposure to roentgen rays. Klin. Wochschr. *18*, 1211–12 (1939).
Weicker, B. and H. Bublitz. Roentgen leukopenia and vitamins (B_2 complex and C). Z. ges. exptl. Med. *105*, 521–531 (1939). (QCIM *26*, p. 1142).

1938

Auvergnat, R., L. Bugnard and A. Legris. The action of x-rays on surface tension of plasma. Acta Unio Intern. contra Cancrum *3*, 304–312 (1938). In Med. Library.
Bosch, H. The effect of roentgen and radium rays and radioactive substances on the formed elements of blood and their place of origin. Habilitationsscrift *1938*, 167p. (Zentr. ges. Radiol. *31*, p. 299).
Brink, C. D. Aplastic anemia following x-ray treatment and pyramidon (aminopyrine) combined. S. African Med. J. *12*, 251–2 (1938). (QCIM *23*, p. 108).
Gandelleni, A. Action of local applications (thoraco-abdominal and hypophysial) of short waves on blood. Ann. di radiol. e fis. med. *12*, 146–154 (1938). (QCIM *34*, p. 199).

Gutzeit, K. Blood iodide. VIII. Influence of x-rays on blood iodides in thyrotoxicosis. Z. Klin. Med. *135*, 166–171 (1938). (CA *33*, p. 9421).
den Hoed, D., B. Levie and M. Straub. Serious injury of the blood in consequence of teleroentgentherapy of the whole body. Acta Radiol. *19*, 151–163 (1938).
Iwatsuru, R. and K. Nanjo. Blood phosphatase content in myeloid leucemia. Japan. J. Med. Sci. VIII, Internal Med., Pediat. Psychiat. *4*, 334–5 (1938). (CA *32*, p. 8552).
Kornblum, K., F. Boerner and S. G. Henderson. The qualitative changes in the formed elements of the blood following therapeutic irradiation. Am. J. Roentgenol. Radium Therapy *39*, 601–612 (1938).
Kornblum, K., F. Boerner and S. G. Henderson. Effects of irradiation on normal blood cells as determined by blood count. Am. J. Roentgenol. Radium Therapy *39*, 235–260 (1938).
Laszlo, D. and W. Fleischmann. The action of rays on cell metabolism. Strahlentherapie *62*, 151–5 (1938).
Liaci, L. Pharmacodynamics and therapeutics with radioactivated water. Biochim. terap. sper. *25*, 213–226 (1938). (CA *32*, p. 7573).
Maingot, G., L. Girard and J. Bousser. Transitory leukocytic attacks followed by lasting leukocytosis and myelogenic leukemia in a radiologist; contribution to pathogenesis and prophylaxis of leukemia in radiologists. Le Sang *12*, 569–582 (1938). (QCIM *24*, p. 750).
Mardersteig, K. Roentgen ray effects on erythropoiesis; experimental studies of reticulocytes following irradiation. Strahlentherapie *61*, 107–129 (1938). (QCIM *23*, p. 1159).
Nekula, J. Effect of deep roentgenotherapy of uterine cancer on blood picture and sedimentation. Wein. klin. Wochschr. *51*, 408–413 (1938). (QCIM *23*, p. 787).
Otuka, M. Influence of irradiation on blood coagulation, platelet count, cholesterol and lecithin content. Mit. med. Ges. Chiba *16*, 40–2 (1938). (Zentr. ges. Radiol. *28*, p. 309).
Popp, L. Effect of roentgen rays on blood coagulation. Romania med. *16*, 98–9 (1938). (QCIM *23*, p. 214).
Segre, M. Changes of coagulation time, bleeding time and morphologic composition of blood after roentgen irradiation of spleen and pituitary body. Arch. sci. med. *65*, 255–302 (1938). (CA *32*, p. 6677).
van Ebbenhorst Tengbergen, J. Roentgen ray effects on blood hematopoietic organs and blood diseases. Nederl. Tijdschr. Geneesk. *82*, 4488–4494 (1938). (QCIM *24*, p. 1099).
Weitz, W. Case of myeloid leukemia in x-ray technician, followed from its inception. Klin. Wochschr. *17*, 1579–1580 (1938).

1937

Arneth, J. Effect of roentgen and radium therapy on qualitative and quantitative thrombocyte picture in chronic lymphatic leukemia; indications and contraindications for use. Strahlentherapie *59*, 238–257 (1937).
Arneth, J. Qualitative and quantitative thrombocyte picture in chronic myeloid-leukemic reaction under influence of roentgenotherapy; indications and contraindications for its use. Strahlentherapie *59*, 104–118 (1937).
Balduini, M. Blood vessel permeability relation to roentgenotherapy in man. Bol. soc. med.-chir., Pavia *51*, 661–680 (1937). (QCIM *22*, p. 232).
Bracci, G. Effect of teleroentgenotherapy on leukocyte count; author's experience. Radiol. med. *24*, 970–3 (1937). (QCIM *23*, p. 787).
Cluzet, J. and P. Ponthus. Action of radioactive emanations on dissolved proteins. Arch. phys. biol. *14*, 5–17 (1937). (CA *32*, p. 1722).
Ducuing, J., P. Marques and O. Miletzky. Total roentgenotherapy in diseases of hematopoietic system; modifications of blood and practical deductions. J. radiol. et electrol. *21*, 250–7 (1937).
Ducuing, J., O. Milesky and P. Marques. Experimental investigation on the modifications of the blood produced by total-body x-irradiation. Le Sang *11*, 483–504 (1937). (QCIM *22*, p. 1112).
Dutrey. Roentgen ray effects on blood of radiologists. Bull. mens. soc. med. mil. franc. *31*, 448–457 (1937). (QCIM *22*, p. 1113).
Engelbreth-Holm, J. Changes in the blood picture of persons exposed to radium and roentgen rays. Bibliot. f. laeger *129*, 59–72 (1937). (QCIM *21*, p 220).
Feller, A. and A. Langer. Evaluation of quantity of rays received by physicians and nurses of radium department and comparison with changed blood picture and protective measures against gamma rays. Acta Radiol. *18*, 547–551 (1937).

Fiessinger, N., M. Gaultier and C. M. Laur. Late purpuric anemia with hypoleukia in workman formerly exposed to roentgen rays. Le Sang *11*, 313–319 (1937). (QCIM *22*, p. 1113).

Gendreau, J. E. and G. Pinsonneault. Anemia due to roentgen rays and radioactive substances (radium); report of 2 new cases with autopsy. Acta Union Intern. contra Cancrum *2*, 227–242 (1937). In Med. Library.

Goudsmit, J. and L. H. Levie. The relation between "aplastic anemia" and the use of water containing emanation. Nederl. Tijdschr. Geneesk. *81*, 1708–16 (1937). (CA *31*, p. 4389).

Laborde, S. and J. Leclercq. Professional illness caused by irradiation and radioactive substances. J. belge Radiol. *26*, 201–253 (1937). (Zentr. ges Radiol. *26*, p. 249).

Makidono, J. Influence of roentgen, ultraviolet and infra-red rays on mineral metabolism of serum. J. Kumamoto Med. Soc. *13*, 1734 (1937). (QCIM *22*, p. 204).

Martzy, I. and K. Hollosi. Blood picture in carcinoma patients before and after radiotherapy. Budapesti orvosi ujsag *35*, 249–252 (1937). (QCIM *22*, p. 217).

Schmitt, F. and Basse. The effect of therapeutic doses of roentgen rays on the electrolyte content of the plasma and erythrocytes in Basedow's disease, leucemia and lung tumors. Strahlentherapie *59*, 119–129 (1937).

Sgalitzer, M. and E. Ungar. Attempt to establish proof of physical fitness of roentgenologists, technicians, etc. Strahlentherapie *58*, 701–9 (1937).

Tanaka, T. Effect of roentgen irradiation of spleen after blockage of reticulo-endothelial system. Tohoku J. Exptl. Med. *31*, 331–356 (1937). (QCIM *22*, p. 216).

Tsukahara, K. Influence of various organ cell constituents previously heated or exposed to x-rays on blood picture and hematopoietic organs. Japan. J. Exptl. Med. *15*, 329–354 (1937). (QCIM *23*, p. 1159).

Weil, P. E. Cryptoleukemia in radiologist; case. Le Sang *11*, 548–550 (1937). (QCIM *22*, p. 757).

Windholz, F. Changes in blood vessels of irradiated tissues. Strahlentherapie *59*, 662–670 (1937).

1936

Aubertin, C. Anemia following total roentgenotherapy with report of case. Presse med. *44*, 1321–3 (1936). (QCIM *20*, p 107).

Brose, H. L. and E. B. Jones. Effect of x-radiation on phosphorus compounds in blood. Nature *138*, 687–8 (1936).

Caizzone, G. Agranulocytosis with anemia and hemorrhagic syndrome; case in person employed in radiologic institution; question of occupational origin. Haematologica *17*, 763–772 (1936). (QCIM *20*, p. 1111).

Delherm and Stuhl. Accidents and results from total teleroentgenotherapy. J. radiol. et electrol. *20*, 170–2 (1936).

Fiessinger, N. and G. Boudin. Hemorrhagic aleukia and agranulocytosis following roentgenotherapy of lymphoid leukemia associated with splenomegaly. Le Sang *10*, 337–388 (1936). (QCIM *19*, p. 98).

Flax, A. Hemorrhagic aleukia following roentgenotherapy; 2 cases. Cluj. med. *17*, 632–6 (1936). (QCIM *20*, p. 1152).

Frontali, G. Increased inorganic phosphorus content of irradiated blood. Z. Kinderheilk, *58*, 188–190 (1936). (QCIM *20*, p. 225).

Goodfellow, D. R. Radiation-induced lymphopenia. Brit. J. Radiol. *9*, 695–711 (1936).

Goodfellow, D. R. Radium and human leucocytes. Acta Radiol. *17*, 1–50 (1936).

Hsu, Y. K., C. P. Chang, C. K. Hsieh and R. S. Lyman. Effect of roentgen rays on the permeability of the barrier between blood and cerebrospinal fluid. Chinese J. Physiol. *10*, 379–390 (1936). (CA *30*, p. 7595).

Levy, M. and L. Gally. Blood cholesterol during radiotherapy of the thyroid. Presse med. *44*, 1147–1151 (1936). (CA *31*, p. 4352).

Löw-Beer, A. and W. Redisch. Roentgen ray effects on capillaries of skin. Strahlentherapie *55*, 85–91 (1936).

Marshak, A. and V. L. Bollman. Absorption of x-rays by sperm and erythrocytes and its relation to susceptibility of tissues to x-rays. Am. J. Cancer *26*, 581–5 (1936). (QCIM *19*, p 1110).

Paulian, D. and I. Bistriceano. Variations in the blood pH of man is influenced by short wave irradiation. Bull. acad. med. *116*, 312–314 (1936). (CA *31*, p. 5824).

Rimbaud, L., G. Anselme-Martin, R. Lafon and R. Castagne. Acute leukemia following radioactive therapy administered without physician's control; case. Arch. soc. sci. med. biol. Montpellier et Languedoc *17*, 118–122 (1936). (QCIM *20*, p. 1112).

Schmitz, H. Effect of 800 kilovolt roentgen ray irradiation on blood counts of cancer patients. (In Medical papers dedicated to H. A. Christian in honor of his 60th birthday. R. T. Monroe, ed. Baltimore. Waverly Press, Inc. 1936. p. 568–580). (QCIM *19*, p. 204).

Schumacher, P. H. and Rusch. The effect of x-rays on the cholesterol content of the blood and serum of carcinoma-free, carcinomatous and sarcomatous women. Strahlentherapie *57*, 532–4 (1936).

Woenckhaus, E. The general action of roentgen rays. IV. Conclusions. Naunyn-Schmiedeberg's Arch. exptl. Pathol. u. Pharmakol. *183*, 294–309 (1936).

1935

Bonanno, A. M. Blood picture changes in uterine cancer after application of vaccine broth and roentgen and radium irradiations. Arch. radiol, *11*, 325–344 (1935). (QCIM *19*, p. 211).

Bosch, H. The question of blood damage in radiologists. Radiol. Rundschau *4*, 154–9 (1935). (Zentr. ges. Radiol. *21*, p. 105).

Brinnitzer, H. N. Blood diseases resulting from irradiation. Strahlentherapie *52*, 699–716 (1935). Review article with extensive bibliography.

Dubinskaya, B. N. and V. S. Zetler. Viscosimetric determination of volume of erythrocytes in anemia due to cancer and changes resulting from radiotherapy. Vrachebnoe Delo *18*, 847–854 (1935). (QCIM *19*, p. 98).

Engelbreth-Holm, J. Morphologic changes of blood in persons exposed by occupation to radium and roentgen rays. Ugeskrift Laeger *97*, 241–6 (1935). (QCIM *17*, p 1097).

Englmann, K. Effect of roentgen irradiation on blood-forming bone marrow in man with special consideration of the effect in relation to intensity of the therapeutic radiation. Fortschr. Gebiete Röntgenstrahlen *52*, Kongressheft 73–5 (1935). (Not in Library).

Goodfellow, D. R. Leucocytic variations in radium workers. Pts. I. and II. Brit. J. Radiol. *8*, 669–683; 752–780 (1935).

Jankovic, A. Effects of x-rays on the chemical constitution of (human) blood. Almanah III Kongr. Slov. Apotekara *1935*, 281–292 (CA *31*, p. 6680).

Marchal, G. and L. Mallet. Total teleroentgentherapy in blood diseases and hematopoietic organs. J. belge Radiol. *24*, 301–349 (1935). (Zentr. ges. Radiol. *21*, p. 233).

Marchal, G., L. Mallet, P. Soulie and C. Grupper. Two cases of hemorrhagic aleukia after roentgentherapy. Le Sang *9*, 766–778 (1935). (QCIM *18*, p. 95).

Matteucci, E. Biologic action of roentgen rays on hematopoietic tissues. Giorn. med. Alto Adige 7, 284–302 (1935). (QCIM *19*, p. 1110).

Murayama, M. Colloid reaction in blood serum after irradiation. J. Orient. Med. (Abstr. Sect.) *23*, 11 (Aug. 1935). (QCIM *18*, p. 1081).

Reding, R. Variations of coagulation time of blood after irradiation. Compt. rend. soc. biol. *119*, 342–4 (1935).

Rogozinski, A. and B. S. Levin. Hemolytic action and roentgen ray dosage. Compt. rend. *201*, 798–9 (1935).

Zunz, E. The action of some hormones on the coagulation of blood. Ann. bull. soc. roy. sci. med. nat. Bruxelles, Bull. *1935*, no. 1–2, 1–25. (CA *30*, p. 8326).

1934

Abe, T. Variations in gaseous content of blood after irradiation of different portions of body with doses of varying amounts. Tohoku J. Exptl. Med. *23*, 394–414 (1934). (QCIM *16*, p. 1080).

Becker, S. The effect of radium emanation treatment upon the alkali reserve of blood. Strahlentherapie *50*, 468–470 (1934).

de Bonis, G. and M. Ribechi. Roentgen ray effects on blood changes; experimental study. Fisiol. e med. (Rome) *5*, 545–562 (1934). (QCIM *19*, p. 1110).

Brochner-Mortensen, K. The effect of x-rays on the blood sugar of man. Strahlentherapie *51*, 675–684 (1934).

Flinn, F. B. Radium salts and emanation. Am. J. Roentgenol. Radium Therapy *31*, 830–6 (1934).

Fuge, K. The effects of therapeutic radium and x-ray treatment on blood cholesterol and the liver. Strahlentherapie *50*, 157–166 (1934).
Herzhaft, S. Effect of roentgen rays on blood cholesterol in cancerous patients. Polska gaz. lek. *13*, 176–8 (1934). (QCIM *15*, p. 204).
Isaacs, R. Relation of cell types in leukemia to sensitivity to radiation. Folia Haematol. *52*, 414–425 (1934). (QCIM *17*, p. 682).
Kornalik, F. Biologic reactions of incorporated rays; cytologic changes in poikilothermal blood cells. Strahlentherapie *49*, 707–11 (1934).
Latkowski, J. Action of short waves on human and animal organism. Polska gaz. lek. *13*, 709–714 (1934). (QCIM *16*, p. 191).
Levin, B. S. and C. Piffault. Roentgen ray effects on hemolysis in vitro. Compt. rend. soc. biol. *116*, 1324–6 (1934).
Schoonover, J. W., E. H. Shields and B. P. Widmann. Alterations in the pH of the blood in cancer following roentgen and gamma irradiation. Am. J. Roentgenol. Radium Therapy *31*, 532–7 (1934).
Sella, M. and P. Taddei. Roentgen ray effects on blood glutathione index. Ann. di radiol. e fis. med. *8*, 188–205 (1934). (QCIM *16*, p. 108).
Ssipowsky, P. W. Direct and indirect action of roentgen rays; experimental studies on hematopoietic system. Beitr. pathol. Anat. u. allgem. Pathol. *94*, 1–19 (1934). (QCIM *16*, p. 1081).
Zanetti, S. and L. Pinelli. Effects of roentgen rays on glycemia, calcemia and chloremia. Ann. di radiol. e fis. med. *8*, 418–443 (1934). (QCIM *17*, p. 203).

1933

Bingenheimer, E. Effect of roentgentherapy of mongolism on blood cholesterol. Z. ges. exptl. Med. *87*, 592–612 (1933). (QCIM *13*, p. 200).
Bloomfield, J. J. and F. L. Knowles. Health aspects of radium dial painting; occupational environment. J. Ind. Hyg. *15*, 368–382 (1933). (QCIM *14*, p. 1063).
Costa, A. Fatal anemia resulting from roentgenotherapy of severe dermatosis of type of Boeck's sarcoid. Folia Haematol. *50*, 30–40 (1933). (*QCIM 14*, p. 102).
Cristol, P., P. Monnier and J. Bassaget. Effect of radium and roentgenotherapy on blood proteins in cancer. Arch. soc. sci. med. biol. Montpellier et Lnguedoc *14*, 53–8 (1933). (QCIM *13*, p. 209).
Deutsch, L. Roentgen ray effects on catalase content of blood. Strahlentherapie *48*, 114–117 (1933).
Engelbreth-Holm, J. Roentgen ray effects on blood sugar; review of literature. Ugeskrift Laeger *95*, 1315, 1334 (1933). (QCIM *15*, p. 1097).
Follmann, J. Leukemic erythrodermia following roentgenotherapy of chronic lymphatic leukemia. Orvosi Hetilap. *77*, 394–7 (1933). (QCIM *14*, p. 748).
Gremme, A. Effect of roentgen irradiation on blood gases and alkali reserve in cancer patients. Arch. Gynäkol. *152*, 667–678 (1933). (CA *72*, p. 5345).
Hayer, E. Modification of normal and pathologic white blood picture by local irradiation. Z. ges. exptl. Med. *86*, 623–637 (1933). (QCIM *13*, p. 1067).
Jackson, H. Jr. and F. H. L. Taylor. Calcium, potassium and inorganic phosphate content of the serum in cancer patients. Effect of roentgen ray radiation on the level of these substances in the blood serum of cancer patients. Am. J. Cancer *19*, 379–388 (1933). (CA *28*, p. 522).
Jorns, G. The influence of short electrical waves on leucocyte oxidase. Strahlentherapie *46*, 384–8 (1933).
Jura, V. Influence of roentgen rays on blood cholesterol of patients with carcinoma. Z. Krebsforsch. *39*, 374–381 (1933). (CA *27*, p. 5810).
Kamogawa, T. Cause of variations in blood sugar content following irradiation. Nagasaki Igakkai Zassi *11*, 268–9 (1933). (QCIM *13*, p. 1067).
Kamogawa, T. Changes in blood sugar content following irradiation of various endocrine organs. Nagasaki Igakkai Zassi *11*, 222–4 (1933). (QCIM *13*, p. 1067).
Kamogawa, T. Relation of so-called "roentgen-hyperglycemia" to doses, type of rays and part of body irradiated. Nagasaki Igakkai Zassi *11*, 243–4 (1933). (QCIM *13*, p. 1067).
Mader, A. and E. Bingenheimer. Influence of roentgen irradiation of mongolian idiot on the blood cholesterol. Jahrd. Kinderheilk. *138*, 9–20 (1933). (CA *27*, p. 2489).
Malcznski, S. Changes produced in blood cholesterol by irradiation with infraroentgen (grenz) rays and roentgen rays. Polska gaz. lek. *12*, 793–5 (1933). (QCIM *14*, p. 205).

Phillips, G. W. Effect of radiation on blood count. Lancet *2*, 745–7 (1933). In Med. Library.

Pirchan, A. and J. Teisinger. Changes in roentgenologists and radiologists and their assistants. Casopis Lekaru Ceskych *72*, 1175–9 (1933). (QCIM *14*, p. 214).

Schwartz, L., F. L. Knowles, R. H. Britten and L. R. Thompson. Health aspects of radium dial painting; scope and findings. J. Ind. Hyg. *15*, 362–7 (1933). (QCIM *14*, p. 1063).

Wallbach, G. Action of very small quantities of thorium X and of benzene in decreasing count of leukocytes. Z. ges. exptl. Med. *87*, 340–358 (1933). (QCIM *13*, p. 727).

Wallbach, G. Effect of thorium X on human leukocytes; corresponding effect of benzene. Strahlentherapie *46*, 675–696 (1933).

Wallbach, G. Influence of thorium X on stimulated cultures of human leukocytes. Z. ges. exptl. Med. *87*, 382–396 (1933). (QCIM *13*, p. 727).

Wallgren, A. Knowledge of biologic action of gamma rays. Acta Radiol. *14*, 111–120 (1933).

Wilhelmy, E. and N. Yü. The question of the number of leukocytes after irradiation with very soft roentgen rays. Strahlentherapie *47*, 531–9 (1933). (Also in Klin. Wochschr. *12*, 512–513 (1933)).

1932

Aubertin, C. Blood diseases of radiologists due to radium and roentgen radiation. Monde med., Paris *42*, 555–566 (1932). (QCIM *12*, p. 1116).

Aubertin, C. Blood of radiologists. J. belge Radiol. *21*, 148–163 (1932). (Zentr. ges. Radiol. *13*, p. 628).

Bosch, H. Changes in blood picture following deep roentgenotherapy; prognostic value. Strahlentherapie *45*, 503–524 (1932).

Eckel, P. Influence of infra-roentgen (grenz) rays on leukocyte increase and blood picture. Strahlentherapie *45*, 525–537 (1932).

Englmann, K. Biological effect of roentgen and radium irradiation. Z. Hals. usw. Heilk. *31*, Kongr. Ber. 2, Teil, 132–141 and 255–279 (1932). (Zentr. ges. Radiol. *14*, p. 725–6).

Flinn, F. B. Radium poisoning. N. Y. State J. Med. *32*, 446–7 (1932). (QCIM *11*, p. 437).

Goulston, D. Increase in fragility of red blood corpuscles after exposure to radium. Brit. J. Radiol. *5*, 775–9 (1932).

Hayer, E. Observation of the blood picture in roentgen irradiation. Strahlentherapie *44*, 687–714 (1932).

Held, A. Influence of thorotrast (thorium dioxide preparation) injection on hemolysin titer of blood. Klin. Wochschr. *11*, 463–4 (1932). (QCIM *12*, p. 187).

Hirschfeld, H. and I. Moldawsky. Results of examination of blood of radiologists and their assistants for presence of so-called toxic (pathologic) granulations. Klin. Wochschr. *11*, 1919–1920 (1932). (QCIM *13*, p. 728).

Issaacs, R. Maturing effect of roentgen rays on blood-forming cells. Arch. Internal Med. *50*, 836–842 (1932). (QCIM *12*, p. 1157).

Krasso, I. and A. Loeffler. Effect of general infra-roentgen (grenz) irradiation on calcium and potassium content of blood in glaucoma. Arch. Augenheilk. *106*, 255–270 (1932). (QCIM *12*, p. 215).

Laubry, C. and G. Marchal. Professional leukemia in radiologist; case. Le Sang *6*, 780–6 (1932). (QCIM *12*, p. 786).

Mottram, J. C. Some blood examinations of x-ray workers. Brit. J. Radiol, *5*, 156–8 (1932).

Nielsen, J. Chronic occupational ray poisoning; a discussion based on a case of leukemia in a radium worker. Acta Radiol. *13*, 385–394 (1932).

Osgood, E. E. Action of benzol, roentgen-rays and radium on blood and blood-forming organs. Ann. Internal Med. *6*, 771–4 (1932). In Med. Library.

Poumailloux, M. Changes of leukocyte formula in physician using roentgen ray with long wave length. Le Sang *6*, 890–1 (1932). (QCIM *12*, p. 786).

Pullinger, B. D. Causes of cell death in irradiated human tissue. J. Pathol. Bacteriol. *35*, 527–540 (1932).

Rof, J. Effect of thorotrast on blood and blood-forming organs. Folia Haematol. *46*, 420–8 (1932). (QCIM *11*, p. 1127).

Salanitro, M. Blood changes produced by thorotrast. Boll. soc. ital, biol. sper. *7*, 1229–1231 (1932). (CA *27*, p. 1052).

Stewart, W. H. and H. E. Illick. Effect of x-ray on leucocyte count. N. Y. State J. Med. *32*, 9–10 (1932). (QCIM *11*, p. 674).
Zaccaria, A. A. Death of Edorado Dreyfuss from anemia caused by roentgen rays. Atti congr. ital. radiol. med. pt. 2, 185–191 (1932). (QCIM *14*, p. 102).

1931

Aubertin, C. Leukemia in radiologists. Gaz. med. France p. 333–5 (1931). (QCIM *10*, p. 732).
Aubertin, C. Leukocytosis in roentgenologists. Bull. mem. soc. med. hop. Paris *47*, 307–310 (1931). (QCIM *9*, p. 700).
Audo-Gianotti, G. B. and Bruna. Blood bilirubin variations from roentgen irradiation of splenic region. Morgagni *73*, 931–942 (1931). (QCIM *10*, p. 208).
Azzi, E. and G. Laschi. Changes in blood after mild irradiation. Bull. sci. med. *103*, 277–9 (1931). (QCIM *11*, p. 983).
Azzi, E. and G. Laschi. Effects of mild irradiation from a distance on constitution of blood. Riv. radiol. fis. med. *3*, 539–566 (1931). (QCIM *10*, p. 1073).
Cajkovac, S. Effect of radisapon (radioactive soap.). Ceska Dermatol. (suppl.) *12*, 437–450 (1931). (QCIM *10*, p. 220).
Cavalletti, V. Aplastic anemia during leukemia treated with roentgen rays. Policlinico Il (Rome) Sez. prat. *38*, 660–2 (1931). (QCIM *10*, p. 733).
Clemente, G. Effect of irradiation of pituitary gland on coagulation time of blood. Folia Med. (Naples) *17*, 1005–1024 (1931). (QCIM *11*, p. 193).
Cooper, T. V. The effect of gamma ray irradiation upon the red corpuscles. Lancet *1*, 1026 (1931). In Med. Library.
Fiessinger, N., P. Decourt and C. M. Laur. Agranulocytosis from roentgenotherapy; case. Le Sang *5*, 32–9 (1931). (QCIM *10*, p. 1075).
Fürst, W. Roentgen ray injuries to blood. München med. Wochschr. *87*, 519–521 (1931). (QCIM *9*, p. 1018).
Gallavresi, L. Changes in patients exposed to intesive radium therapy. Radiol. med. *18*, 926–958 (1931). (QCIM *10*, p. 219).
Geremia, A. Effects on roentgen rays on blood catalase. Minerva med. *1*, 685 (1931). (QCIM *10*, p. 209).
Gloor, W. and A. Zuppinger. Blood investigations by protracted fractionated irradiation. Strahlentherapie *40*, 438–464 (1931).
Goto, M. Effects of roentgen rays on phagocytosis of leukocytes; clinical and experimental studies. Tohoku J. Exptl. Med. *18*, 54–96 (1931). (QCIM *10*, p. 734).
Heeren, J. Complementary properties "alexin reaction" of blood serum following irradiation Strahlentherapie *42*, 189–198 (1931).
Hegler, C. and W. Griesbach. Case of radiation-aleukia cured through spleenectomy. Röntgen Praxis *3*, 75–9 (1931). (Zentr. ges. Radiol. *10*, p. 339).
Käding K. Influence of roentgen irradiation on alkali reserve in blood. Strahlentherapie *42*, 571–583 (1931).
Kikuchi, S. Biologic action on roentgen rays in decreasing sugar content of blood; experimental study. Tohoku J. Exptl. Med. *17*, 571–8 (1931). (QCIM *10*, p. 1073).
Liverani, E. Effect of roentgen rays on equilibrium of blood sugar and cholesterol. Arch. farmacol. sper. *52*, 53–66 (1931). QCIM *10*, p. 211).
Liverani, E. Lactic acid in the blood of cancer patients treated with x-rays and with radium. Arch. farmacol, sper. *51*, 177–188 (1931). (CA *25*, p. 4928).
Martland, H. S. The occurence of malignancy in radioactive persons . . . Am. J. Cancer *15*, 2435–2516 (1931). In reprint of Martland's articles on radium poisoning.
Milani, E. Three important points in the x-ray treatment of myelogenous leukemia the spontaneous leukocytic crises; the reversal of sensitivity ratio of red and white cells; the survival of irradiated myelocytes. Radiology *17*, 1032–7 (1931).
Mottram, J. C. Interpretation of blood counts in radium and x-ray workers. Lancet *1*, 42–3 (1931). In Med. Library.
Schaal, G., M. A. Gruschetzkaja and E. J. Zwilichowskaja. Biologic action of roentgen rays on potassium and calcium electrolytes in human blood serum. Strahlentherapie *40*, 111–124 (1931).
Schwarzwald, M. Changes in blood picture after total roentgen irradiation of body. Ceska Dermatol. (suppl.) *12*, 537–542 (1931). (QCIM *10*, p. 219).
Sokolovitch, M. Effect of x-ray therapy on partition of phosphorus compounds in blood in disease. Brit. J. Exptl. Pathol. *12*, 147–150 (1931).

Tuma, V. and F. V. Novak. Physicochemical changes in blood after irradiation with roentgen rays. Casopis Lekaru Ceskych *70*, 193–7 (1931). (QCIM *9*, p. 1017).

Viale, G. Effect of irradiation on blood coagulation: role of spleen and suprarenals. Boll. soc. ital. biol. sper. *5*, 185–7 (1931). (QCIM *10*, p. 213).

Willms, E. Effect of roentgen rays on decrease in blood cholesterol. Strahlentherapie *42*, 171–180 (1931).

Woldrich, A. Changes in blood of radium workers in Jaochimethal. Med. Klin. (Munich) *27*, 17–19 (1931). (QCIM *9*, p. 103).

Woodard, H. Q. and H. R. Downes. The effect of radiation on the acidity of the blood. Am. J. Roentgenol. Radium Therapy *25*, 271–5 (1931).

1930

Böhm, A. Blood picture change after infra-roentgen (grenz) irradiation. Strahlentherapie *35*, 592–6 (1930). (QCIM *7*, p. 209).

Capelli, L. Activity of radiations in serum anaphylaxis; effects of radiations on precipitation in serum. Radiol. med. *17*, 1308–1316 (1930). (QCIM *9*, p. 980).

Capelli, L. Anaphylaxis and roentgen rays; effects on alkali reserve in blood. Radiol. med. *17*, 1361–9 (1930). (QCIM *9*, p. 207).

Capelli, L. Radioproteinemia and radio-anaphylaxis after radiotherapy of cancer. Radiol. med. *17*, 1150–6 (1930). (QCIM *8*, p. 222).

Castellino, P. G. Roentgen ray effects on cutaneous reticulo-endothelial system. Arch. radiol. *6*, 681–9 (1930). (QCIM *9*, p. 1017).

Dellepiane, G. D. Roentgen ray effect on granulo-fixation activity of reticulo-endothelial system. Arch. internat. pharmacodynamie *39*, 294–304 (1930). (QCIM *9*, p. 1017).

Dyes, O. Anemia, age and roentgen sensibility. Strahlentherapie *36*, 552–561 (1930).

Fürst, W. Comparative researches on injury to blood with old method of irradiation and with protective metalix tubes. Strahlentherapie *37*, 659–692 (1930).

Goldstein, M. J. and J. P. Neworoschkin. Changes of catalase and lipase in blood of treated subjects. Strahlentherapie *36*, 736–744 (1930). (Also in Z. eksper. biol. med. ser. A no. 38, *14*, 37–42, 1930. QCIM *10*, p. 1073).

Gutzeit. Diseases of physicians, roentgen technicians, etc. caused by roentgen rays and radioactive substances. Klin. Wochschr. *9*, 455–460 (1930). (QCIM *7*, p. 1015).

Hummel, R. Relations between decreased cholesterol and roentgen intoxication. Strahlentherapie *38*, 308–314 (1930).

Isaacs, R. Blood changes in the leukaemias and the lymphomata and their bearing on roentgen therapy. Am. J. Roentgenol. Radium Therapy *24*, 648–656 (1930).

Jagunow, S. Hematologic crises (changes in blood picture) in persons with gynecologic cancer treated with radium. Monatschr. f. Geburtsh. u. Gynäk. *86*, 296–304 (1930). (QCIM *9*, p. 981).

Joos, G. and J. Heeron. The influence of roentgen rays on the lactic acid picture of the blood. Frankfurt. Z. Pathol. *39*, 466–472 (1930). (CA *24*, p. 3558).

Kalman, J. Leucocyte count following radium irradiation of cancers of female genitals. Zentr. Gynäk. *54*, 1357–1366 (1930). (QCIM *8*, p. 723).

Lambin, P. Anemias caused by roentgen rays and radioactive bodies. Rev. belge sci. med. *2*, 623–650 (1930). (QCIM *8*, p. 1016). (Also in Arch. electric med. *38*, 497–511, 1930. QCIM *9*, p. 981).

Leitner, J. Roentgen ray effects on blood sedimentation. Fortschr. Gebiete Röntgenstrahlen *41*, 743–7 (1930). (QCIM *8*, p. 1053).

Mattick, W. and M. Reinhard. Further studies of the effect of radiation on blood cholesterol in malignant disease. J. Cancer Research *14*, 426–433 (1930). (CA *24*, p. 5857).

Mazzanti, C. Modifications of alkali reserve of blood plasma after application of radium and of roentgen rays. Giron. ital. dermatol. e sifilol. *71*, 955–6 (1930). (QCIM *8*, p. 1016).

Nuzzi, P. Effects of irradiation of testicle on cholesterol in blood. Morgagni *72*, 1662–6 (1930). (QCIM *8*, p. 1016).

Rivosh, F. I. and E. R. Novotelnov. The effect of roentgen rays on the inorganic phosphorus content of blood and urine of patients with carcinoma. Voprosy Onkologii *3*, 150–6 (1930). (CA *25*, p. 2163).

Roffo, R. H. and H. Degiorgi. The acid function in the transformation by x-rays of cholesterol in blood serum. Bol. inst. med. exptl. estudio cancer (Buenos Aires) 7, 292–7 (1930). (CA *29*, p. 5871).

Roffo, A. H. and H. Degiorgi. Comparison of modifications produced by roentgen irradiation in carbon dioxide of serum with those produced in Roffo's test both before and after irradiation in same patient. Bol. inst. med. exptl. estudio cancer (Buenos Aires) 7, 775–9 (1930). (QCIM *8*, p. 209).

Siciliano, L. and C. Banci-Buonamici. Roentgen ray effects on bone marrow. Arch. radiol. *6*, 1108–1136 (1930). (QCIM *9*, p. 1017).

Stoenesco, P. and C. T. Nicolau. Electric changes in plasma and red corpuscles. Bull. mem. soc. med. hop. Bucarest *12*, 232–4 (1930). (QCIM *10*, p. 1073).

Turano, L. Roentgen ray effects, modifications in capillaries. Arch. radiol. *6*, 349–363 (1930). (QCIM *8*, p. 1053).

Wegelin, C. Pathologic anatomy of roentgen anemia; cases. Beitr. pathol. u. allgem. Pathol. *84*, 299–316 (1930). (QCIM 7, p. 1055).

1929

Alberti, O. Dr. Roberto Jacotti, another roentgen ray victim. Radiol. med. *16*, 633 (1929). (QCIM *6*, p. 1125).

Brdiczka, G. Biological effect of roentgen rays in tuberculosis. I. Roentgen ray effect on leukocyte curve in pulmonary tuberculosis. Beitr. Klin. Tuberk. 72, 298–311 (1929). (Zentr. ges. Radiol. 7, p. 833). Pt. II not pertinent.

Corradini, G. Effect of diagnostic irradiation on leukocyte count. Riv. di clin. med. *30*, 825–830 (1929). (Zentr. ges. Radiol. *8*, p. 626).

De Tullio, R. Effect of irradiation of the spleen on blood sedimentation. Studium *19*, 110–116 (1929). (QCIM *6*, p. 227).

Fürst. Effects of earlier methods of irradiation and modern metalix tubes on changes in blood picture. Arch. Gynakol. *137*, 990–2 (1929). (QCIM *6*, p. 1124).

Grosglik, A. Leukamid following roentgen irradiation in chronic leukemia. Strahlentherapie *33*, 498–514 (1929).

Holm. Roentgen ray modifications of blood. Ann. med. phys. *22*, 17–20 (1929). (QCIM *5*, p. 1009).

Knott, F. A. and W. L. Watt. Effects of deep x-ray on blood cells; preliminary note on phagocytic test. Brit. Med. J. *1*, 542–3 (1949). In Med. Library.

Lavedan, J. Research on the blood of professional radiologists. Arch. inst. radium Univ. Paris *1*, 477 (1949). Not in library.

Lazzarini, L. Lukemia from use of roentgen rays. Gazz. ospedali *1*, 401–3 (1929). (Zentr. ges. Radiol. 7, p. 525).

Martland, H. S. Occupational poisoning in manufacture of luminous watch dials. . . J. Am. Med. Assoc. *92*, 466, 522 (1929). In Med. Library.

Pfalz, G. J. Evidence that both protein injections and irradiation with low voltage rays increase bactericidal power of blood. Med. Klin. (Münich) *25*, 637–640 (1929). (QCIM *6*, p. 1124).

Pfalz, G. J. Increased bactericidal power of blood following weak irradiation. Arch. Gynäkol. *138*, 93–110 (1929). (Zentr. ges. Radiol. *8*, p. 55).

Roffo, A. H. and H. de Giorgi. Destruction of serum cholesterol in vitro. Strahlentherapie *33*, 385–8 (1929).

Saidl, J. Effect of roentgen rays on quantity of blood cholesterol. Casopis Lekaru Ceskych. *68*, 729–733 (1929). (QCIM *6*, p. 216).

Schönig, A. Effects of irradiation on reticulo-endothelial system; relation to roentgen intoxication. Strahlentherapie *33*, 55–69 (1929).

Schrutz, K. and O. Vychytil. Effects of roentgenotherapy in spastic whooping cough on blood picture. Jahrb. Kinderheilk *123*, 200–219 (1929). (Zentr. ges. Radiol. 7, p. 121).

Serio, F. and S. Sgroi. Transitory polycythemia in x-ray stimulation of bone marrow. Boll. soc. ital. biol. sper. *4* 519 (1929). (Zentr. ges. Radiol. *8*, p. 309).

Wallbach, G. Different sources of variability in action of benzene, roentgen rays and thorium X in reducing number of leukocytes. Z. ges. exptl. Med. *68*, 621–655 (1929). (QCIM 7, p. 722).

Zunz, E. Effect of roentgen rays and radium on blood coagulation. Ann. med. phys. *22*, 1–3 (1929). (QCIM *5*, p. 194).

1928

Burgheim, F. Blood cholesterol after roentgen irradiation. Comment on Hubert's article. Klin. Wochschr. 7, 802 (1929). Reply by Hubert. ibid 7, 802–3 (1928).

Clemente, G. Reduction of number of erythrocytes and increase of leukocytes after roentgen irradiation of pituitary. Folia Med. (Naples) *14*, 463–502; 573–588 (1928). (Zentr. ges. Radiol. *6*, p. 413).

David, O. Roentgen ray effects on capillary action. Arch. Dermatol. Syphilol. *155*, 99–101 (1928). (QCIM *4*, p. 963).
Evans, W. H. and R. E. Roberts. Splenomedullary leukemia in an x-ray worker with discussion of previously reported cases. Lancet *2*, 748–751 (1928). (QCIM *4*, p. 965).
Ginsburg, L. S. and I. S. Shister. Changes in blood caused by roentgenotherapy of fungous growths of head and face. Vestnik Rentgenol. i Radiol. *6*, 321–9 (1928). (QCIM *5*, p. 1009).
Gorini, P. Absence of modifications of blood cholesterol in children after inhalations of irradiated air. Clin. Pediat. *10*, 789–809 (1928). (QCIM *5*, p. 193).
Gourevitch, R. G. Variations in blood calcium during roentgen treatment of diseases of pituitary body. Vestnik Rentgenol. i. Radiol. *6*, 65–70 (1928). (QCIM *4*, p. 173).
Hubert, R. Cholesterol content of blood serum after irradiation with roentgen rays. Klin. Wochschr. 7, 208–211 (1928). Comments on this entered under Burgheim, F.
Lacassagne, A. Modifications of blood due to roentgen rays and radioactive substances. Progr. med. *43*, 1077–1087 (1928). (QCIM *4*, p. 173).
Lacassagne, A. and Gricouroff. Effects of roentgen rays and radon on leukocytes in vitro. Arch. exptl. Zellforsch. *6*, 303–315 (1928). (QCIM *4*, p. 659).
de Laet, M. Professional diseases due to work with radioactive bodies. Ann. med. leg. criminol. et police sci. *8*, 443–452 (1928). (QCIM *4*, p. 931).
Matthews, H. B. and V. P. Mazzola. Biochemical changes in blood; 100 irradiated cases. Am. J. Obstet. Gynecol. *16*, 97–102 (1928). (QCIM *4*, p. 931).
Moldslowsky, J. W. The influence of the profession of radiologists on the erythropoietic function of bone marrow. Folia Haematol. *36*, 145–173 (1928). (Zentr. ges. Radiol. *6*, p. 109).
Novotelnova, E. R. and F. I. Rivosh. Effect of roentgen rays on fluctuations of blood chlorides and sugar in cancer patients. Vestnik. Rentgenol. i. Radiol. *6*, 205–222 (1928). (QCIM *4*, p. 174).
Pellegrino, F. Effect of roentgen rays on blood sugar regulation in malignant tumors. Riv. patol. sper. *3*, 246–8 (1928). (Zentr. ges. Radiol. *5*, p. 765).
Pendergrass, E. P. Effect of primary and secondary roentgen rays on blood; dangers and methods of protection. Dental Cosmos *70*, 637–641 (1928). (QCIM *4*, p. 964).
Protas, L. R. Variations in blood calcium in bronchial asthma and Basedow's disease during roentgen treatment. Vestnik Rentgenol. i Radiol. *6*, 71–8 (1928). (Zentr. ges. Radiol. *5*, p. 611).
Spence, K. C. Effect of irradiation on blood chemistry in tuberculosis. Brit. J. Actinotherapy *3*, 148–9 (1928). (QCIM *4*, p. 174).
Starnotti, C. The changes in blood produced by roentgen therapy. Arch. farmacol. sper. *46*, 49–89 (1928). (CA *23*, p. 892).
Takaizumi, M. Roentgen ray effect on hematopoietic system. Trans. Japan. Pathol. Soc. *18*, 161–5 (1928). (QCIM *6*, p. 1124).

1927

Barbolin, A. Change in morphological contents of blood in myeloid leukemia under influence of roentgenotherapy. Vrach. Gaz. *31*, 771–2 (1927). (QCIM *2*, p. 1087).
Behrens, A. Blood portein changes after roentgen irradiation. Strahlentherapie *26*, 602–6 (1927).
Bond, C. J. Effect of certain lipoids on cellular constituents of blood. Rept. Brit. Assoc. Adv. Sci. *95*, 368 (1927). (QCIM *3*, p. 839).
Breitländer, K. and C. H. Lasch. Roentgen irradiation causes no changes in serum albumins in blood. Klin. Wochschr. *6*, 743 (1927).
Brounton, C. E. Radium and blood cholesterol. Quart. J. Med. *20*, 321–9 (1927). (CA *21*, p. 3231).
Cazzamali, P. Role of reticulo-endothelial system in wound healing and its behavior under radiation stimuli. Clin. chir. *30*, 986–990 (1927). (Zentr. ges. Radiol. *4*, p. 350).
Dellepiane, G. and P. Badino. Roentgen ray effect on nitrogen content of blood. Riv. patol. sper. *2*, 212–213 (1927). Zentr. ges. Radiol. *4*, p. 278).
Gigon and M. Lüdin. Roentgen rays reduce carbon and nitrogen in blood. Schweiz. med. Wochschr. *57*, 221–3 (1927). (QCIM *1*, p. 643).
Isaacs, R. and A. C. Danielian. Maintenance of leukocyte level and changes during iradiation; a study of the white blood corpuscles apearing in the saliva and their relation to those in the blood. Am. J. Med. Sci. *174*, 70–87 (1927).

Jacobs, E. and R. Montojima. Change in cholesterol metabolism due to roentgen irradiation in cancerous patients. Z. Krebsforsch. *26*, 57–71 (1927). (Zentr. ges. Radiol. *5*, p. 93).
Jansson, G. Variations in radiosensibility of leukocytes and myelocytes; according to their age. Acta. Radiol. *8*, 427–461 (1927).
Koch, L. Behavior of chlorine of erythrocytes after roentgen irradiation. Strahlentherapie *25*, 470–5 (1927).
Kotchneva, N. P. Effects of radon upon amount of blood sugar; 2 cases. Ann. roentgenol. et radiol. *2*, 379–387 (1927). (QCIM *3*, p. 167).
Lazarew, N. W. and A. Lazarewa. Changes of functional condition of blood vessels after irradiation of human skin. Strahlentherapie *26*, 347–362 (1927).
Legget, B. Radiological blood changes. Brit J. Acinotherapy *1*, 19–25 (1927). (Zentr. ges. Radiol. *4*, p. 54–5).
Margreth, G. and M. Lambranz. Behavior of blood cholesterol following irradiation of liver. Boll. soc. ital. biol. sper. *2*, 507 (1927). (MCIM *2*, p. 185).
Mattick, W. L. and K. Buchwald. A clinical study of the effect of radiation on blood cholesterol in malignant diseases. J. Cancer Research *11*, 86–94 (1927). (CA *22*, p. 115).
Muramatsu, T. Effect of roentgen rays upon isohaemoagglutination. Japan. Med. World 7, 287–292 (1927). (Zentr. ges. Radiol. *4*, p. 800).
Patti, F. Blood sedimentation before and after radium treatment of cancer of the female genital tract. Riv. ital. ginecol. *6*, 377–387 (1927). (Zentr. Ges. Radiol. *5*, p. 155).
Patti, F. Influence of radium therapy upon blood picture in uterine cancer. Arch. ostet. e ginecol. *14*, 177–185 (1927). (Zentr. ges. Radiol. *4*, p. 229).
Pilger, W. and S. Wigoder. Effect of deep ray therapy on ionisation of serum; preliminary report. Brit. J. Radiol. *32*, 356–8 (1927). (QCIM *2*, p. 182).
Rubin, E. H. and O. Glasser. The effect of roentgen irradiation on the velocity of erythrocyte sedimentation. Am. J. Roentgenol. Radium Therapy *18*, 520–7 (1927). (CA *21*, p. 790).
Rud, E. Examination of blood of patients with uterine cancer during radium therapy. Strahlentherapie *25*, 195–238 (1927).
Schrutz, K. and O. Vychytil. Radiotherapy of spasmodic cough and its influence on blood picture. Casopis Lekaru Ceskych. *66*, 604–6 (1927). (QCIM *2*, p. 191).
Tattoni, A. Blood sugar variations in roentgen therapy of thyroid. Morgagni *69*, 1521–1535 (1927). (QCIM *2*, p. 198).
Tattoni, A. Modifications of blood morphology by roentgen irradiation of thyroid gland. Morgagni *69*, 1321–7 (1927). (QCIM *2*, p. 191).
Valief, I. Effect of thorium X on the sedimentation of red blood corpuscles. Strahlantherapie *26*, 363–378 (1927).
Wichels, P. and A. Behrens. Effect of roentgen rays on blood protein bodies. Z. ges. exptl. Med. *56*, 387–397 (1927). (QCIM *2*, p. 192).
Yunoki, S. Function of spleen in gynecology and obstetrics; comparative study of influence of spleen extirpation or exposure to roentgen rays upon clotting time of blood. Japan. J. Obstet. Gynecol. *10*, 39–41 (1927). (Zentr. ges. Radiol. *5*, p. 458).
Zunz, E. and J. LaBarre. The effect of x-rays of different wave lengths and of gamma rays and radium on coagulation of blood, blood sugar level and the calcium content. Ann. physiocochem. physiol. biol. *3*, 844–873 (1927). (CA *21*, p. 598).

1926

David, O. Effect of roentgen rays on capillaries. Strahlentherapie *23*, 366–8 (1926).
Detre, L. Effect of roentgen rays on polycythemia ending in panmyelophthisis. Med. Klin. (Münich) *22*, 1297–9 (1936). (QCI *12*, p. 579).
Fraenkel, S. Effect upon cell in isolated radiation of blood. Brit. J. Radiol. *31*, 266–7 (1926). (QCI *12*, p. 599).
Gigon, A. and M. Lüdin. Action of roentgen therapy on some constituents of blood. Schweiz. med. Wochschr. *56*, 814–16 (1926). (QCI *12*, p. 599).
Holl, E. Blood picture and prognosis in irradiated cancer of uterus. Arch. Gynäkol. *127*, 708–716 (1926). (Zentr. ges. Radiol. *1*, p. 153).
Isaacs, R. Effect of roentgen ray irradiation on red blood cell production in cancer and leukemia. Am. J. Med. Sci. *171*, 20–37 (1926).
Jacoby, H. The effect of the inhalation of oxygen and radium emanation on the blood sugars. Deut. med. Wochschr. *52*, 1222–3 (1926). (CA *20*, p. 3038).

Kotta, E. and J. Förster. Action of roentgen rays on blood. Strahlentherapie *21*, 644–667 (1926).
Kotzareff, A. Changes of blood and serum after local and intravenous application of radium emanation in cancer. Schweiz. med. Wochschr. *56*, 151–8 (1926). (QCI *11*, p. 74).
Levy-Dorn, M. and F. Burgheim. Influence of roentgen irradiation on cholestrin content of blood of healthy and diseased body. Strahlentherapie *22*, 538–549 (1926).
Margreth, G. and M. Lambranzi. The change in cholesterol content of blood by irradiation of the zone of the suprarenal glands. Boll. soc. ital. biol. sper. *1*, 724–6 (1926). (CA *21*, p. 3083).
Martland, H. S. Histopathology of certain anemias due to radioactivity. Proc. New York Pathol. Soc. *26*, 65–72 (1926). (In Martland's reprints on radium poisoning).
Martland, H. S. Microscopic changes of certain anemias due to radioactivity. Arch. Path. Lab. Med. *2*, 465–472 (1926). (In Martland's reprints on radium poisoning).
Matz, P. B. Effect of x-rays on blood of roentgen ray workers. U. S. Vet. Bur. Med. Bull. *2*, 10–16 (1926). (QCI *11*, p. 455).
von Pannewitz, G. Effect of x-ray on acid-base equilibrium. Strahlentherapie *24*, 327–330 (1926). (Also in Arch. klin. Chir. *143*, 697–709; 1926).
Pohle, E. A. Studies on suspension-stability of human blood. II. Changes of sedimentation rate of erythrocytes in vitro and in vivo after x-ray explosure. Radiology *6*, 55–8 (1926).
Pohle, E. A. Suspension stability of human blood in roentgen therapy. Ann. Clin. Med. *4*, 987–1001 (1926). (Zentr. ges. Radiol. *2*, p. 96).
Reitter, G. S. and H. S. Martland. Leucopenic anemia of the regenerative type due to exposure to radium and mesothorium, report of a case. Am. J. Roentgenol. Radium Therapy *16*, 161–7 (1926). (In Martland's reprints on radium poisoning).
Schmidt, W. Biological effect of roentgen rays. Strahlentherapie *23* 681–6 (1926).
ten Doornkaat Koolman, M. Experimental studies in influencing erythrocytes by roentgen rays. Strahlentherapie *21*, 668–680 (1926).

1925

Brulé, M. and R. Boulin. Pernicious anemia due to radium. Bull. mem. soc. med. hop. Paris *49*, 528–533 (1925). (QCI *10*, p. 781).
Buckman, T. E., G. A. Daland and M. Weld. The blood phosphorus in chronic myelogenous leukemia especially as influenced by roentgen ray therapy. Arch. Internal Med. *35*, 389–401 (1925). (CA *19*, p. 2079).
David, O. Examination of effect of roentgen rays on blood vessels and capillaries. Brit. J. Radiol. *30*, 462–4 (1925). (QCI *11*, p. 455).
Lankhout, J. Aplastic anemia from professional use of roentgen rays. Ned. Tijdschr. Geneesk. *2*, 2789–2790 (1925). (QCI *11*, p. 440).
Martland, H. S., P. Conlon and J. P. Knef. Some unrecognized dangers in the use and handling of radioactive substances. J. Am. Med. Assoc. *58*, 1769–1776 (1925). In Med. Library.
Monttram, J. C. and A. N. Kingsbury. Action of radium and x-rays correlating production of intestinal changes, thrombopoenia and bacterial invasion. Brit. J. Radiol. *30*, 18–25 (1925). (QCI *10*, p. 780).
Pagniez, P., F. Coste and I. Solomon. Effect of x-rays on the acid-base equilibrium of the blood. Compt. rend. soc. biol. *92*, 489–491 (1925).
Pincussen, L. and I. Makrineos. Changes of metabolism with irradiation. I. Changes of the potassium and calcium content of blood with irradiation. Biochem. Z. *161*, 61–6 (1925). (Pt. II not pertinent.)
Pohle, E. A. Studies on suspension-stability of human blood. I. The velocity of the sedimentation of erythrocytes in x-ray therapy cases. Radiology *5*, 206–210 (1925).
Ross, J. M. Aplastic anaemia following treatment with x-rays. Lancet *1*, 867–9 (1925). (QCI *10*, p. 781).
Spurling, R. G. and J. S. Lawrence. Direct effect of irradiation on leucocytes. Am. J. Med. Sci. *169*, 157–160 (1925). (Also in J. Radiol. *6*, 322–324; 1925).
Weil, P. E. Leukemia and pernicious anemia consecutive to occupational injury from radioactive substances. Presse med. *33*, 1297–9 (1925). (QCI *10*, p. 781).
Weil, P. E. and (A.) Lacassagne. Aplastic pernicious anemia due to radium. Bull. mem. soc. med. hop. Paris *49*, 535–6 (1925). (QCI *10*, p. 781).

Weil, P. E. and A. Lacassagne. Pernicious anemia and myeloid leukemia from radioactive substances. Bull. acad. med. (Paris) *93*, 237–241 (1925). (QCI *10*, p. 781).

1924

Amundsen, P. Blood anomalies in radiologists and persons employed in radiological service. Acta Radiol. *3*, 1–7 (1924).

Bernhard, F. Roentgen-ray irradiation of spleen and blood coagulability. Arch. klin. Chir. *130*, 93–109 (1924). (CA *19*, p. 139).

Bock, A. Study of the alteration of the blood picture after irradiation. Strahlentherapie *16*, 775–791 (1924).

Cameron, A. T. and J. C. McMillan. Roentgen ray sickness and chloride retention. Can. Med. Assoc. J. *14*, 679–683 (1924). (CA *18*, p. 3217).

Carman, R. D. and A. Miller. Occupational hazards of the radiologist, with special reference to changes in the blood. Radiology *3*, 408–419 (1924).

Dazzi, A. Morphological changes of the blood following irradiation of the splenic area, with so-called stimulating doses. Radiol. med. *11*, 529–558 (1924). (Reference in article by Isaacs, Am. J. Med. Sci. *171*, 37; 1926).

Desjardins, A. U. and W. J. Marquis. Blood count and blood pressure in high voltage therapy. Radiology *2*, 252–9 (1924). Review article.

Dobrovalskaia-Zavadskaia, N. Action of radium rays on blood vessels. Lyon chir. *21*, 397–427 (1924). (QCI *9*, p. 678).

Fogelberg, H. Changes in the blood, especially in the blood sugar concentration after exposure of the thyroid gland to x-rays. Acta Med. Scand. *1924*, suppl. VII, 68–72. (CA *19*, p. 1449).

Frei, C. and A. Alder. Action of roentgen rays on blood and agglutinin formation. Schweiz. med. Wochschr. *54*, 670–5 (1924). (QCI *9*, p. 701).

Guilbert, R. Petit and M. LeMay. The action of x-rays on certain organic enzymes and cells. Arch. elec. med. *32*, 309–315 (1924). (CA *19*, p. 3114).

den Hoed, D. Reaction of blood to roentgen irradiation. Ned. Tijdschr. Geneesk. *2*, 1707–14 (1924). (QCI *9*, p. 701).

Konrich, F. and E. Scheller. Effect of x-rays on cholesterol content, hydrogen-ion concentration, freezing-point depression and surface tension of blood. Strahlentherapie *18*, 263–282 (1924).

Kroetz, C. Biochemistry of irradiation. I. Influence of ultra-violet light and x-rays upon the actual blood reaction and upon the stimulation of the respiratory center. II. Influence of ultra-violet light and roentgen rays upon the water, salt and protein content of serum. Biochem. Z. *151*, 146–166; 449–466 (1924).

Lacassagne, A. and J. Lavedan. Blood count under experimental irradiation. Medecine *5*, 683–8 (1924). (QCI *9*, p. 701).

Lacassagne, A. and J. Lavedan. Modification of blood after irradiation. Paris med. *51*, 97–103 (1924). (QCI *9*, p. 677).

Macht, D. I. and E. C. Hill. The effect of ultraviolet, x-ray and radium radiation on the toxicity of normal blood. J. Gen. Physiol. *6*, 671–6 (1924).

Magat, J. and J. Rother. Roentgen rays and blood catalase. Med. Klin. (Münich) *20*, 715–117 (1924). (QCI *9*, p. 701).

Matoni, H. H. Blood after roentgen irradiation: comment on Kiehne's and Sellheim's articles. Münch. med. Wochschr. *71*, 785–6 (1924). (QCI *9*, p. 701). (Articles by K and S could not be located).

Minot, G. R. and R. G. Spurling. The effect on the blood of irradiation, especially short wave length roentgen-ray therapy. Am. J. Med. Sci. *168*, 215–241 (1924).

Mouquin. Blood changes in deep radiotherapy. Paris med. *2*, 227–9 (1924). (QCI *9*, p. 677).

Pagniez, P., A. Ravina and I. Solomon. Action of roentgen rays in accelerating coagulation of blood. Presse med. *32*, 545–8 (1924). (QCI *9*, p. 124).

Pagniez, P., A. Ravina and I. Solomon. Irradiation of various organs and effect on coagulability of blood. Compt. rend. soc. biol. *90*, 1227–8 (1924).

Rud, E. Blood findings in personnel of roentgen and radium institutes. Ugeskrift Laeger *86*, 438–442 (1924). (QCI *9*, p. 677).

Schmitz, H. Clinical significance of chemical and serum analyses of blood of uterine cancer carriers subjected to measured radiation doses. Am. J. Obstet. Gynecol. 7, 449–462 (1924). (QCI *9*, p. 677).

1923

Cori, C. F. and H. De Niord. Biological reaction of x-rays; influence of x-ray treatment on complement content of blood of cancer patients. Am. J. Roentgenol. Radium Therapy *10*, 830–4 (1923). (QCI *8*, p. 644).
Cori, K. F. and G. W. Pucher. Biological reactions of x-rays; effect of x-rays on rates of specific hemolysis. J. Immunol. *8*, 201–9 (1923).
Faber, K. A fatal case of aplastic pernicious anemia in a radiologist. Acta Radiol. *2*, 110–115 (1923).
Faber, K. Professional roentgen anemia. Ugeskrift Laeger *85*, 8–10 (1923). (QCI *8*, p. 51).
Herzfeld, E. and H. R. Schinz. Examination of blood and serum immediately before and after irradiation. Strahlentherapie *15*, 84–97 (1923).
Hirsch, E. F. and A. J. Petersen. Blood with deep roentgen-ray therapy. J. Am. Med. Assoc. *80*, 1505–7 (1923). In Med. Library.
Klewitz, F. Variation in blood concentration after roentgen rays. Klin. Wochschr. *2*, 171 (1923).
Knipping, H. W. and H. L. Kowitz. The effect of roentgen radiation on the white cells of plasma. Fortschr. Gebiete Röntgenstrahlen *31*, 660–4 (1923–4). Not in library.
Minouflet, G. and P. Schrumpff-Pierron. The influence of x-rays on the blood of healthy and pathological tissues. Arch. elec. med. *31*, 161 (1923). Not in library.
Mottram, J. G. Some effects of exposure to radium on the blood platelets. Proc. Roy. Soc. Med. *16*, (Section of pathol.) 9–13 (1923). (QCI *8*, p. 623).
Mouquin, A. Action of deep roentgen radiation of the blood. Medicine *4*, 473 (1923). (Referenced in article by Minot and Spurling, Am. J. Med. Sci. *168*, 240; 1924).
Pagniez, (P.), (A.) Ravina and (I.) Solomon. Action of roentgen rays on coagulation of blood. J. radiol. et electrol. 7, 153–7 (1923).
Risse. Some blood changes during roentgen and radium action. (Arch. Gynäkol, *120*, 181 (1923). (Referenced in article by Carman and Miller, Radiology *3*, 416; 1924).
Straub, H. and K. Gollwitzer-Meier. Blood gas analysis. XIII. The influence of x-rays on hemoglobin and blood corpuscles. XIV. The effect of radiant energy of short wave length on a heterogeneous system. The discharge potential of colloids. Biochem. Z. *136*, 128–139 (1923); ibid *139*, 302–320 (1923).
von Mikulicz-Radecki, R. Roentgen irradiation and rapidity of sedimentation of blood corpuscles. Strahlentherapie *16*, 223–231 (1923). (Also in Arch. Gynäkol. *120*, 187–193; 1923).
Wilbouchevitch, A. and E. Busser. Effect of radium on the hemolytic and antitryptic properties of serum from cancer patients. Compt. rend. soc. biol. *89*, 61–2 (1923).
Wolmershauser, O. The behavior of blood pressure and leukocytes during roentgen irradiation and their relation to the vegetative nervous system. Strahlentherapie *16*, 235–244 (1923).

1922

Amundsen, P. Examinations of the blood of persons handling radium and roentgen rays. Tids. Norske Laegeforening *42*, 862–870 (1922). (CA *17*, p. 2455).
Aubertin, C. Treatment of rheumatism by thorium X. Action on the white blood cells. Bull. mem. soc. med. hop. Paris *38*, 582–8 (1922). (CA *16*, p. 2554).
Eden, R. The significance of the hemaglutination groups for free transplantation and the change of the agglutination group by medicines, narcosis and roentgen raying. Deut. med. Wochschr. *48*, 85–7 (1922). (CA *17*, p. 817).
Förster, W. Influence of leukocytosis of irradiation of spleen. Münch. med. Wochschr. *69*, 1215–16 (1922). (QCI 7, p. 625).
Girard, M., G. Giraud and L. Pares. Hemoclastic crisis after intensive roentgen-ray exposures. Presse med. *30*, 885–7 (1922). (QCI 7, p. 624).
Golden, R. The alkali reserve in röntgen-ray sickness. Arch. Internal Med. *30*, 629–637 (1922). (CA *17*, p. 821).
Heim, K. Blood changes under roentgen-ray treatment. Arch. Gynäkol. *116*, 291–316 (1922). (QCI *8*, p. 644).
Henkel, M. and H. Gueffroy. Coagulation of blood in deep roentgenotherapy. Zentr. Gynäkol. *46*, 409–413 (1922). (QCI 7, p. 108).
Levin, I. Action of radium and x-rays on blood and blood-forming organs. Am. J. Roentgenol. *9*, 112–116 (1922). (QCI 7, p. 624).

Loeb, L. The effects of roentgen rays and radioactive substances on living cells and tissues. Am. J. Roentgenol. *9*, 497–501 (1922). (QCI 7, p. 443).

Mahnert, A. Influence of x-rays on carbon dioxide capacity of blood. Klin. Wochschr. *1*, 1840 (1922).

Pagniez, P., A. Ravina and I. Solomon. Influence of irradiation of the kidney on the time of coagulation of the blood. Compt. rend. soc. biol. *87*, 349–351 (1922).

Pfahler, G. E. Effects of x-rays and radium on blood and general health of radiologists. Am. J. Roentgenol, *9*, 647–656 (1922). (QCI 7, p. 604).

Schroeder, J. H. Intensive deep roentgen irradiations. J. Am. Med. Assoc. *79*, 1240 (1922). In Med. Library.

Wassertrüdinger, O. Irradiation of spleen in relation to coagulation of blood. Zentr. Chirg. *49*, 734–7 (1922). (QCI 7, p. 109).

Watanabe, M. and Y. Harrada. The influence of roentgen rays on hematopoiesis. Japan. Med. World *2*, 234 (1922). (Referenced in article by Isaacs, Am. J. Med. Sci. *171*, 37; 1926).

1921

Bordier, H. A case of fatal anemia. Bull. mem. soc. radiol. med. *9*, 158–160 (1921). (Referenced in article by Carman and Miller, Radiology *3*, 415; 1924).

Feissly, R. Acceleration of blood clotting by means of roentgen rays. Münch. med. Wochschr. *68*, 1418–19 (1921). (CA *16*, p. 3974).

Giraud, M., G. Giraud and G. Pares. Hemoclastic crisis of penetrating radiation sickness. Presse med. *29*, 746–8 (1921). QCI *6*, p. 259).

Hauenstein, J. Blood and metabolism studies with radium emanations. Münch. med. Wochschr. *68*, 809–810 (1921). (CA *17*, p. 3368).

Kaznelson, P. and J. H. Lozant. General increase of body functions following therapeutic roentgen ray exposures. Münch. med. Worchschr. *68*, 132–5 (1921). (CA *15*, p. 3671).

Larkins, F. E. A case of acute aplastic anemia. Lancet *1*, 801 (1921). (QCI *6*, p. 374). Also in Arch. Radiol. Electroth, *25*, 380 (1921).

Leitch, A. Immediate effects of x-ray on blood lymphocytes. Arch. Radiol. Electroth. *26*, 122–8 (1921). (QCI *6*, p. 555).

Masieri, N. Influence of roentgen therapy on the morphological content of blood. Gazz. ospedali *42*, 962 (1921). (Referenced in article by Minot and Spurling, Am. J. Med. Sci. *168*, 239; 1924).

Mottram, J. C. Effect of increased protection from radiation upon blood condition of radium workers. Arch. Radiol. Electroth. *25*, 368 (1921). (QCI *6*, p. 537).

Mottram, J. C. Use of blood counts to indicate efficiency of x-ray and radium protection. Brit. Med. J. *2*, 269 (1921). In Med. Library.

Pagniez, (P.), (A.) Ravina and (I.) Solomon. Action of roentgen rays on the coagulation of blood. Bull. mem. soc. radiol. med. France *9*, 256–8 (1921). (Referenced in article by Carman and Miller, Radiology *3*, 416; 1924).

Russ, S. Discussion on changes induced in blood constituents by radiations. Brit. Med. J. *2*, 268 (1921). In Med. Library.

Russ, S. The immediate effect of x-rays on the blood lymphocytes. Arch. Radiol. Electroth. *26*, 146–9 (1921). (QCI *6*, p. 555).

Russ, S., H. Chambers and G. M. Scott. Further observations of the effect of x-rays upon the lymphocytes. Arch. Radiol. Electroth. *25*, 377–380 (1921). (QCI *6*, p. 554).

Saelhof, C. C. Influence of x-ray organ stimulation on coagulation mechanism. Am. J. Roentgenol. *8*, 179 (1921). (QCI *6*, p. 97).

von der Hütten, F. Coagulation after roentgen irradiation of spleen and liver. Münch. med. Wochschr. *68*, 846 (1921). (CA *17*, p. 578).

Westman, A. Alterations in blood in illnesses treated with x-ray and radium. Acta Radiol. *1*, 349–357 (1921–22).

Wöhlisch, E. The coagulation of blood in cases of splenectomy. Münch. med. Wochschr. *68*, 228–230 (1921). (CA *15*, p. 3673).

Zumpe, R. The alteration in the blood picture and its prognostic value in radiation therapy of cancer. Strahlentherapie *12*, 696–731 (1921).

1920

Mahnert, A. Changes in the protein content of blood serum caused by narcosis, cancer, pregnancy and x-ray. Hoppe-Seyler's Z. physiol. Chem. ***110***, 1–28 (1920).

Mottram, J. C. Red cell blood content of those handling radium for therapeutic purposes. Arch. Radiol. Electroth. *25*, 194–7 (1920). (QCI 5, p. 463).
Mottram, J. C. and J. R. Clarke. Leucocytic blood-content of those handling radium for therapeutic purposes. Proc. Roy. Soc. Med. (Sect. Electro-Therap.) *13*, 25–32 (1920). Also in Arch. Radiol. Electroth. *24*, 345 (1920); Am. J. Roentgenol, 7, 355 (1920). (QCI *5*, p. 462).
Siegel, P. W. The alteration of the blood picture after gynecological roentgen, radium and thorium-X irradiation and its clinical value. Strahlentherapie *11*, 64–139 (1920).
Stephan, R. The problem of the coagulation of the blood (critical remarks on the paper of Szenes, "Raying of glands and coagulation of blood"). Münch. med. Wochschr. *67*, 992 (1920). (CA *15*, p. 395).
Szenes, A. Raying of glands and coagulation of blood. Münch. med. Wochschr. *67*, 786–8 (1920) . (CA *15*, p. 394).
Tichy, H. Acceleration of blood clotting by irradiation of the liver. Zentr. Chirurg. *47*, 1389–1390 (1920). (CA *16*, p. 3947).

1919

Mello, A. DaS. Effect of radioactivity on the blood and the hematopoietic organs. Arch. Brasil. Med. *9*, 123–7 (1919). (CA *14*, p. 1856).

1918

Luden, G. Studies on cholesterol. V. The blood cholesterol in malignant disease and the effect of radium treatment on blood cholesterol. J. Lab. Clin. Med. *4*, 849–862 (1918). Other parts not pertinent.
Millet, J. A. P. and T. Mueller. Some phases of radium action with special reference to the hematopoietic system. J. Cancer Res. *3*, 145 (1918). (Reference in article by Isaacs, Am. J. Med. Sci. *171*, 37 (1926).

1917

Rosenow, G. The resistance of leucocytes to thorium-X. Berl. klin. Wochschr. *54*, 577–8 (1917). (CA *12*, p. 940).

1916

Schweitzer, B. Alteration of blood after thorium X radiation. Münch. med. Wochschr *63*, 341 (1916). (Referenced in Minot and Spurling Am. J. Med. Sci. *168*, 240; 1924).

1915

Nurnberger, L. Clinical blood examination with gynecological deep therapy. Deut. med. Wochschr. *41*, 730 (1915). (Referenced in article by Minot and Spurling, Am. J. Med. Sci. *168*, 240; 1924).
Portis, M. M. Blood changes in workers with roentgen rays, and apparatus for protection. J. Am. Assoc. *65*, 20–1 (1915). In Med. Library.

1914

Cavazzeni and Minelli. The autopsy of a radiologist. Arch. Roentgen-ray *18*, 393 (1914). Abstract. (Referenced in article by Minot and Spurling, Am. J. Med. Sci. *168*, 239; 1924).
Demieville. Variations of blood especially the white corpuscles by radiotherapy. Fortschr. Gebiete Röntgenstrahlen *22*, 465–6 (1914–15). Not in library.
Rosenow, G. The resistance of the different types of leucocytes to thorium X. Z. exptl. Med. *3*, 385–396 (1914). (CA *9*, p. 820).

1913

Cumberbatch, E. P. Fatal leucopenia following x-ray treatment. Arch. Roentgen-ray *18*, 187 (1913–14). (Referenced in article by Carman and Miller, Radiology *3*, p. 415; 1924).

1912

Aubertin, C. Modifications in blood of radiologists. Compt. rend. soc. biol. *72*, 84–6 (1912). (Referenced in article by Minot and Spurling, Am. J. Med. Sci. *168*, 238; 1924).
Aubertin, C. Studies of blood of radiologists. Arch. elec. med. *20*, 150–7 (1912). Not in library.
Brill, O. and L. Zehner. The effect of injection of a solution of radium salt on the blood picture. Berl. klin. Wochschr. *49*, 1261 (1912). (Referenced in article by Minot and Spuring, Am. J. Med. Sci. *168*, 238; 1924).
Chambers, H. and S. Russ. The action of radium emanations on some of the main constituents of normal blood. Proc. Roy. Soc. (London) *84B*, 124–136 (1912).

1911

Pappenheim, A. and J. Plesch. Experimental and histological investigations on the principle of the action of thorium-X on the organs in general and the hematogenic apparatus in particular. Folia Haematol. *14*, 1–12 (1911). (CA 7, p. 380).

Rebattu, Bissand and Richard. Modification of the Arneth neutrophile formula of the blood under influence of inhalation of radium emanation. Compt. rend. soc. biol. *74*, 471–3 (1911). (CA *8*, p. 1464).

1908

Aubertin, C. and E. Beaujard. Action of x-rays on the blood and bone marrow. Arch, med. exptl. anat. pathol. *20*, 273–288 (1908). (Referenced in article by Minot and Spurling, Am. J. Med. Sci. *168*, 238; 1924). Also in Folia Haematol. *6*, 31–41 (1908).

Aubertin, C. and A. Delamarre. Action of radium on blood. Compt. rend. soc. biol. *64*, 437 (1908). (Referenced in article by Minot and Spurling, Am. J. Med Sci. *168*, 239; 1924).

Bergonie, J. and L. Tribondeau. Experimental study of the action of x-rays on the red corpuscles of blood. Compt. rend. soc. biol. *65*, 147 (1908). Not in library.

1907

Salomonsen, C. J. and G. Deeyer. The law of the hemolytic effect of Becquerel rays. Compt. rend. *144*, 999–1001 (1907).

1906

Benjamin, E., V. Reuss, H. E. Sluka and G. Schwartz. Contribution to the question of the influence of roentgen rays on blood. Wien. klin. Wochschr. *19*, 788–794 (1906). (Referenced in article by Minot and Spurling, Am. J. Med. Sci. *168*, 238; 1924).

Klieneberger, C. and H. Zoeppritz. Contribution to the question of the specific formation of leukotoxins in blood serum as a result of roentgen irradiation of leukemia, pseudoleukemia and lymphosarcoma. Münch, med. Wochschr. *53*, 850–4; 911–15 (1906). Not in library.

Milchner, R. and W. Wolff. Remark on the question of leukotoxin formation by means of roentgen irradiation. Berl. klin. Wochschr. *43*, 747–8 (1906). Not in library.

Warthin, A. S. An experimental study of the effects of roentgen rays upon the blood forming organs with special reference to the treatment of leukemia. Intern. Clinics 117th series *4*, 243 (1906). (Referenced in article by Minot and Spurling, Am. J. Med. Sci. *168*, 241; 1924).

1905

Aubertin, C. and E. Beaujard. Action of x-rays on blood and hematopoietic organs. Compt. rend. soc. biol. *58*, 217 (1905). (Referenced in article by Isaacs, Am. J. Med. Sci. *171*, 37; 1926).

Aubertin, C. and E. Beaujard Comparative action of x-rays on blood in myeloid and lymphatic leukemia. Compt. rend. soc. biol. *58*, 177 (1905). Not in library.

Curschmann, H. and O. Gaupp. Proof of roentgen-leukotoxins in blood with lymphatic leukemia. Münch. med. Wochschr. *52*, 2409 (1905). Not in library.

Heinke, H. Experimental investigation of the influence of roentgen rays on bone marrow; beside a comment on the roentgen therapy of leukemia, pseudoleukemia and sarcoma. Deutsch. Z. Chir. *78*, 196 (1905). (Referenced in article by Minot and Spurling, Am. J. Med. Sci. *168*, 239; 1924).

Heineke, H. Experimental investigation of the influence of roentgen rays on the inner organs. Mitt. Grenzg. Med. Chir. *14*, 21 (1905). (Referenced in article by Isaacs, Am. J. Med. Sci. *171*, 36; 1926).

Linser, P. and E. Helber. Effect of roentgen rays on blood. Verhandl. d. Kong. innere Med. Wiesb. *20*, 143–8 (1905). Not in library.

Linser, P. and E. Helber. Experimental investigation of the effect of roentgen rays on the blood and remarks concerning the influence of radium and ultraviolet light. Deut. Arch. klin. Med. *83*, 479–498 (1905). Not in library.

Morris, R. S. Action of roentgen rays on the blood: an experimental study. Am. Med. *10*, 946 (1905). (Referenced in article by Isaacs, Am. J. Med. Sci. *171*, 37; 1936).

1903

Henri, V. and A. Mayer. Action of radiation from radium on hemoglobin, transformation and methemoglobin. Compt. rend. soc. biol. *64*, 1412 (1903). (Reference in article by Minot and Spurling in Am. J. Med. Sci. *168*, 239; 1924). Volume no. for Compt. rend. reference may be in error, believe it should be *54*.

ADDENDA

References without Dates

Fuita, A. Atomic bomb investigation. Keio Univ. 14 p. NP 299.

Gudzent and Hugel. Influence of varying large doses of radium emanation on the blood picture. Radium in Biol. *2*, 202–214; Zentr. Biochem. Biophys. *16*, 121. (CA *8*, p. 1466).

Knowlton, N. P., Jr. The changes in the blood of humans chronically exposed to low level gamma radiation. Los Alamos Sci. Lab. 16p. AECU 1021. On Microcard.

Knowlton, N. P., Jr. The value of blood counts in individuals exposed to ionizing radiations. Los Alamos Sci. Lab. 25p. AECU 397. On Microcard.

Lenser, P. and K. Sick. The relation of uric acid and purine bases in the blood and urine to the action of roentgen rays. Deut. Arch. klin. Med. *89*, 413–431. (CA *1*, p. 1979). (It is possible that the spelling of the first author should be Linser. However, CA spells it Lenser.)

Appendix II

Report of the Subcommittee on Toxicity of Internal Emitters

Austin M. Brues, Argonne National Laboratory, Chairman
Thomas F. Dougherty, University of Utah
Miriam P. Finkel, Argonne National Laboratory
H. L. Friedell, Western Reserve University
Harry A. Kornberg, General Electric Company, Richland, Wash.
Kermit Larson, University of California, Los Angeles
Wright Langham, Los Alamos Scientific Laboratory
Hermann Lisco, Argonne National Laboratory
William P. Norris, Argonne National Laboratory
J. Newell Stannard, University of Rochester
Joseph D. Teresi, Naval Radiological Defense Laboratory
Raymond E. Zirkle, University of Chicago
Consultants:
R. J. Hasterlik, University of Chicago
L. D. Marinelli, Argonne National Laboratory
Jack Schubert, Argonne National Laboratory
Charles L. Dunham, U. S. Atomic Energy Commission

TOXICITY OF INTERNAL EMITTERS

The Subcommittee on Toxicity of Internal Emitters met on January 10 and 11 at Argonne National Laboratory. They discussed the subject from many aspects including a certain amount of classified literature which is not referred to in this report but which it is believed would not alter the conclusions. The agenda were not followed literally in order to promote free discussion, so this report contains a somewhat rearranged version of the discussion.

Statement of the Problem.—The question of safe working exposure levels for industrial and laboratory practice has been intensively studied and is presented in the National Bureau of Standards Handbook 52. (Several members of the Subcommittee agreed that while this represents the best body of data and opinion. it is not the last word, in the matters of use of the biological half-life for all isotopes and the high relative biological effectiveness of alpha rays, in particular. It is thought that the Handbook will probably undergo some changes and that these changes will in general be in the direction of withdrawing a little from the conservatism with which the figures were originally reached.)

Important matters of public concern are: (1) radio-elements (that is, fission or activation products) close to a ground burst of an atomic bomb; (2) more remote fallout; (3) gradual world-wide fallout, especially of strontium-90; (4) consequences of a nuclear reactor accident near a populated center. Other prob-

lems of course exist, but most of the answers will be encompassed in a consideration of these.

The categories of data to be considered include: (1) acute and sub-acute radioelement toxicity and its relation to external radiation damage in an area of heavy contamination; (2) nature and dosage requirements of chronic radiation damage from various radio-elements; (3) physical, ecological, and physiological conditions determining absorption of radio-elements; (4) metabolic handling of radio-elements after their absorption; (5) therapy.

Problems requiring special consideration were: (6) realistic appraisal of absorption from the gut and retention in and absorption from lung; (7) influence of particulate or "hot spot" irradiation, especially in lung and skeleton; and (8) permissible dosage to a large "innocent" population in relation to that to industrially exposed persons for which the present levels are drawn.

The nuclides considered in particular fall in the following groups:

(1) Radium, strontium, barium, calcium; readily absorbed with long-term retention in the skeleton.

(2) Iodine; readily absorbed, of short physical half-life but highly concentrated in the thyroid.

(3) The rare earths, yttrium, and plutonium and other actinides; nearly insoluble and poorly absorbed.

(4) Ruthenium; absorbability ambiguous due to multiplicity of chemical forms.

(5) Cesium; in all probability readily absorbable.

(6) Activation products, various and unpredictable, generally considered unimportant.

Of the above, strontium and iodine received the most consideration. The background of natural radioactivity was also given consideration because of its relation to large population exposure.

Fallout Conditions.—The larger particulates fall out first and smaller ones at a greater distance, for obvious physical reasons. Fission products are plated on the larger, near-in particulates, probably reducing their absorbability. The strontium isotopes, since they exist for a short period after fission as krypton isotopes, escape to some extent from entrapment in larger particles; accordingly, they appear in a smaller ratio than their fission yield in the near fallout and are somewhat enriched at a distance: also, they appear as smaller particles and are more readily absorbed. Rabbits analyzed in Nevada showed a higher skeletal concentration at 133 miles from the test than at greater or less distances.

A detailed study of the effects of the fallout from the thermonuclear explosion of March 1, 1954 on the Marshall Island inhabitants, which is to be published soon (see Cronkite et al. in the references) discusses the body burden from material absorbed into the human body under the conditions of exposure encountered there. Estimates based on excretion rates indicate an initial body burden of Sr^{89} of about the permissible amount, while the I^{131} retained is estimated to have delivered a thyroid dose of the order of 100 rep. Other radioelements (including Ca^{45} and fissile material) are relatively negligible. The opinion of the Subcommittee, that products of neutron activation would be unimportant beside fission product activities, is borne out by this. Data in Table I, from a Japanese publication, are essentially confirmatory, and include autopsy findings which bear out the inferences from measurements on human excreta. It is noted that these low levels of internal contamination existed in persons who received a significant fraction of the human external lethal dose from gamma radiation.

The chemical nature of fallout elements has apparently not been well enough studied to yield many useful inferences about absorbability.

Acute Toxicity.—This would be encountered in pure form only in the event of absorption of specific products. In exposure to large amounts of mixed fission products or by inhalation in a fission cloud, external gamma and beta radiation would be expected to be by far the predominant source of injury.

Acute toxicity from a variety of isotopes generally manifests itself as acute radiation sickness. In combination, bone-seeking and colloidal isotopes act synergistically, due in large part to the fact that spleen and bone marrow are both irradiated in the combined treatment. Feeding of insoluble beta emitters may cause intestinal death, but only after enormous doses, because of the rapidity of their passage through the intestinal tract and because in those places where they remain the longest most of the energy is absorbed by the fecal material which is present there.

Subacute changes depend on the distribution of the element in the body. Hematologic effects are, of course, seen where bone marrow or total body irradiation predominate, and it might be mentioned that strontium-89 or 90, although they produce marked hematologic changes and acute radiation syndrome, produce no or almost no leukemia in mice. The doses required for these subacute changes may be an order of magnitude below those giving acute toxic symptoms. Premature aging, greying of hair, retinal changes, reduced blood volume, and changes in the blood colloids have been observed in dogs given radium and plutonium but not with strontium-90. No explanation of this is available, but these are animals run in parallel experiments. Arterial calcification also occurs in rats after they are given radium in subacute doses, but has not been seen with other nuclides.

Chronic Toxicity: Site of Injury.—It may be stated generally that the more important nuclides will act quite differently depending on the route of administration and their absorbability through the lung or gut, it being generally assumed that these will run parallel.

Materials such as yttrium, the rare earths, and plutonium are very little absorbed by either route, and the predominant hazard will therefore be to the lung. Those with experience in this agree on a general picture of the fate of inhaled, optimal-size particles (those most likely to gain access to the alveoli) in the range of 0.1 to 2.0 microns in diameter. About 25 per cent of this material is exhaled at once; 50 per cent is trapped in the bronchi and is carried up and swallowed within a short time (the half-time being about 9 hours); 25 per cent is deposited in the alveoli and, of this, three-fifths reaches the gut, by way of respiratory passages, leaving a 10 per cent deposition in the lungs. That fraction disappears in cases of inhaled radium sulfate in human cases which have been followed, with a half-time between 100 and 200 days, a very small amount being absorbed.

Absorption of these insoluble materials from the intestine is taken as 0.003 per cent. That was a consensus and obviously may not refer to all possible circumstances. Speaking generally, 10^{-4} is about the highest figure for intestinal absorption which would be found under most conditions. There appears, however, to be an exception in very young animals. Mice before 16 days of age absorb 2 to 3 per cent of plutonium administered either as the citrate by stomach tube or in milk from a plutonium-poisoned animal. From milk it continues to be absorbed in the ratio of 1:300 by young adult mice, although when it is present as the citrate it is not. Parenthetically, one suspects that this reflects an ability of the very young animal to absorb particulate material from the intestinal tract, for example, the milk factor. No data are available on other species so far as is known. Citrate does not promote absorption of plutonium in post-suckling animals but versene does.

Soluble materials are presumed to be absorbed both from alveoli and intestinal tract. Thus the route of entry becomes a matter of indifference. Absorption of specific elements will be discussed later.

As to whether any insoluble materials may be handled by plants in such a way as to promote their absorption, there is no positive information. They are generally not taken up by plants as well as by animals. It is reported, however, that for some reason hickory concentrates yttrium from the soil.

Effects on the Lung.—One microgram of plutonium (or 0.06 microcuries) introduced into the mouse lung as an "optimal" aerosol has proved toxic after a few months resulting in bronchial metaplasia, cellular infiltrations, and fibrosis, and carcinomas appeared after a year. The retained amount at that time has gone down to about one-twentieth of the dose administered, which is about what one would infer from the calculations stated above. Ruthenium administered in a similar way also produces pathologic changes, but data are not sufficient to indicate effective dose comparisons. It may be added that fibrotic changes occur in the human lung after 2,000 r of x-ray and rather regularly after 3,000 r. The Subcommittee on the respiratory tract headed by Dr. Wager has reported in greater detail on this topic.

Ruthenium.—The chloride of ruthenium is absorbed from the rat intestine to the extent of 3 per cent. This value is probably higher than for most of the chemical forms, except of course the tetroxide vapor which, if it were encountered (which is unlikely) would be well absorbed from the lung; this has been shown experimentally.

After absorption of ruthenium the chronic effects would most likely be in the skeleton, based on tracer work and some medium level toxicity work. While

ruthenium was found in animals after the Australian test, it is generally considered to be much less important than strontium.

Cesium.—The gamma ray of the cesium isotope 137 has been seen in normal individuals in the last year but in an amount of radioactivity far below that of body potassium. It was encountered as a deformation in the gamma spectrum of total body potassium and radium. No additional information is available, except that it appears to be somewhat variable and it my be correlated with the intake of milk, which apparently is, relatively speaking, a fairly rich source of radioactive cesium.

Activation Products.—Calcium was mentioned as a component of the Bikini ash, but toxicologically speaking would be a very minor contaminant of strontium. Activation of usual environmental elements would also yield corresponding minor amounts of insoluble material (rare earths and silicon) plus P^{32} and Na^{24}. It was reported that $Zinc^{65}$ was formed in high concentration in muscles of post-test Pacific fish, and it is recognized that other unexpected nuclides might turn up under specialized conditions.

The Alkaline Earths.—Strontium has quitely properly received major attention. It is a bone-seeker like the others of the series, and there is ample experimental evidence of its carcinogenicity in both masses 89 and 90. Since the best available human data regarding absorption and toxicity are from radium cases, this occupies first attention.

Metabolism.—Human radium retention in the age range 20 to 35, based on a 24-year follow-up, is described approximately by a power function of time with an exponent near —0.5; that is, retention varies as the inverse square root of time, being about 50 percent of the injected dose at the end of one day and following this function thereafter.

It may be remarked that indeed the retention of most absorbed materials of whatever nature can best be described by a power function unless one wants to construct a series of exponentials, although it may actually be a series of many exponentials. In all instances that have been carefully studied, even that of the retention of tritium oxide in the form of water, several exponentials exist. The rare earths and actinides show a much less steep slope than the alkaline earths.

There is also a species difference; the dog has a slope of —0.2 to —0.3, depending on age, which makes a great deal of difference in the accumulated dose in this animal.

Integrating this function in man, the cumulative radiation dose, assumed after a very brief period to be limited to the skeleton, goes up as the square root of time, and the rate of loss by excretion relative to the amount retained (specific loss), varies inversely with time. This gives one a way of determining when an exposure took place. It has been pointed out that this function gives a quantitative picture very different from that assuming an exponential half-life (which based on loss by chronic patients, where the loss is very low after 20 years, becomes very long).

It is clear, if this expression is correct, that throughout a human lifetime a man would accumulate little more than 100 days' intake. Observations on adolescent boys indicate that the accumulation to age 17 represents about 40 days' intake.

Evidence now bein accumulated in at least two clinics indicate that strontium-85, which is given because it is less toxic than the beta emitting isotopes, is handled by men in a nearly identical manner to radium. The initial loss varies considerably with age, being least in the younger individuals.

Toxicity.—The best available data on radium toxicity brought to our attention deal with a series of patients that received known amounts of pure radium chloride 24 years ago and were followed. The assumed minimum burden producing serious disease after this period has been taken as 1 microgram, but this has been questioned because of the fact that other preparations were in many cases contaminated with meso-thorium and, perhaps more seriously, with radiothorium. One bone tumor has appeared in one of these patients having 3 micrograms pure radium. The patient with the lowest burden in the series, that is, 0.4 microgram, shows diffuse minimal changes by x-ray, whereas other patients at higher doses from 0.6 to 1.0 microgram do not show detectable changes at this time. It can be assumed that the belief that the lowest effective burden is 1.0 microgram of pure radium is probably not in error by more than a factor of 2 from the standpoint of effects at 20 to 25 years.

Assuming the power function of —0.5 cited above, it can be shown that the total radiation dose accumulated at any time is equal to twice the time multiplied

by the burden. Taking the mass of the skeleton as 7 kilograms, some accumulated dosages are given in Table II.

Strontium 90—Radium Comparison.—Data on late toxicities of strontium and radium in small animals have indicated that a factor of 10 on an energy basis is roughly correct. This would suggest that alpha and beta radiations are relatively equivalent in terms of energy. This may be approximately correct, although the relative biological effectiveness of alpha radiation to x-ray of 10 is presently assumed in calculating permissible doses where experimental data are lacking.

Relative Biological Effectiveness (RBE), Alpha and Beta Rays.—Alpha rays from slow neutron absorption in animals containing boron, have indicated that a factor of 1.4 relative to X rays is roughly correct. Experiments with inhaled radon and injected radon show factors of 1.4 to 1.5 for acute effects, and 3.0 for chronic effects. The RBE for mice seems to pass through a maximum around the ionization density of fast neutrons. Observations on yeast also show a maximum in the intermediate range. The consensus is that a radium to strontium–90 factor of 10 may be a little low but not as low as it was formerly assumed. It was agreed by the Subcommittee to calculate radium and strontium both in roentgen equivalents for the present, although this may give strontium a small additional factor of safety over radium.

Absorption of Strontium.—There are considerable variations in the degree of absorption of strontium depending on the contents of the gastrointestinal tract and the demand for calcium. Absorption of radiostrontium by plants has been investigated, and it appears that the uptake is relatively independent of the concentration of carrier; in other words, it is taken up as a contaminant of the water absorbed. This breaks down only at high levels, where radiation effects on the physiology of the plant enter in. Under fallout conditions, it may also be deposited on leaves. Experiments have indicated that, under a variety of conditions, between one and ten per cent of the strontium–90 in the soil is available to a crop of plants. About five per cent of the strontium taken up by the plants is then retained by animals foraging on them after two to four weeks.

Data on the actual stronium–90 content of biological materials and human beings as a result of past and present fallout have indicated that human bones have now reached an amount equal to one-thousandth of a microcurie (that is, of presently accepted permissible human skeletal content) in young children, declining to approximately zero in persons above 40 years of age. Such calcium-rich sources of the isotope as cheese and milk yield values which are higher in respect to the Sr^{90} : calcium ratio.

Another way of looking at the same question is to consider uptake. When this is done, it appears that the strontium retention in man from the diet is 0.3 to 0.6 times what it would be if strontium were an ideal tracer for calcium.

Radioiodine.—Under existing fallout conditions, cattle in Tennessee have shown up to 10^{-3} $\mu c/gm$ of thyroid, but human thyroids have not shown more than 1/100 of this concentration. External counts on monitors in the Nevada test areas have shown not more than 10^{-4} $\mu c/gm$ of thyroid, with all probabilities in favor of a smaller concentration. In view of the short half-life of I–131 it can be concluded that the hazard of bomb tests from this standpoint is negligible.

It has been suggested that the human thyroid is less radio-sensitive than other tissues, such as bone, since after many years of treatment of Graves' disease with radioactive iodine, no cases of resulting carcinoma have been reported. The customary dosages of I^{131} in such cases yield at least 4000 rep to the gland. On the other hand, carcinoma of the thyroid found in children and young adults has almost invariably been preceded by x-ray treatment to the upper part of the body, in amounts such as to yield as little as 200 r to the infant thyroid. It has been estimated that less than 3% of such treated cases yield carcinoma; nevertheless, the data suggest that 200 r is a potentially carcinogenic dose to the infant thyroid. While the possibility exists that the carcinogenic action may be an indirect, hormonal one, it must still be recognized that this, like leukemia, is an instance of significant carcinogenesis by less than 1000 rep. It seems likely that the infant thyroid is unduly suspectible, but that the adult thyroid is not.

Radiation from Particles and Hot-Spots.—One matter which has caused some concern is the effect of intense radiation of a few cells from particulate sources of radiation.

The only available experimental evidence that bears on this question is an experiment in which the skin was irradiated with beta rays, diffusely over the surface, and by point sources yielding the same amount of radioactivity. It was shown that the point sources were considerably less efficient.

In the case of the skeleton in chronic radium or radiostrontium poisoning, a large part of the dose is from the hot-spots due to concentration of the radioactive material in a few haversian systems that were forming at the time the radioelement was administered. All of the integrated skeletal dosages shown in table 2 (except from X rays, where the bone dose is about four times the air dose given) must therefore be looked at with the consideration that the maximal dose in these hot areas is about ten times as great as the average.

Permissible dosage to Large Populations.—This is a matter on which no complete agreement was reached by the Subcommittee. First responses to this question ranged all the way from the permissible industrial level down to no radiation at all. The uncertainty existing here stems from our ignorance as to whether there is a true threshold for such late effects as malignant tumors, and as to the degree of variation in response to equally exposed individuals.

It was agreed that the only rational approach must take into account the natural radiation background to which the population is exposed. Figures on this, from various sources, are given in table II. It is noteworthy that considerable differences exist from place to place, due mainly to differences in gamma radiation from the environment, and in part to variations in radium content of individuals. Since these existing variations have not given rise to any changes in incidences of tumors or other pathologic states sufficient to attract attention, it was felt that an amount of internal radiation sufficient to double the large population background could certainly be considered safe.

Part B of table II shows integrated skeletal radiation dosages which have given rise to various degrees of pathologic change. Two patients from the Elgin Hospital series are included, since it is known that pure radium was administered and the retention curve has been determined. It is noted that tumors require dosages in the thousands of rep; that observable pathologic changes have required a few hundred; that the natural background (including skeletal radium) varies from 7 to 30 in a lifetime (higher in isolated areas, perhaps); that large populations in high radium areas approach 5 rep in a lifetime from natural skeletal radium alone; while 1/1000 of the permissible strontium burden yields 0.2 rep in a lifetime. Exceeding this latter burden by fifty times would yield 10 rep in 70 years. This would not more than double the usual low skeletal background radiation and leave it well within the range of values; the highest backgrounds encountered in any large areas would be raised about one-third. The general belief of the Subcommittee is that this would produce no perceptible effect.

It is noted that the International Commission on Radiological Protection, using a somewhat more arbitrary procedure, has adopted a figure twice as great for the large population (one tenth of the industrial permissible level) so that there is a large measure of agreement.

Therapy by Removal of Radioelements.—This subject was not discussed by the Subcommittee, but was treated thoroughly at a meeting in October, 1955, the transactions of which will be published by Argonne National Laboratory. A summary of the present status follows:

Clinical and experimental evidence to date shows that there are two effective methods of removing radioelements from the body or prophylactically minimizing their deposition. These are the use of zirconium citrate and of chelating agents, particularly ethylenediamine tetracetic acid (EDTA). Both of these have their optimum effectiveness if given immediately after exposure. The use of the two methods in combination, at least experimentally, appears to be more effective than either one alone.

The chelating agents are mainly effective in removing radioelements from the soft tissues and causing their excretion. Under optimal conditions (that is, large doses administered early) they reduce bone deposition by a factor of two. They are effective on the transuranic elements and rare earth fission products, but for known chemical reasons they are not effective on the alkaline earths such as strontium and radium.

Zirconium citrate appears to be effective as therapy for almost all types of fission products, as is to be expected from the postulated modes of its action. It is particularly useful in minimizing bone deposition of radioelements and if given early, at least in the dog, it has been shown to remove almost all the plutonium from all the tissues.

No method has yet been developed for the removal of significant amounts of strontium. From the chemical standpoint the only promising approach to date appears to be one using agents which pick up the radio element by cation exchange, such as zirconium citrate. Other experimental approaches, particularly involving

dietary and hormone therapy with known influence on skeletal metabolism, are under investigation without clear clinical implications at present.

Treatment of individual situations will necessarily be influenced by consideration of route of entry (e. g. lung or in a wound) and the isotopes involved, bearing in mind that most of the experimental work has dealt with intravenous administration and that clinical experience to date has been severely limited.

TABLE I.—*Data on Fallout (calculated from Tsuzuki, loc. cit.)*

A. ANALYSIS OF "ASH" FROM TEST OF MARCH 1, 1954, MADE ON MARCH 26

Isotope	Half-life	Percent of total activity on March 26		Relative atomic yield	Fission yield
		Measured	As of Mar. 1		
Fission products:					*Percent*
Sr^{89}	53 d.	1.0	1.4	74	4.6
Sr^{90}–Y^{90}	27 y.	0.04	0.04	200	5
Zr^{95}–Nb^{95}	65 d.	8.0	10.5	(500)	6.4
Y^{91}	61 d.	8.0	10.8	660	5.9
$Ru^{103, 106}$; $Te^{129, 132}$; $I^{131, 132}$	----------	15.0	?	?	13.5
Ba^{140}–La^{140}	12 d.	11.0	50	300	6.1
Ce^{141}	33 d.	7.0	12.3	410	6.0
Ce^{144}–Pr^{144}	282 d.	4.0	4.3	610	5.3
Pr^{143}	14 d.	16.0	59	830	6.0
Nd^{147}	11 d.	9.0	46	510	2.6
				(Relative to fission yield)	
Activation Products:					
S^{35}	87 d.	0.05	0.06	5.2	0.05
Ca^{45}	152 d.	0.2	0.22	34	0.3
Other:					
U^{237}	7 d.	20	260	1,820	18
Pu^{239}	24,000 y.	0.0004	0.0004	3,500	35

NOTE: After extrapolating activity back to March 1, relative yield is obtained multiplying by the half-life in days. Where 2 isotopes were measured, half of this value is taken for the parent; an intermediate value was taken for Zr^{95} since the daughter has a 35-day half-life.

B. ANALYSIS OF AUTOPSY MATERIAL 207 DAYS AFTER THE FALLOUT (FIGURES IN $\mu c \times 10^{-3}$ PER KILO WET WEIGHT)

	Liver	Kidney	Lung	Muscle	Bone
Ru and Te	----------	0.9	----------	0.2	2
Corrected for decay (as Ru^{106})	----------	1.3	----------	0.3	3
Zr and Nb	1	1	0.4	0.3	2
Corrected for decay	9	9	3.6	2.7	18
Ce and Pr	2	1	0.5	0.5	20
Corrected for decay	8.4	1.7	0.8	0.8	34
Sr	0.6	0.4	0.1	----------	1
Sr^{89} (if 97%)	9.0	6.0	1.5	----------	15
Sr^{90} (if 3%)	0.27	0.18	0.05	----------	0.45

NOTE.—This indicates that internal radiation was well within permissible limits throughout, amounting to a few mrep/day. In the event the figures for skeletal Ce and Sr were transposed in the report, the Sr^{89} burden appears to be at the permissible level.

TABLE II.—*Background and effective radiation dosages*

A. NATURAL BACKGROUND RADIATION, MREP/YEAR

	Libby	Burch and Spiers	Sievert	Other
Cosmic:				
Sea level	35	16	----------	Lea gives 730, which is probably in error.
5,000 feet	50	----------	----------	
10,000 feet	100	----------	----------	
15,000 feet	170	----------	----------	
20,000 feet	375	----------	----------	
Earth gamma	----------	58	94–296	Sievert gives one value of 520.
Earth, granite	110	----------	----------	
Earth, sedimentary	43	----------	----------	
Over ocean	20	----------	----------	
Body K^{40}	19	18.2	----------	
Body C^{14}	1.5	1.0	----------	
Body radium (dose to skeleton)	[1] 6.7–67	----------	----------	Rochester, N. Y., value 16.

[1] Extremes in Illinois.

NOTE.—This emphasizes the variability in background, even at sea level. In various localities the value might vary from 100 to 420 mr/year; the latter might be taken as the maximum which any large population receives.

B. VARIOUS LEVELS OF SKELETAL IRRADIATION (IN REP)

Patient* with sarcoma (pure radium) at 24 years	6,000
1.0 μc radium retained 24 years after dosage	2,000
Patient* with minimal skeletal changes, 24 years	800
Permissible burden (0.1 μc) sustained 24 years	100
Minimum dose of x-ray reported to induce tumor	1,500
Normal range of background radiation, external and internal, 70 years	7–30
High large-population Illinois radium level, 70 years	4.7
1/1000 permissible Sr^{90} level, 70 years	0.2

NOTE.—patients cited are known to have received pure radium injections. In other reported instances where the thorium chain may have been included in the dose, the radium dose would be calculated as low as 300 rep.

Recommendations.—The Subcommittee made the following suggestions in relation to its study:

Attention should be given to the physiological state of the animal in relation to absorption and toxicity of radioelements, particularly in the case of absorption through the usual routes (lug and gastrointestinal tract). Present information is partial and is practically limited to the alkaline earths and iodine.

Further information on the retention of alkaline earths as a function of age and species and other variables, is needed.

The relation of experimental data on life shortening to the probable picture in man needs clarification, and further verification of the apparent lengthening of life at low doses. These problems are of importance in the isotope toxicity field as well as in relation to external radiations.

The past work on distribution of various radioelements which have not received intensive study should be extended, since unusual radiochemical toxius may be expected to appear occasionally.

Account should be taken of the applicability of the power function to the retention of radioelements, since it would appear that in some cases the present intake levels are much too stringent owing to our past reliance on the half-life concept. Further critical evaluations of the RBE, particularly for alpha radiation, is very desirable for similar reasons. The RBE should be evaluated separately for the several modes of damage.

It is believed that there may be a considerable number of persons who have received radium in the past, who are alive and not seeking medical help. Any means which could be found to obtain an unbiased group of individuals would be extremely desirable, since only in this way can the degree of variability in human response be estimated. For similar reasons, any promising environmental study involving areas of different natural background should be encouraged.

BIBLIOGRAPHY

This Bibliography includes only recent unclassified literature considered by the panel at its meeting or used in tabulations in this report. At the end is a small selection of recent reviews.

Howard L. Andrews, Radioactive Fallout from Bomb Clouds, Science, *122*, 453–456, 1955

J. W. Healy, The Calculation of Maximum Permissible Concentration for Long Lived Isotopes, to be published, 1946

National Bureau of Standards Handbook 52, Maximum Permissible Amounts of Radioisotopes in the Human Body, 1953

Merril Eisenbud and John H. Harley, Radioactive Fallout in the United States, Science, *121*, 677–680, 1955

P. R. J. Burch and F. W. Spiers, Radioactivity of the Human Being, Science, *120*, 719–720, 1954

W. B. Looney, R. J. Hasterlik, A. M. Brues, and E. Skirmont, A. Clinical Investigation of the Chronic Effects of Radium Salts Administered Therapeutically (1915–1931) Am. J. Roentgenol., *73*, 1006–1037, 1955

J. C. Aub, R. D. Evans, L. H. Hempelmann, and H. S. Martland, Late Effects of Internally Deposited Radioactive Materials in Man, Medicine, *31*, 221–329, 1952

William P. Norris, Thomas W. Speckman and Philip F. Gustafson, Studies of the Metabolism of Radium in Man, Am. J. Roentgenol., *73*, 785–802, 1955

M. Tsuzuki, Erfahrungen uber Radioaktive Schadigung der japanische Fischer durch Bikini-asche, Muench. Med. Wochsch. *31*, 988–994, 1955

L. Van Middlesworth, Radioactivity in Animal Thyroids from Various Areas, Nucleonics, *12*, 56–57, 1954 and unpublished data, 1956.

W. F. Libby, Dosages from Natural Radioactivity and Cosmic Rays, Science, *122*, 57–58, 1955

W. F. Libby, The Fallout of Radioactive Strontium, talk at Northwestern University, January 19, 1956

Recommendations of the International Commission on Radiological Protection, Brit. J. Radiol., Suppl. *6*, 1–92, 1955.

E. P. Cronkite, V. P. Bond, C. L. Dunham and S. H. Cohn, The Effects of Ionizing Radiation in Human Beings, to be published, 1956.

R. M. Sievert and B. Hultquist, Variation in Natural Gamma Radiation in Sweden, Acta Radiol. *37*, 388–399, 1952

Reviews:

J. Furth and E. Lorenz, Carcinogenesis by Ionizing Radiations, in Radiation Biology, Ed. by A. Hollaender, Vol. *1*, 1145–1201, McGraw-Hill, 1954

A. M. Brues, Ionizing Radiations and Cancer, Advances in Cancer Research *2*, 177–195, 1954

J. Furth and J. L. Tullis, Carcinogenesis by Radioactive Substances, Cancer Research *16*, 5–21, 1956

J. Schubert, Removal of Radioelements from the Mammalian Body, Ann Rev. Nucl. Sci., *5*, 369–412, 1955

Appendix IV

Report of the Subcommittee on Permanent and Delayed Biological Effects of Ionizing Radiations From External Sources

Henry A. Blair, Department of Radiation Biology, Chairman
George W. Casarett, Department of Radiation Biology
Louis H. Hempelmann, Division of Experimental Radiology
John B. Hursh, Department of Radiation Biology
Marylou Ingram, Department or Radiation Biology
Thomas R. Noonan, Department of Radiation Biology
James K. Scott, Departments of Pharmacology and Pathology
Lawrence W. Tuttle, Department of Radiation Biology

All of the above personnel are members of the faculty of the University of Rochester School of Medicine and Dentistry, Rochester, New York.

PERMANENT AND DELAYED EFFECTS OF IONIZING RADIATIONS FROM EXTERNAL SOURCES

I. INTRODUCTION

While it was recognized soon after their discovery that x-rays and the radiations from radioactive materials could cause acute injury to living tissue it did not become apparent until later that they could also give rise to more subtle permanent and delayed effects which are of prime significance in considering the problem of permissible human exposure.

Laboratory studies of long term effects have been relatively few, partly because their importance was not early appreciated and partly because they are very expensive and time consuming owing to the necessity for maintaining considerable numbers of animals for all, or most, of the their normal life spans. In consequence the data on late effects in animals are meager in certain areas. Such data as there are in man are sufficiently in agreement with those on other mammals to lead to the expectation that extrapolation from lower mammals to man will be possible with fair accuracy.

It is the present purpose to review the long term biologic effects (other than genetic) of radiations from external sources, with emphasis on man, although results of animal experimentation will be drawn upon to illustrate fundamental principles and mechanisms in radiation biology.

II. PERMANENT AND DELAYED EFFECTS OF IONIZING RADIATION IN GENERAL

If animals, which have been irradiated, escape death initially they appear to recover but tend to die prematurely. It is becoming established that in mammals, shortening of life span is a general effect of whole body exposure to ionizing radiation.

There has been a tendency to seek the cause of premature death as a consequence of irradiation in increased incidence of specific disease, especially cancer. While this may be justified in the case of partial body irradiation from external sources or from locally deposited radioactive materials, it probably is not for whole body irradiation. There is evidence that at the median death time populations of irradiated animals have approximately the same incidence of the same diseases as do the controls at their median death time. This is more reasonably interpreted as indicating that irradiation produces primarily the syndrome of premature aging, with concomitant disease, rather than that it, in itself, induces all the diseases of advanced age.

The major permanent and delayed effects of total-body irradiation may be listed categorically in terms of pathologic entities as follows:

A. Increased incidence and/or severity, at given ages, of disease entities to which particular animal species or populations are susceptible. This may occur, and probably usually does, largely as a result of accelerating the time of onset of these diseases and may therefore be considered a part of the acceleration of aging produced by irradiation. It appears that the clinical history of the animals is compressed or telescoped in time in these respects. Included with these disease entities are cancers of various types to which the animals are susceptible, and possibly also cataracts which develop late in a species in a manner similar to the development of cataracts with age in that species.

B. Temporally accelerated involutional, hypoplastic or atrophic, and fibrotic changes in tissues and organs, which changes do not constitute individual disease entities but which are identical in appearance with those occurring during the "normal" course of aging. There is a high degree of correlation between the degree of temporal acceleration of this pathologic picture of accelerated aging and the size of total-body dose or daily dose rate. It should be emphasized, however, that an accurate evaluation of the degree of acceleration of involutional changes produced by certain doses can only be observed in experimental animals at times, after single doses, long after maximum regeneration and repair of the single insult has occurred, or with chronic irradiation, after irradiation has been stopped and recent acute effects on cells have repaired as much as they will.

Although differences exist among species, some of the radiation-accelerated senescent changes in tissues and organs which have been observed are:

(1) Hypoplasia or atrophy of many tissues and organs, especially the thymus, lymph nodes, splenic lymphatic tissue, bone marrow, germinal epithelium, skin, and bone growth centers. Hypoplastic changes in hemopoietic organs are reflected in lowered blood cell counts.

(2) Fibrotic changes which are fairly generalized, but which are commonly noticed in blood vessels generally, in skin where there appears to be increased collagenous tissue and decreased or degenerate elastic tissue, in and/or around lymphatic organs and endocrine organs, and in the myocardium, where focalized fibrosis occurs with age. If the small arteries of the kidney are affected sufficiently, there may develop nephrosclerosis and hypertensive disease.

(3) Pigmentary changes, including greying of hair and increases of pigments in cells of the epidermis, connective tissues, endocrine glands, myocardium and other tissues.

General physiologic changes which are usually associated with aging and which may be accelerated by irradiation include, among others: decreased muscular strength and endurance, lowered recuperative powers and tissue repair capacity and speed, lowered fertility and potency, generalized reduction of elasticity, and increased blood pressure.

Factors included in these first two categories are among those which constitute the general change which may be called "radiation-accelerated aging." The fundamental mechanisms of this change are as incompletely understood as are those of "normal" aging, except that radiations and their known effects are recognized as accelerating agents. The subsequent development of the histologic changes in both radiation-accelerated and "normal" aging appear qualitatively identical. However, it is at present sometimes difficult to distinguish between accelerated aging effects and induced pathologic effects except by arbitrary definition.

C. Induced disease entities or definitive effects, not necessarily specific for irradiation, which rarely occur spontaneously in animals of any age in the species in question, or certain effects and diseases whose know pathogeneses may differ from those occurring in the aging process but which may produce similar final results. In the latter case such effects occur earlier than the specifically comparable effects associated with accelerated aging.

Included in this category are, among other effects:

1. Nephrosclerosis and related hypertension produced essentially by the relatively direct damaging effects of radiation on cells in the kidneys, e. g. components of vessel walls, so that this disease occurs well in advance of any considerable degree of acceleration of other senescent changes. According to some data, renal hypertension once established and progressive, may increase the vascular sclerotic processes in many regions throughout the body and these changes are sometimes associated also with progressive hypoplasia or atrophy of organs in which the vessels become fibrotic.

2. Cataract formation which occurs well in advance of the generalized aging processes if the radiation dose to the lens has been sufficiently high to damage or destroy cells of the anterior epithelium and/or depolymerize the cement substance between fibers. There are two general histologic types of radiation cataract, the purely vacuolar type, which is reversible, and the granular type which is irreversible. The senile cataract, although similar in the ultimate effect to radiation cataracts, is generally more progressive, occurs usually when other aging changes are considerable, and may have a different pathogenesis.

3. Decreased fertility resulting from direct damaging actions of radiation on gametogenic cells and gametes. The mechanisms by which direct actions of radiations decrease fertility are quite different and more numerous, as far as we know, compared with those involved in decrease of fertility due to aging or accelerated aging.

In senescence of the testis, as in testicular damage resulting from infectious and cachectic diseases and physical and chemical damaging agents, the spermatozoa and more mature spermatogenic elements are affected first and most markedly in the seminiferous epithelium, and decreased fertility, if potency is retained, is due to lack of production of sufficient numbers of spermatozoa. In the case of the senescent ovary, there is depletion and/or failure of development of follicles, which is associated with a complicated endocrine disturbance.

Irradiation effects on the gametogenic cells and gametes themselves may decrease fertility in more than one way, prior to and in addition to, the effects involved in acceleration of aging, and these mechanisms, other than those genetic and relating to the fertility of future populations, are as follows:

(a) by reducing the number of gametes produced through effects on primitive radiosensitive precursors of the gametes. This reduction may be partial or complete, or temporary with partial or complete recovery, or permanently complete, depending upon dose. The number of gametes produced may be reduced also indirectly by other diseases which irradiation causes or accelerates.

(b) by damaging in various ways the gametes produced so that they are incapable of fertilization.

(c) by damaging chromosomes of gametes in such ways that they are capable of fertilization but produce zygotes which are incapable of complete and normal development to full-term viable foetuses and die *in utero*. The results of such effects constitute the condition known as "semisterility," which is genetically transmissible to viable offspring.

4. Increase in incidence of other non-neoplastic diseases not common to the species even during the senescent period. There are not sufficient conclusive data in this area of the problem to warrant discussion, but it would appear worthwhile to indicate that research is needed in this aspect of the study of long-term effects of irradiation.

5. Induction of neoplasms rarely, if ever, occurring spontaneously in a species, but caused by the destructive actions of radiation on specific tissues with the initial establishment of precancerous states. It is possible that the later influences which cause the development of neoplasia from these precancerous states may be brought about by the same mechanisms which cause the development or accelerated development of the neoplasms which are of common occurrence in the species with advancing age.

III. PERMANENT AND DELAYED EFFECTS OF IONIZING RADIATION IN PARTICULAR

A. The Shortening of Life Span by Ionizing Radiation

Since there is some evidence that radiation effects depend upon the age of the animal at the time of exposure, some consideration will be given first to the lethal dose as a function of age.

1. *Lethal Dose as a Function of Age.*—The average of median acute lethal dose, LD_{50}–30 days, for young adult mammals is similar for those species which have been studied and lies within or near the range 600 ± 300 roentgens. While it is customary to refer to LD_{50} of a given strain as if it were a specific property, independent of age, this is not justifiable.

In the mouse the susceptibility is maximal at 30 days then decreases rapidly to that seen in young adults. In the rat, which has been studied more extensively, the LD_{50} at three months of age is about double that at three weeks. Beyond three months it diminishes with age and there is some indication that, for the adult animal, the LD_{50} decreases about as the life expectancy. More study of this relation is required to make it wholly quantitative, but it is evident, now, that the susceptibiliy of a whole population is not describable by a single LD_{50}. The published values are usually obtained from the young adult and are therefore maximal or nearly maximal for the strain. In attempts to estimate LD_{50} in man this age dependence should be taken into consideration.

2. *Life Shortening by Single Doses.*—Existing data on rodents subjected to single whole-body doses of radiation are compatible with the view that life is shortened in proportion to the dose for doses less than about two-thirds of LD_{50}. In this range life shortening is about 25% of the adult span per LD_{50}. With greater doses this effect increases more rapidly, attaining about 50% for survivors of LD_{50}. No similar data are available for longer-lived mammals, or for man, but it may be possible eventually to obtain some estimate of the effects of single doses in man from the Japanese survivors.

There are a few data from the rat indicating that shortening of life for a given single dose is about the same, independently of the age at which it is administered to the adult animal, providing the animal is destined to live long enough to make the shortening wholly manifest.

Owing to the difficulty of detecting small changes with limited numbers of animals it cannot be assumed with confidence that life-shortening is proportional to dose down to small doses at the rate given above. Actually there is reason to suppose that the effect of small doses is less than that indicated by the present data. This point will be discussed in considering the effects of chronic irradiation.

3. *Life Shortening by Multiple Doses or Chronic Irradiation.*—Small laboratory animals subjected to irradiation either at high daily rates for short periods or low daily rates for long periods suffer about 7% life shortening per LD_{50} and the effect is proportional to dose, or nearly so, for doses up to about three times LD_{50}. The data for low daily rates and small total doses in the 100 r range are not definitive, there being almost as many showing prolongation, as shortening, of life. While this again is probably due to failure to use sufficient numbers

of animals to measure small effects, it leaves to be resolved the possibility that low daily doses actually prolong life, unlikely as this may seem.

No satisfactory explanation has been offered for the greater shortening of life from single doses than from divided doses of the same total magnitude. Nevertheless the difference appears to be well established for large single doses. However, because divided doses, at rates at least as high as 120 r per day, cause the same effect per accumulated roentgen as divided doses at quite low daily rates, it is difficult to understand why a single dose of 120 r would not act the same. Possibly it does and the apparent disagreement is owing to inaccuracy of single dose data for doses in the 100 r range and below.

4. *The Concept of Irreversible Injury.*—To account for shortening of life as an after effect of irradiation it may be supposed that radiation injury is in part reversible and in part irreversible and that the irreversible component is equivalent to premature aging in the sense that it ultimately deprives the animal of part of its expected life span. This view of irreversible injury does not prescribe whether it is equivalent to abrupt aging at the time of injury or consists in the initiation of aging processes which gradually develop. It is interesting that limited observations in the rat indicate that irreversible injury is measurable, after an interval of presumed complete repair, as a reduction in acute lethal dose. This suggests that aging or its counterpart is laid down at least partially at the time of injury and is potentially observable as some form of persisting lesion, but as yet this phenomenon has not been related to histopathologic changes to be discussed below.

5. *The Effect of Chronic Irradiation in Man.*—The deaths of 82,441 physicians reported in the Journal of the American Medical Association from January 1, 1930, until December 31, 1954, were reviewed by Shields Warren.[1] In physicians grouped according to possible radiation exposure the average age of death was as follows:

	Years
No known contact with radiation	65.7
Some exposure (dermatologists, gastroenterologists, tuberculosis specialists, urologists)	63.3
Radiologists	60.5
U. S. population over 20 years of age	67.1

In comparison with non-exposed physicians the shortening of life of radiologists is 5.2 years or 11% of the adult life span (after 20 years). If extrapolation from the animal data, reviewed above, is permissible, this would be expected to result from chronic whole body exposure to about 1.5 LD_{50} dose or possibly 1000 roentgens. Although this exposure was partial body and possibly less effective, it seems unlikely that the equivalent whole body exposure differed from the above value by a factor greater than 2 or 3. Consequently it appears that, within these limits at least, extrapolation from short-lived animals to man may be made with some confidence on the basis of percent life-shortening per unit dose.

B. Acceleration of Aging by Irradiation

There are no definitive data on the effects of total body irradiation on the aging processes *per se* of man. Perhaps some information on this effect in man will be forthcoming in time as a result of observations which may be made on survivors of the atomic bombing in Hiroshima and Nagasaki. The delays effects which have been observed in these Japanese are reported subsequently under other headings.

In view of the relation between the degree of acceleration of aging observed histologically in experimental animals and the size of the total body dose which the animals received, and in view of the probability that populations of irradiated animals lying prematurely die essentially as the result of premature aging and associated diseases, it seems reasonable to expect that a direct relation may exist in irradiated populations between the amount of life shortening caused by irradiation and the degree to which irradiation has accelerated aging, and that any total-body dose of external radiation which has caused shortening of life in a population of animals has caused a proportional acceleration of aging.

Some of the localized tissue effects produced by partial body irradiation in humans are histologically similar to changes occuring in the syndrome of premature aging. However, many of these changes, some of which are mentioned

[1] Before the Radiation Research Society, May 19, 1956, Chicago, Illinois.

below, are effects induced directly rather than phenomena of accelerated aging.

In the human skin single loses of 500 r to 700 r of x-rays may produce permanent epilation. Somewhat smaller doses causing temporary epilation may cause decreased pigmentation or greying of hair which returns in the irradiated areas. These doses in the erythema dose range or somewhat higher may also cause increased pigmentation of the skin in the irradiated regions, some degree of epidermal atrophy, and some decrease in sebaceous and sweat glands. Hyperkeratotic areas of skin, vascular sclerosis, and dermatitis may also be late sequelae of irradiation of skin. Surface doses of about 1600 r more or less may produce considerable permanent dilatation of capillaries (telangiectasia) in the area irradiated.

Changes in skin are also late effects of chronic irradiation and are seen commonly in the skin of the hands of persons working with radiations. Roentgen dermatitis and the roentgen ulcers which often develop from this condition are considered precancerous conditions because of the frequent development of malignancy in such regions.

Renal hypertension may be produced in man within periods from several months to several years by single localized doses of x-rays of about 3000 to 5000 r or by chronic irradiation, e. g. by a total dose of 2300 r in 35 daily doses to the abdomen, but there are no definitive dose-effect on the late incidence of kidney disease and arteriosclerosis related to generalized radiation acceleration of aging in man. In rats, nephrosclerosis, with renal hypertension, and generalized arteriosclerosis are characteristic delayed effects associated with accelerated aging changes late in their lives following single total-body doses of 500 and 600 r or greater. Much larger doses localized to the kidney are required to cause these renal changes and associated effects to appear early in the life of irradiated animals.

Irradiation of parts of the brain of man with total doses of about 5000 r or more given as a single dose or within 2 or 3 weeks in fairly large fractions may cause progressive sclerosis of blood vessels with subsequent secondary degeneration of brain tissue and sometimes rupture of blood vessels and hemorrhage from one to several years after exposure.

Hypoplastic and atrophic changes, often associated with arterial sclerosis, have been observed in human hemopoietic organs long after localized irradiation. Permanent effects have also been observed in bone heavily irradiated, and in the length of bones irradiated heavily over their epiphyseal ends during the growth period.

The fragmentary data on delayed effects of localized irradiation in human tissues which are similar qualitatively to changes which are associated with aging, are difficult to interpret in terms of accelerated aging, especially since many of the human cases from which the data are obtained suffered from malignancy or other serious disease processes.

C. Late Hematologic Effects of Irradiation

Data collected during the period after World War II may be summarized very briefly as follows:

With respect to occupational or daily exposures over periods of months or years, doses in the range of 0.5 r/day have been reported effective in producing slight depression of the number of circulating lymphocytes and total leukocytes in the peripheral blood of man. A 77 week period of exposure to doses averaging 0.2 r/week effected a decrease in the number of leukocytes in another group of workers. One authority has found some statistical evidence indicating that doses as low as 0.5 r/year may depress the lymphocyte count very slightly. These and similar findings (which are considered in more detail and tabulated in the review cited at the end of this paper) are not unequivocal, of course, and do not indicate with certainty that injury has occurred to the personnel in question, but they indicate in a general way, the presently accepted estimates of the "lowest effective dose."

Slightly larger exposures, for example a total X-ray dose of 40 r given in increments of 15–20 r, or a total dose of 200–300 r received as a series of daily 5–10 r exposures, caused depression of the peripheral blood lymphocyte level in man, the larger cumulative dose being associated with decreased numbers of all types of white blood cells.

Survivors (924) of the Hiroshima atomic bombing, receiving doses estimated at 400 r total-body, and all showing epilation, revealed relative lymphopenia 2 years after exposure. There was greater than normal variability in the blood picture of this group. Four of 5,075 survivors exposed at less than 1,500 meters in the

Nagasaki bombing revealed, after latent periods of 4 to 7 years, refractory (fatal) anemia, with associated leukopenia and thrombocytopenia.

A statistical analysis in 1954 of the hematologic data obtained by the Atomic Bomb Casualty Commission on Hiroshima survivors 5 to 8 years after the bombing indicated that there was no increase in leukopenia, leukocytosis, or anemia in the exposed as compared to the control population. Up to 1953 no cases of aplastic anemia had been found in the survivors in Hiroshima.

Observable changes in the structure of leukocytes (in contrast to the changes in numbers described above) appear to offer considerable promise as sensitive biological indicators of radiation exposure.

Two of the most sensitive morphologic indicators of radiation effect on blood are increased numbers of refractile neutral red bodies in lymphocytes (observed in humans receiving .05 r per day) and an increased incidence of lymphocytes with bilobed nuclei in peripheral blood, which has been observed in a considerable number of cyclotron workers after they had worked about 3½ months during which they received exposures which did not exceed the M. P. E. The incidence of the abnormal lymphocytes returned to normal after extra shielding was installed.

Although there is much conflicting information and opinion relative to late hematologic effects of exposure to ionizing radiation, a review of the available data leads to the formation of certain fairly clear impressions if not definite conclusions.

Early radiologists and radiation workers developed blood pictures characterized by moderate lymphocytosis and leukopenia. These changes were often sufficiently definite to be recognized on an individual basis, as well as by statistical analysis of grouped data. They have been interpreted in various ways, of which the most likely seems to be as follows: initial injury causes a depression of lymphopoiesis, which is followed by a recovery phase characterized by a compensatory increase in activity. During chronic exposure lymphopoiesis presumably "escapes" from the depressing effect and enters the compensatory hypertrophy state. Most of the early workers had exposures which would undoubtedly greatly exceed present maximum permissible doses.

Studies made during the more recent period reveal that the most characteristic changes following chronic exposures in or below the maximum permissible dose range are slight lymphopenia and morphologic alteration of the leukocytes, particularly lymphocytes. The absence of lymphocytosis might be explained by the fact that even chronic exposures are much more intermittent now than earlier. Furthermore, since both injury and the compensatory hypertrophy are, within limits, proportional to the magnitude of exposure, it is possible that present chronic exposures are too slight, as well as too intermittent, to produce adequate stimulus for "escape" into the compensatory hypertrophic phase.

In any event, morphologic changes are probably the most sensitive indication of radiation injury. This is not surprising, for one might expect to find larger numbers of young cells being released into the blood stream following transient bursts of increased leukocytopoiesis. Furthermore, when parent cells have been injured by radiation, abnormalities of mitosis (with the production of abnormal daughter cells) would be expected to occur more frequently than normally. Hence abnormal as well as early cells should appear in the peripheral blood in increased numbers.

The relationship of this type of change to the incidence of leukemia and other latent effects of exposure is very poorly understood, and it is highly desirable that more information be obtained. The compilation of data representing sensitive hematologic indices of radiation exposure in large groups of radiation workers should be vigorously pursued so that eventually there may be enough long-term studies of the health of the workers to permit adequate evaluation of the significance of the more sensitive radiation-induced biologic changes. Should such changes prove to be truly premonitory of an increased incidence of latent effects, it would be important to adapt biologic monitoring procedures accordingly.

One of the greatest hindrances to the present interpretation of highly sensitive hematologic and presumably other biologic changes is the absence of adequate physical monitoring data for exposures below the maximum permissible dose range, so that evaluation of exposures of individual radiation workers is grossly inadequate for the interpretation of hematologic data of the type under discussion. It is important that in selected situations, individual physical monitoring be instituted which is in the same range of sensitivity, with regard to quantitative interpretation, as the biologic monitoring.

D. Carcinogenesis by Radiation from External Sources

This section deals primarily with malignant disease in human populations exposed to ionizing radiation.

It has been clearly established that malignant disease may arise in tissues heavily irradiated by ionizing radiation. Animal experimentation and experience with irradiated human subjects show that almost any tissue can become neoplastic under the proper conditions of exposure. This report does not treat of the type of radiation-induced malignancy which is usually the result of intense, repeated exposure of a small portion of the body, but will consider only malignant disease in populations in which the entire body or at least a large portion of the body of human subjects has been exposed to acute or chronic doses of ionizing radiation from external sources.

Leukemia is commonly associated with exposure of the body to radiation. The close association between exposure and the disease has been described under three different conditions of exposure. The first is that noted in the radiologists who have been chronically exposed. It should be pointed out that the actual number of cases of leukemia is not great even though the incidence is much higher than that noted in the general male population and in other physicians. March, in a review, was able to find only 37 published cases. Furth and Lorenz state that one reason for such a low incidence of leukemia in these persons who were probably heavily exposed in the early days of x-ray technology is that their exposure was partial body rather than total-body. The average age of the 14 radiologists dying of leukemia from 1928–1949 as 58.8 years.

The increased incidence of leukemia in the Japanese exposed to the nuclear explosion in Hiroshima and Nagasaki is the only example of this disease occurring after a single acute exposure of the entire body to ionizing radiation. In this case there is a good correlation between the diseases and the dose. Even in the highest exposure group, however, the incidence is still small (1.25%). Most of the cases are of the myeloid type. However, lymphoid leukemia is known to be comparatively rare in Japan.

There are two examples which illustrate the increased incidence of leukemia under the third condition, i. e., that observed in persons given therapeutic treatment with x-rays to a large portion of their body. One is found in persons with ankylosing spondylitis who have received intensive treatment with x-rays to the entire length of their spine. A total dose of 2,000 r was not unusual in a treatment series and such series were often repeated. There is a good correlation between the incidence of leukemia and the number of treatment courses. The second example is found in children given one or more treatments to their chests in infancy for enlargement of their thymus glands. In one study of children receiving 100 to 1500 r in 1 to 3 treatments to the chest, 7 of 1,722 treated children developed leukemia. This incidence was ten times that expected for children in the state in which the study was done. There were no cases of leukemia in 1,795 untreated siblings at the time of the study. There were 3 cases of leukemia among 604 children receiving less than 200 r, and 4 cases among 804 children receiving more than 200 r. The children in this study showed leukemia incidence of about 0.4%, but they may represent a special situation since the treatment was given when they were very young and since there is no apparent correlation with the x-ray dose.

Aside from leukemia, the only studies on malignant disease in persons exposed to radiation from external sources are found in statistical surveys by Dublin and Spiegelman and by Warren of the cause of death in radiologists and other physicians. Dublin and Spiegelman find that the incidence of malignant disease is lower in physicians than in the general male population of comparable age. It is slightly higher in radiologists but, apparently, not significantly different from that in other specialists or non-specialists who presumably have not received the same exposure to radiation. Interesting is the observation that the highest cancer incidence, twice that in the total group, is found in psychiatrists and neurologists. The data of Shields Warren on a much larger series of physicians show a somewhat higher percentage of cancer deaths in radiologists than in other physicians.

In consideration of animal experiments, uncertainty exists as to whether or not there is a true dose threshold for the production of malignant tumors by irradiation. The answering of this question would require an extremely expensive, massive experimental program employing very large numbers of animals and much time. Experimentation of this kind with low doses is further complicated by the occurrence of spontaneous malignancies to which various

species of animals are susceptible, and also by the variability in response of equally exposed animals. If the fundamental cause, or one of the indispensable factors, in radiation carcinogenesis is the induction of somatic mutations, it would appear possible that radiation carcinogenesis, or perhaps the induction of precancerous states by irradiation, has no dose threshold. However, this reasoning may be applied to other disease states and accelerated aging as well.

In any event the incidence of cancer in exposed populations is not sufficiently great to be regarded as an important contribution to the degree of premature death occurring in a group such as that of the American physicians discussed above.

E. Radiation Cataracts

In this section emphasis is placed upon the effects of the radiations from nuclear disintegrations and high energy particle accelerators on the development of ocular, especially lenticular, lesions in humans. This in no sense deprecates the excellent work done with other animals in which the pathologic development, biochemical changes, and dose-time relationships of lenticular abnormalities have been elucidated. The present reservoir of several thousand persons who have been exposed to the radiations from atomic weapons, and the few hundred humans exposed to the beams of particle generators during the last ten to fifteen years makes it apparent that a reasonably accurate evaluation of the magnitude of the problem of radiation-induction of cataracts in humans can be made from the information currently available.

There are no quantitative or definitive dose-effect data from humans or animals in regard to increased incidence of cataracts late in life as a result of radiation acceleration of aging processes. It is hoped that long-term observation of persons exposed to radiation and of animals in life-span experiments will provide such information.

It is not surprising that a great variety of possible causes of cataracts have been discovered. Histologically the lens is such a simple structure that its possible ultimate response to injury is limited almost exclusively to cataract formation. Considering only idiopathic forms of the disease and excluding those cataracts resulting from injury, including radiation injury, metabolic disease, congenital defects, etc., one is left with a phenomenon definitely related to increasing age.

Despite much effort it is still not yet clear just how the senescent process causes this local change. However, there are differences histologically in the development of this change as compared with the development of radiation-induced cataract. In the aging process the lens grows continuously throughout life but the growth rate becomes slower with advancing age, never reaching zero until the tissue dies in cataract formation. Radiation-induced cataract is the result of direct destructive actions of radiation on the anterior epithelium and possibly on the cement substance between fibers.

It is interesting to note that before it was recognized that radiation cataracts were appearing in cyclotron workers, Evans reported in 1948 that cataract production in mice by fast neutrons relative to x-rays increased significantly with chronic exposure. Young animals exposed during the pre-natal or early post-natal period show markedly greater lenticular radiosensitivity than do older animals.

By December 1948 it was known that at least five nuclear physicists of mean age 31 had incipient cataracts. In January 1949, eleven physicists were examined and ten were found to have cataracts, in three cases severe with definitely impaired vision, in four cases moderate, and in three cases minimal. They were estimated to have received, over periods of 10 to 250 weeks, a median dose of fast neutrons of 50 n, while the range of doses was 10 n to 135 n. At the time the cataractogenic exposures were being received, most of the men were given periodic blood counts, which revealed no change in blood picture warning of overexposure to radiation.

In adult humans exposure to x-rays in excess of about 2000 r has been thought necessary to produce cataracts.

Fillmore, in a survey of the Hiroshima Japanese survivors, based in part upon studies by Kimura in 1949, about 5 years after the detonations, reported 98 cases of cataracts, eighty-five of which were among the 922 survivors 1000 meters or less from the hypocenter. In 1955 Sinskey reported the results of an intensive investigation of 3700 exposed and nonexposed individuals made between May 1951 and December 1953, six to eight years after exposure. There were 154 survivors with posterior subcapsular polychromatic plaques large

enough to be visible with the ophthalmoscope. These radiation-induced pathologic changes in the lens did not in general impair vision significantly when examined, and in most cases were correctable with proper lenses to provide normal vision. Of this group only 25 individuals had vision less than 20/25.

According to Sinskey's study, the human lens is quite sensitive to nuclear radiation in doses which produce epilation and other acute effects but are insignificant with respect to impairment of vision.

Of the approximately 8000 exposed survivors of Hiroshima and Nagasaki who have been examined during the last decade there have been found 10 cases of severe cataract, approximately 25 cases of slightly impaired vision due to posterior polychromatic plaques and perhaps two hundred cases with minimal pathologic lenticular lesions detectable by competent slit lamp examination.

It may be concluded that the atomic bomb explosions over Japan have resulted in negligible loss of vision to date.

F. Effects of Ionizing Radiation on Gametogenesis and Fertility

1. *The Male.*—Spermatogonia are the most radiosensitive cells of the seminiferous epithelium and one of the most sensitive of the body with respect to inhibition of division and with respect to the destructive actions of radiation. Apparently both inhibition of mitosis and destruction of spermatogonia, and differentiation of these cells following irradiation contribute to their disappearance from the seminiferous tubules, the relative contribution of each mechanism varying quantitatively according to size and mode of administration of dose.

The delay in the beginning of regeneration of these cells after irradiation is dependent to a considerable extent upon the dose. Following reduction or depletion of spermatogonia the later germ cell generations undergo maturation-depletion and disappear in the order in which they are formed until a point of maximum hypoplasia is reached. The destruction of some of the cells of more mature generations by relatively high doses may hasten this process. Spermatocytes, spermatids, and spermatozoa are of increasing radioresistance in the order given. The time for the development of maximum hypoplasia of the seminiferous epithelium is about 3 or 4 weeks, sometimes longer, depending upon dose and species, and this time is close to that required for the development of a spermatozoon from a spermatogonium.

Histological sterility, by definition a lack of spermatogenic elements and sperm, may be temporary or permanent and the two often appear very similar upon casual histologic examination. The time factor is of great importance in the prognosis as regards sterility.

For relatively low doses and for certain laboratory animals whose germinal recovery capacity is relatively large, regeneration, if it is to occur, begins before the height of depopulation of germinal epithelium is reached or soon thereafter. With certain higher doses given to such animals, a delay of beginning of regeneration for about 10 months is considered by some to be indicative of permanent sterilization. Actually, in much of the work employing large single doses of radiation the animals were not studied for the maximum time possible or desirable.

It is probable that the critical interval of time for beginning of regeneration varies among species and that for some of the larger animals, including man, whose powers of germinal regeneration are comparatively low, active regeneration may be delayed following severe radiation effects much longer than 10 months. Whether permanency of sterilization or length of the temporary sterile period is due to effects on cells involved directly in spermatogenesis or rather indirectly to effects on supporting tissues is not clear.

The testicular effects of irradiation are qualitatively similar in all mammals studied, including man, but vary quantatively according to differences in testicular radiosensitivity and recovery capacities among species. Whether a dose of radiation sterilizes permanently or temporarily depends at least as much on the natural capacity for regeneration of primitive spermatogenic cells as on the radiosensitivity of the spermatogenic cells existing at the time of irradiation.

Sertoli cells and interstitial (endocrine) cells are relatively radioresistant. Male mammals may be sterilized permanently without prominent histologic changes in the interstitial cells and without decrease in sexual potency or libido.

Following single doses of irradiation and preceding the sterile or subfertile period produced there is a period of fertility, the length of which is much less dependent on low doses than on doses high enough to affect the fertilizing capacity of mature sperm. Lower doses are required to destroy the fertilizing

capacity of sperm than are necessary to affect the viability or motility of sperm present at the time of irradiation and therefore during the initial fertile period.

This first period of continued fertility is due largely to sperm mature at the time of irradiation and possibly to some sperm in the spermatid stage or even a few in the spermatocyte stage, depending upon the dose and the length of the fertile period. The subsequent period of infertility or sterility is due to decreased numbers of sperm produced, and the fertile period following the period of sterility, if recovery occurs, is due to sperm that were developed from cells which were in the spermatognoial stage or were primordial undifferentiated cells at the time of irradiation.

In the initial fertile period litter size is subject to reduction with sufficient dose and the amount of reduction is dependent upon the dose. Litter size in the fertile period after the sterile period is usually normal or perhaps slightly less than normal. Reduction in litter size is explained on the basis of induction in sperm, and perhaps to some extent in precursors of sperm, of chromosomal aberrations which do not interfere with fertilization but which cause death of the zygote or embryo *in utero*. In terms of human considerations the equivalent result would be manifest in the form of increased incidence of spontaneous abortion following death of embryos or foetuses *in utero*.

This reduction in litter size caused by irradiation is called "semisterility", and the condition is transmissible genetically to viable offspring. Chronic irradiation at low daily dose rates appears to be much less effective in the production of semisterility, according to existing data. This may be explained on the basis that low doses are delivered to sperm populations which are continually renewed in the genitalia, and that relatively fewer sperm are subject to doses large enough to induce the chromosomal defects involved in semisterility. The lesser reduction in litter size during the second fertile period after irradiation with large single doses suggests that similar chromosomal defects in primitive spermatogenic cells or primordial undifferentiated cells are either not as significant in terms of the production of semisterility or are largely eliminated in some manner. More long-term investigations of this problem in chronic radiation experiments seems desirable to verify the degree to which regenerated sperm populations are defective in terms of semisterility.

The so-called sterile period may be a period of complete sterility or a period of subfertility or of fertility with reduced spermatogenesis, as manifest by partial atrophy of seminiferous epithelium and partially reduced sperm counts. Since critical or minimal numbers of normal potentially effective sperm per ejaculate are necessary for consistent successful reproduction, practical sterility or infertility may be associated with considerable but subnormal degrees of spermatogenesis. When spermatogenesis is partially arrested the number of sperm produced decreases and the percentages of sperm motile, alive, and normal tend to decrease also. With chronic irradiation spermatogenesis may stabilize at reduced levels for long periods of time if complete arrest does not occur, and further depressions may be slow in occurrence. Reduced sperm count and decreased quality of sperm persist accordingly.

The effects of irradiation on seminiferous epithelium are direct in that irradiation of the body with testes shielded does not produce them.

The effects of x-rays, gamma rays, and neutrons on spermatogenesis and reproduction are qualitatively similar, but neutrons are more potent in their effects on spermatogenesis and five or six times as potent in reducing litter size in matings done during the initial fertile period.

In regard to the efficiency of fractionated versus undivided doses of the same total size in producing testicular effects, there are experimental reports indicating no difference, others indicating less effect with fractionation, and others showing greater effects with fractionation.

Protraction of the dose fraction has little influence apparently on testicular effects unless the protraction is extreme, in which case the effect of a given total dose may be decreased, probably by virtue of permitting biologic recovery processes to operate at a more favourable rate with respect to the rate of production of injury by radiation.

The effects of fractionation of dose on the testes depends upon the size of the dose fraction, the interval of time between fractions, and the total dose. In general, fractionation has less influence on the effect of small total doses than on the effect of large total doses. Fractionation of large doses appears to increase damage in the mechanisms responsible for regeneration of germinal epithelium.

The dose-effect relationships in different species often appear contradictory, but are probably in reality complementary. For each species there is probably a

different dose-time relationship, in irradiation with divided doses, which is optimum for the efficient production of radiation injury. The empirical work which has been done on the testis has already made this apparent. Theoretically, in a tissue in which stem cells are radiosensitive and have the capacity both for active division and for differentiation, the most efficient mode of administration of radiation (per roentgen) to produce sterility in animals of a given species would be that designed, with respect to dose-time relationships, to take advantage of the biologic actions and reactions of the cells themselves. One of the most efficient dose-time relationships in spaced irradiation of the germinal epithelium would be one in which the dose fraction was small enough to permit attempts at division in spermatogonia but large enough to injure many of these cells to the extent that they die when mitosis is attempted, and one in which the time interval between exposures is such that the following exposure is administered when the effect of the previous dose is diminishing. A change of this inter-dose time interval in either direction would decrease the efficiency of the irradiation with respect to utilization of mitotic-linked death of spermatogonia.

In the case of cells having the capacity both for division and differentiation, irradiation tends to diminish the number of resting cells and dividing cells and by inhibition of division to increase the number of differentiating cells. It may be possible to increase to a maximum this effect in spermatogonia by suitable arrangement of the dose-time relationship in chronic irradiation. If the dose-time relationship optimum for maximum differentiation effects was quite different from that optimum for maximum mitotic-linked death, an optium compromised might be found, or these biologic effects could be handled separately with greater efficiency than is now the case.

There has been little investigation of the effects of irradiation on gametogenesis and reproduction in mammals, except for the work on rodents and some recent work on dogs. The single doses to the testes required to cause complete or nearly complete atrophy of the seminiferous epithelium are similar in size in these small animals and in the dog and man as well, all of the doses being within the LD_{50} range. However, the regenerative capacity of the seminiferous epithelium of the small laboratory animals is so great that very large single or divided doses, well above total-body LD_{100} doses, are required to sterilize permanently most or all of the animals of a group.

It would appear from data at hand that the dog, of all of the animals investigated in these respects, is the animal most similar to the human in terms of radiosensitivity and regenerative capacity of seminiferous epithelium. In general both dog and man reveal similar sensitivity which is greater than that in other experimental animals. In both cases, however, there is only little and fragmentary information on the effects of irradiation on spermatogenesis and reproduction, the minimal single or chronic permanent sterilization dose has not been studied definitively, and there is only little known of the regenerative capacity following irradiation.

The following table summarizes careful observations on male beagle dogs subjected to chronic exposure to x-rays from a 1,000 kvp x-ray machine and, in some cases, to neutrons from a cyclotron, 5 or 6 days per week.

Dose/week	Approximate total dose	Duration of exposure	Observations
0.3 r	62 r	4 yr	No significant change in sperm count.
0.6 r	124 r	4 yr	Do.
0.6 r	62 r	2 yr	Little change in germinal epithelium.
0.6 *n*	31 *n*	1 yr	Do.
3.0 r	156 r	1 yr	80 percent sterile; 20 percent reduced sperm counts.
3.0 r	312 r	2 yr	Substantial atrophy of germinal epithelium.
6.0 r	312 r	1 yr	Aspermic.
6.0 r	624 r	2 yr	Marked atrophy of germinal epithelium.
10.2 *n*	398 to 561 *n*	39 to 55 wks	Extreme atrophy of germinal epithelium.
15.4 r	477 r	31 wks	Aspermic after 375 r; sterile 1.25 years postirradiation so far.
15.4 r	634 r	41 wks	Aspermic after 375 r; sterile 1 year postirradiation so far.

Although cases of testicular atrophy in humans following irradiation have been observed since 1904, and were commonly observed soon after the Hiroshima and Nagaski bombings, little is known at present of the ultimate fate of the lesions produced in survivors and the effects of these lesions on fertility.

Regeneration of testes rendered atrophic by various doses and modes of irradiation has not been studied definitively in man. There are, however, isolated cases which have been studied to some extent, and there are reports of a zoospermia or oligonecrospermia or sterility in radiologists. Most of these cases were not studied carefully and extensively and in very few instances are there any reports or accurate estimates of doses involved. However, on the basis of the rather meager data available, certain estimates may be hazarded.

A single x-ray dose of 500 to 600 r is thought to produce permanent sterility for the human male and a dose of 250 r is thought to produce sterility for about one to two years.

If it is permissible at all to compare the meager human data with the results of many animal experiments in which regeneration was studied, the much more delayed and lower rate of testicular regeneration in man is apparent. Marked depletion of germinal epithelium is produced in the small experimental animals by doses in the LD_{50} range, but regeneration of the seminiferous epithelium is complete or nearly so in a matter of 3 to 5 months.

Man as well as the dog may have fewer of the radioresistant primordial cells, precursors of spermatogonia, or these cells may have less potential than is the case in the smaller or lower mammals. Other possible reasons for the delayed and slow recovery may be intimately associated with differences in metabolic rate and normal differences in rates of spermatogenesis. Factors which determine the relatively late maturation of the normal human testis may also modify the rate of regeneration of the human testis.

2. *The Female.*—Irradiation of the mammalian ovary can cause profound atrophy of the organ with temporary or permanent sterility depending upon the dose. Changes in the ovaries may be followed by dependent atrophic changes in accessory genitalia in most mammals.

The ova and follicular cells are the most radiosensitive cells in the mammalian ovary and cells of the corpora lutea and interstitial cells are relatively radioresistant. The radiosensitivity of the ova and folicular cells varies with their functional states at the time of irradiation. There are also marked differences in radiosensitivity between species. In most laboratory mammals the developing and mature follicles and ova appear to be more radiosensitive than the primordial follicles and ovocytes and some primary follicles persist after fairly large doses of radiation and may begin to develop long after irradiation.

Irradiation may sterilize the ovary by preventing the development of primary follicles of the ovary and by destroying the ova and follicular cells. Histologically, permanent ovarian sterility is indicated by the lack of ovarian follicles.

A dose of radiation which destroys all developing follicles causes failure of development of corpora lutea, which may lead to decrease of interstitial gland cells in animals which have these glands, since new cells will fail to be developed from corpora lutea.

Care should be used in the extrapolation of data from the mouse to human problems in regard to the ovary. The mouse ovary is peculiar in many respects. In it the primary follicles and ovocytes are exceptionally radiosensitive as compared with developing and mature follicles. The mouse ovary also has the tendency to develop invaginated tubular downgrowths of germinal epithelium and ovarian tumors, and these changes are easily accelerated and increased by relatively low doses of radiation. The peculiar differences in the mouse ovary, or the underlying causative mechanisms, are probably responsible for the exceptional radiosensitivity and the irreversibility of the effects of relatively low doses of radiation on the mouse ovary, as compared with ovaries of other laboratory mammals and the human female. In the female mouse a single x-ray dose of 150 r results in permanent sterility.

The size of the litter produced in the initial fertile period after irradiation of female animals is reduced and the size of the litter from irradiated females declines more rapidly with rising dose than the size of the litters from irradiated male mice.

Total body irradiation appears to produce greater effects on the ovary and on fertility in female animals than irradiation of ovaries alone with equivalent doses.

Since sterilization of the human ovary is a radiotherapeutic practice under certain circumstances, considerable data have accumulated on the radiosensitivity of this organ.

Single doses to the ovaries of 125 to 150 r may produce amenorrhea in 50% of women. A single dose of 170 r can produce temporary sterility for a period of 12 to 36 months. A dose of 500 r produces permanent sterility in most women,

but young women may require a larger dose. Doses between 500 r and 624 r have produced permanent sterility in 94% of a group of women (34 of 36 patients), and a localized dose of 625 r has produced permanent castration in a whole group of 72 patients.

3. *Sterility Doses for Men and Women.*—It seems quite possible that the single doses necessary to cause permanent sterility in 100% of men and women may not be far apart. However, there is insufficient information on men to permit an intelligent guess as to the exact amount of the difference.

It would appear that both male and female humans are probably among the most radiosensitive of those mammals studied, with respect to gonadal effects of irradition. It is also probable that the differences between single temporarily sterilizing doses and single permanently sterilizing doses of radiation are relatively small in the case of humans as compared with most of the laboratory mammals studied. On the basis of the data available the single gonadal dose of x- and gamma-radiation which would permanently sterilize most human males and females may be of the order of 500 to 625 r.

In animals with relatively poor gonadal regenerative capacity, such as the human, chronic irradition may be relatively of more serious consequence, and this tends to be supported by data from experiments on dogs. It is such exposure which may constitute the greatest practical human hazard as far as sterility is concerned.

A few experimental data indicate greater radiosensitivity in prepubertal animals, especially foetuses.

G. Effects of Irradiation on Growth and Development

This section is concerned with post-natal development and growth and does not include a detailed discussion of the effects of irradiation on prenatal development and organogenesis *per se.*

Regenerative and repair processes of the body appear to be fairly sensitive to the effects of ionizing radiation and inhibition of these processes may be very persistent, especially if vascular integrity and patency are impaired. Much more quantitative investigation of these aspects of the problem is needed, under circumstances of both total-body and localized irradiation.

Quantitative studies with rats seem to indicate that growth, as measured by body weight, is decreased by repeated exposure to as little as 24 r per week of whole body irradiation. It has been shown that a significant decrease in body weight can be produced by a schedule of repeated whole body exposures which does not cause any decrease in levels of hemoglobin or absolute neutrophils.

Localized irradiation of the epiphysis has been shown to cause measurable inhibition of bone growth and shortening of bones in humans and animals. In general, the greatest effect is seen in the youngest animals. Localized irradiation of the jaws has been followed by decrease in tooth growth.

Studies on children exposed to the atomic bomb in Japan indicate that growth and maturation are slightly retarded. The production of malformation by exposure of embryos or foetuses to irradiation has been investigated extensively in experimental animals. The production of relatively severe malformations in viable human offspring by irradition *in utero*, known from some clinical experience, has been confirmed by studies in Japanese atomic bomb survivors. The study of post-natal effects upon growth produced by irradiation of the foetus, however, has been neglected generally.

An extensive series of measurements on 4800 children at 6, 7, and 8 years after exposure to the atomic bomb at Hiroshima revealed in general that growth was retarded and maturation delayed. In another study involving several hundred children surviving the atomic bombings at Hiroshima and Nagasaki in 1945 and studied in the 2nd, 4th, and 5th years after irradiation, it was reported that the physical growth and development of the children were adversely affected, and the resulting retardation of their height, weight, and skeletal development was still evident at the end of 1950. The investigators expressed the belief that factors other than radiation may have contributed to the effects described. This study has been considered by some to be at variance with other studies on the same material.

Studies of children who had been irradiated *in utero* during the atomic bombings in Japan are noteworthy. In one series of 74 irradiated and 91 control children, roentgenographic survey failed to reveal differences in incidence of skeletal abnormalities between the exposed and control groups. A study of 4400 individuals who had been exposed to the bomb *in utero* or as children up to age 10 revealed 33 cases of microcephaly, with associated mental retardation

in 15 cases, and 19 cases of leukemia. There were also cases of mild visual disability among those now 16 to 19 years old who were exposed within 1800 meters of hypocenter. Observations on 205 children 4½ years old, who had been exposed at Hiroshima within approximately 1200 meters of hypocenter during the first half of uterine life, indicate that central nervous system defects were produced.

The mechanisms of growth inhibition by radiation are not understood. Biochemical and cytologic studies of animals in which growth has been inhibited appears to be indicated. The late effects, including life-span studies of exposure to ionizing radiation in pre-natal and early life merit further study.

IV. COMMENTS AND RECOMMENDATIONS

It appears likely that the after-effects of whole body external irradiation are quite general, consisting of irreversible injury to all the organ systems to at least some degree. Specific organ pathology or the incidence of specific disease is not prominent, however, except following large single doses or high intensity chronic irradiation and in even these cases, for the most part, the disease entities are not unusual, but occur earlier in the life of the animal. Although not clearly established at low chronic dosage levels premature aging with shortening of life span appears to be common to all whole body exposure. This effect is sufficiently large that it may provide a better criterion for limitation of exposure than increased incidence of specific disease.

The effects of partial body irradiation on life-span have not been studied except with internally deposited radioactive materials for which the local dosage is not usually well known. Consequently, comparisons with whole body effects are difficult. With partial body irradiation, when highly localized at least, local pathology is probably the best criterion for exposure limitation.

Because most pathologic studies have been made on animals dying or sacrificed during chronic irradiation, rather than after exposure and repair, the permanent after-effects have not been well separated from the total injury and related quantitatively to dose.

Animals prematurely aged by irradiation have not been studied to determine those changes which presumably have occurred in their physiological efficiency.

Except for alterations of pre-natal development very little is known of the after-effects of either whole or partial body irradiation in the young in comparison to mature animals.

Agents which, when administered to animals at the time of irradiation, permit them to survive doses which are ordinarily lethal, do not, according to scanty existing information, reduce the late effects. Consideration should be given to the possibility of increasing the reversibility of radiation injury and of diminishing thereby the late effects.

In anticipation that definitive information on man can be approached only by extrapolation from animals along with comparison to meager human data, studies on animals should be widely extended, not only with respect to experimental numbers to increase the accuracy of observation, but also to a greater variety of species to ascertain the generality of quantitative dose-effect relations.

V. REFERENCES

This report is based largely on the following detailed reviews, which were prepared in anticipation of the report. These reviews contain bibliographic references to most of the specific experimental works and reviews consulted in the preparation of the report.

Each of the following papers is an University of Rochester Atomic Energy Project Technical Report.

1. Blair, H. A. Data Pertaining to Shortening of Life-Span by Ionizing Radiation. University of Rochester Report UR–442 (1956).
2. Casarett, G. W. The Effects of Ionizing Radiations from External Sources on Gametogenesis and Fertility in Mammals. University of Rochester Report UR–441 (1956).
3. Hempelmann, L. H. Malignant Disease in Human Populations Exposed to Ionizing Radiation. University of Rochester Report UR–446 (1956).
4. Hursh, J. B., and Noonan, T. R. Some Late Effects of External Irradiation on Growing and on Adult Mammals. University of Rochester Report UR–445 (1956).

5. Ingram, M. Latent Hematological Effects of Exposure to Ionizing Radiations. University of Rochester Report UR–444 (1956).
6. Tuttle, L. W. Radiation Cataracts. University of Rochester Report UR–443 (1956).

Appendix 5

(A Letter from Japan)

St. Paul's University,
(Rikkyo Daigaku),
Ikebukuro, Tokyo, Japan, June 4, 1957.

Dr. C. Holifield,
Chairman of Radiation Subcommittee of Joint Committee on Atomic Energy, United States of America.

Dear Dr. Holifield: Enclosed is a copy of our comment on Dr. Libby's paper which was publicated at the meeting of the American Physical Society on April 26, 1957. I should appreciate it very much if you would kindly arrange our paper to offer at the committee.

Very sincerely yours,

Mituo Taketani, *Professor*,
Iwao Ogawa, *Assistant Professor*,
Tadayoshi Doke, *Assistant Professor*,
Department of Physics, St. Paul's University, Tokyo, Japan.

St. Paul's University (Rikkyo Daigaku)

IKEBUKURO, TOKYO, JAPAN

Distributed to: Dr. Masao Tuzuki, Dr. Fumio Yamazaki, Professor Koichi Murachi, Professor Yasuo Miyake, Professor Eizo Tajima, Professor Yoshio Hiyama, Professor J. Rotblat, Professor L. Pauling

1. It seems quite sure that the data submitted by many nations to the Scientific Committee of the United Nations in April 1957 show no significant differences among these data concerning the levels and distributions of strontium 90 accumulated on the ground at present. However, with respect to the estimated value in the future we find appreciable differences, e. g., contrary to Dr. Libby, who insists that the levels of strontium 90 accumulated on the ground in the United States may not exceed the present amount if the nuclear explosion tests are stopped, the British scientists made the estimate of the amount as about 2.5 times of the present one in about 10 years. Although these differences come from the diverse estimates of the amount of strontium 90 remained in the stratosphere, based on our own data we have made the nearly same estimate as that of the British scientists.

2. According to Dr. Libby most people obtain their calcium through milk products. Then he assumes a discrimination factor of 20 against strontium 90 from the soil to the human bones. However, in the world there are many people who obtain primely their calcium through vegetables, especially in the Orient. In such a case, it seems quite natural that we should estimate the discrimination factor appreciably lower than the case of depending on milk products. For instance, in Japan 50 percent of calcium source for the people is rice. Accordingly we should naturally estimate the discrimination factor as about 4.

3. Taking into account the above discussions, we may conclude that, if nuclear weapon tests are continued at the present rate for the coming three or four years, the radiation dose from strontium 90 deposited in the bones of those who have the Japanese-like food habit will reach the average value of the dose from natural radiations, e. g., cosmic ray, terrestrial radiation, and natural Ra in human bones.

4. Furthermore, Dr. Libby, in his report, seems to have made a definite mistake about the hazards by the very weak radiation. He investigated the correlation between the altitude difference of the cosmic ray and occurrence of bone cancer and leukemia, but such a method is nonsense unless the many kinds of natural radiations other than the cosmic ray are taken into account, because their doses are three times of the doses irradiated by cosmic ray, and these variation from place to place is rather appreciable.

On such a problem, the authorities in the MRC report of UK state that: "On the whole the experiments seem in favour of a proportionality between the frequency of tumours produced in a given length of time and the amount of radiative material in the body even at low dose levels."

If we accept such a proportionality, we may be able to conclude that the number of people in whose bodies cancers are produced will increase considerably. However, we consider that the detection of such hazards due to the nuclear weapon tests is very difficult, because a *percentage* of the occurrence of the hazards is quite small, although large in the absolute number. Taking into consideration the probability mentioned above, we should keep the dose from strontium 90 in human bone lower than that from natural radiation as possible.

5. Concerning caesium 137 very few data are available at present, and so we can not as yet make any accurate estimate of the amount in the human body in the future. However, this element seems to become an important material in the future if nuclear test explosions are continued at the present rate.

6. Recently we have continuously detected a considerable amount of long-life α-emitter in the air. Although we have not yet completed the radio-chemical analysis, we are afraid that there may be included an appreciable fraction of elements with very low MPC., such as Plutonium 239.

JAPANESE GENETICISTS ON RADIATION

The following "Statement concerning the genetic effects of radiation upon man" was prepared in April by the Genetics Society of Japan and the Japan Society of Human Genetics and sent out by them to a number of colleagues in other countries.

"With the increasing utilization of atomic energy, man inevitably has greater chance of being exposed to radiation than he has previously had. Generally speaking, any kind of radiations causes some damage to organisms. Particularly, their genetic effect is serious for the following reasons:

"1. It has been demonstrated by many experiments that radiations induce genetic changes or 'mutations' in organisms. Man cannot be exempt from this rule. Some such mutations occur naturally, but radiations raise their frequency.

"2. The great majority of these mutations are deleterious to mankind. Their effect may appear in the next generation, but more commonly only in subsequent generations. Therefore, the apparent escape of the next generation from such an effect does not ensure the genetic safety of all descendants.

"3. The incidence of mutation increases in proportion to the total dose of radiation given to the gonad. Whether irradiation is continuous or intermittent, the same amount of mutation is induced in either case, provided that the total dose is the same, since the mutation which was once induced persists even after the end of irradiation and is handed down to progeny. Thus the genetic effect of radiations through the gonad is fundamentally distinct from their direct damage to the body, which may disappear after the end of irradiation.

"4. Human population acquires natural mutations which are of very low incidence. These mutations are removed by natural selection, and the newly-appearing mutations and those removed by selection are mutually balanced, the incidence of mutant genes is thus kept in equilibrium. Additional mutations artificially induced by irradiation cause the break-down of this equilibrium, and an increase of the mutant genes possessed by the population. Such a change will lead to a gradual increase of individuals handicapped in physical strength or in mental capacity, increases the sacrifices of individuals and the burdens of the society, and leads to eventual disaster for mankind.

"From what has been pointed out above, we are led to conclude that any amount of radiation, however small it may be, is deleterious to the heredity of man. Although a certain dose has been set as 'permissible' for people engaged in the operation of X-rays and radioactive apparatus or substances, this is only aimed at the safety and health of those people themselves. However, as far as the gentic effect on their descendants is concerned, there is no theoretical limit below which danger may be entirely excluded.

"Although there can be hardly any question about the necessity for the peaceful utilization of atomic and other radiation energies, it is still all the more important to guard against any misuse or misoperation of such energies. This is not only for the safety of the present generation, but also for the health and prosperity of our descendants. Also, we must be on guard against the gentic effects of atomic or hydrogen bomb tests, which increase the level of radioactive contamination in the air and water.

"Under such circumstances, we geneticists eagerly hope that the general public will realize the urgency of the question at issue, and that effective means for its solution will be taken promptly."

Appendix 6

A Selection of Correspondence and Statements to and by the Atomic Energy Commission Concerning the Scientific and Technical Aspects of Fallout.

Statement by Lewis L. Strauss, Chairman, United States Atomic Energy Commission, February 1955

At a news conference on December 17, 1954, I stated that the staff of the Atomic Energy Commission was studying the subject of fallout and expressed the hope that information about it would be made public at a later date. "Fallout" is the word now applied to a phenomenon that follows the explosion of a nuclear weapon. Such an explosion, if the fireball touches the surface of the earth, draws up large amounts of materials into the bomb cloud. These materials subsequently fall back to earth as radioactive particles over a large area, mostly down-wind and relatively close to the point of explosion—although the lighter particles are carried great distances. The main radioactivity of fallout decreases very rapidly with time—for the most part, within the first hours after the explosion. An in-the-air explosion where the fireball does not touch the earth's surface does not produce any serious radiological fallout hazard.

Since nuclear weapons are in possession of the USSR, the Commission believes the American people wish to be informed regarding the dangers of nuclear explosions and the measures which individuals can take to protect themselves if an atomic attack should ever occur. Therefore, the Commission has condensed in the attached Report the information which can be made public at this time on the effects of the explosions of high-yield nuclear weapons.

The following excerpts and summarized sections contain the highlights of the Report itself.

FALLOUT PATTERN OF 1954 TEST IN THE PACIFIC

The very large thermonuclear device tested at Bikini Atoll on March 1, 1954, was detonated on a coral island and the ensuing fallout contaminated an elongated, cigar-shaped area extending approximately *220 statute miles down-wind and varying in width up to 40 miles.* In addition, there was a contaminated area up-wind and cross-wind extending possibly 20 miles from the point of detonation. Data was collected from 25 points on 5 atolls located from 10 to 330 miles down-wind (generally east) from Bikini Atoll. Due to an unexpected shift in the direction of the prevailing winds in the higher altitudes, the fallout missed the observation rafts that had been placed farther north previous to the test firing. The estimated contour of the pattern of fallout is, therefore, based only in part on data obtained from actual measurements and partly on calculations.

Data from this and other tests permits *estimates* of casualties which would have been sufferer within this contaminated area if it had been populated. These *estimates* assume: (1) that the people in the area would ignore even the most elementary precautions; (2) that they would not take shelter but would remain out of doors completely exposed for about 36 hours; and (3) that in consequence they would receive the maximum exposure. Therefore, it will be recognized that the estimates which follow are what might be termed *extreme* estimates since *they assume the worst possible conditions.*

On the basis of our data from this test and other information, it is estimated that. following the March 1, 1954, test explosion, there was sufficient radioactivity in a down-wind belt about 140 miles in length and of varying width up to 20 miles to have seriously threatened the lives of nearly all persons in the area *who took no protective measures.*

Some distance farther from the point of detonation, at about 160 miles down-wind and along the exis of the ellipse, the amount of radioactivity would have seriously threatened the lives of about one-half of the persons in the area who took no protective measures.

Near the outer edge of the ellipse, or approximately 190 miles down-wind, it is estimated that the level of radioactivity would have been sufficient to have seriously threatened the lives of 5 to 10 percent of any persons who might have remained exposed out of doors for all of the first 36 hours.

Thus, about 7,000 square miles of territory down-wind from the point of burst was so contaminated that survival might have depended upon prompt evacuation of the area or upon taking shelter and other protective measures.

At a distance of 220 miles or more down-wind, it is unlikely that any deaths would have occurred from radioactivity even if persons there had remained exposed up to 48 hours and had taken no safety measures.

The estimates cited above do not apply uniformly throughout the contaminated area inasmuch as the intensity of radioactivity within a region of heavy fallout will vary from point to point, due to such factors as air currents, rain, snow, and other atmospheric conditions. Because of this and because most persons, if given sufficient warning, probably would evacuate the area or take shelter and other precautionary measures, the actual percentage of fatalities could reasonably be presumed to be considerably smaller than these extreme estimates.

PROTECTION AGAINST FALLOUT

In the area of heavy fallout the greatest radiological hazard is that of exposure to *external* radiation, which can be greatly reduced by simple precautionary measures. Exposure can be reduced by taking shelter and by simple decontamination measures. Test data indicates that the radiation level, i. e., the rate of exposure, indoors on the first floor of an ordinary frame house in a fallout area would be about one-half the level out of doors. Even greater protection would be afforded by a brick or stone house. Taking shelter in the basement of an average residence would reduce the radiation level to about one-tenth that experienced out of doors. Shelter in an old-fashioned cyclone cellar, with a covering of earth three feet thick, would reduce the radiation level to about 1/5000, or down to a level completely safe, in even the most heavily contaminated area. Designs of shelters of simple yet effective construction have been prepared by the Civil Defense Administration and are available to the public.

Radioactive material deposited during the fallout may or may not be visible but would be revealed by radiation detection instruments such as Geiger counters. Any falling dust or ash that can be seen down-wind within a few hours after a nuclear explosion should be regarded as radioactive until measured by a radiation detection instrument.

Care should be taken to avoid the use of solid foods or liquids that may contain fallout particles.

If fallout particles come into contact with the skin, hair, or clothing, prompt decontamination precautions such as have been outlined by the Federal Civil Defense Administration will greatly reduce the danger. These include such simple measures as *thorough bathing of exposed parts of the body and a change of clothing.*

INTERNAL RADIATION EFFECTS

Two other factors must be considered in evaluating possible hazards from radioactive fallout. The first is the effect of internal radiation from fallout particles swallowed in food or liquids. The second is the effect of radiation upon the germ cells which transmit inherited characteristics from one generation to another. It should be noted that in neither case is there reason to believe that weapons testing programs of the United States have resulted in any serious public hazard.

The radioactive forms of strontium and iodine are the constituents of fallout which are of principal concern as internal sources of radiation through ingestion. The concentrations of these substances from nuclear detonations to date have been monitored at many localities, and the amounts detected have been insignificant, compared to concentrations which would be hazardous.

GENETIC EFFECTS OF RADIATION

There is a wide range of admissible opinion as to the genetic effects which radiation might have upon future generations, and conclusive data is not available at present on which to base an incontrovertible forecast. However, it is important to recognize that the average amount of radiation exposure received

by residents of the United States from all nuclear detonations to date has been about the same as the exposure received from one chest X-ray. The Commission's medical and biological advisers do not believe that this small amount of additional exposure is any basis for serious concern at this time.

BLAST AND HEAT EFFECTS

Two important characteristics of any nuclear explosion, other than those from fallout, are the effects of blast and heat, which are of the same nature for a thermonuclear bomb as for the earlier and smaller atomic bombs. The intensity and area of the blast and heat effects increase in relation to the greater energy yield of the explosion. Much information on these two effects has already been published by the Atomic Energy Commission, but it might be recalled that an atomic bomb of the earliest type, equivalent to 20,000 tons of TNT, would produce blast and heat sufficient to destroy, or damage severely, buildings within a radius of more than one mile from the explosion point. The United States has developed fission bombs many times as powerful as the first atomic bombs, and hydrogen weapons in the ranges of millions of tons (megatons) of TNT equivalent.

PROTECTION AGAINST BLAST AND HEAT

The hazard from both burn and blast effects well *outside* the central target area would be reduced greatly by shelter. Clothing or almost any kind of shelter would reduce the danger of direct burns, although there might be danger of clothing and structures becoming ignited. Also, shelter would materially reduce the hazard of blast injury by affording protection against flying or falling debris. As is generally known, the shelter afforded by ordinary city buildings would not suffice within the central area surrounding the point of explosion of a large nuclear weapon. For this reason, the Federal Civil Defense Administration recommends evacuation of the central areas of target zones on early warning of approaching attack.

FALLOUT FROM NEVADA TESTS

Only relatively small nuclear test explosions are conducted at the Nevada Test Site, in contrast to the tests of high-yield thermonuclear devices at the Pacific Proving Grounds. In Nevada, as well as in the Pacific, all tests are planned for times when forecast weather conditions minimize the possibility of fallout hazard. High air bursts at the Nevada Test Site have produced no significant fallout; heavy fallout from near-surface explosion has extended only a few miles from the point of burst. The hazard has been successfully confined to the controlled area of the Test Site. The highest actual dose of radiation at an off-site community has been estimated to be *less than one-third of the greatest amount of radiation which atomic energy workers are permitted to receive each year under the Atomic Energy Commission's conservative safety standards.*

CONCLUSION

In the event of war involving the use of atomic weapons, the fallout from large nuclear bombs exploded on or near the surface of the earth would create serious hazard to civilian populations in large areas outside the target zones. The Atomic Energy Commission hopes that these dangers will never be experienced by mankind. However, until the possibility of an atomic attack against us is eliminated by a *workable international plan for general disarmament*, the study and evaluation of the effects of weapons which might be used against us and the improvement of our means of self-defense are a paramount duty of our Government.

A Report by the United States Atomic Energy Commission on the Effects of High-Yield Nuclear Explosions

1. Considerable information on the effects of the explosions of atomic weapons has been made public by the Government since the first nuclear detonations in 1945. The handbook, "The Effects of Atomic Weapons", published in 1950, is being revised and brought up to date to include the effects of thermonuclear weapons, as a result of the most recent tests at the Pacific Proving Grounds. References to the effects of thermonuclear explosions have been made in several

official statments, beginning with Chairman Strauss' description of the phenomenon of "fallout" at a White House news conference on March 31, 1954. The following statement is designed to condense and correlate information, some of which already has been made public and other portions of which have been of a classified nature until now.

2. The effects of nuclear tests are evaluated for civil defense planning as well as for military and technological purposes. So long as nuclear weapons are in possession of any unfriendly power, the Commission believes the American public will wish to be as fully informed as possible as to the nature and extent of the dangers of nuclear attack and of the protective measures that can be taken by individuals and communities to avoid or minimize those dangers if we should be attacked.

3. Test conditions, which must necessarily form the principal basis of evaluating the effects of nuclear explosions, may differ markedly from those which might be expected if nuclear weapons were used against our population in wartime. It would be difficult to predict the size or kind of bomb an enemy might use against us in event of war, the exact means of its delivery, the height at which it would be exploded, or the number of bombs which might reach a given target. Nevertheless, the facts to follow are the fundamental ones at this time.

FOUR EFFECTS OF DETONATIONS

4. A nuclear detonation produces four major characteristics—blast, heat, immediate nuclear radiation, and residual radioactivity. Of these, the first three are essentially instantaneous, while the fourth has a more protracted effect. The phenomena of blast, heat, and nuclear radiation from the detonation of a thermonuclear bomb are of the same nature as those of earlier and smaller atomic bombs. The nature of the phenomena is, in general terms, standardized whether the bomb be a 20,000-ton (TNT equivalent) atomic weapon or a thermonuclear one of many times that power. The intensity and area of the blast, heat, and nuclear radiation increase in relation to the greater energy yield of the explosion. Information on these effects has been extensively publicized; therefore, the remainder of this report deals principally with effects other than heat and blast.

5. Residual radioactivity, although in no sense exclusive to high yield thermonuclear detonaions, does become a mater of major concern when a large thermonuclear device of the type used in the 1954 tests in the Pacific is exploded. The fallout of radioactivity from such an explosion, may, under certain conditions, settle over wide areas. Therefore, the extent and severity of this radioactive fallout has been a subject of continuing study since the first full-scale thermonuclear tests at the Pacific Proving Grounds on November 1, 1952. The results of these studies and of our evaluation of data obtained from the latest tests in the Pacific in March, 1954, are described in subsequent parts of this report.

6. It should be noted that if we had not conducted the full-scale thermonuclear tests mentioned above, we would have been in ignorance of the extent of the effects of radioactive fallout and, therefore, we would have been much more vulnerable to the dangers from fallout in the event an enemy should resort to radiological warfare against us.

BLAST AND HEAT EFFECTS

7. The effects of blast and heat from a nuclear explosion are relatively localized. One A-bomb of the earliest type equivalent to 20,000 tons of TNT (20 kilotons) would produce blast sufficient to destroy or damage severely residences within a radius of more than one mile from the point of burst. Within a radius of about a mile and a half, residences would be so damaged as to be unusable without repairs. A principal hazard to human beings would come from flying and falling debris and from fires due to such causes as broken gas and electric lines or overturned stoves. The area in which injuries to human beings would be caused by blast, therefore, would be about the same as the area of damage to structures.

8. The United States, as announced previously, has developed fission bombs many times as powerful as the first A-bombs, and hydrogen weapons in the ranges of millions of tons (megatons) of TNT equivalent. For these larger weapons, the blast effects can be calculated approximately by means of a scaling law, namely, the distance at which a given blast intensity is produced varies as the cube roots of the yields of the explosions.

9. Similarly, the heat and burn effects of nuclear explosions can be estimated from accumulated data. These effects, of course, are influenced by prevailing atmospheric conditions. The time element also is a prime factor. Very large weapons deliver heat over an appreciably greater period of time than smaller weapons. A given quantity of heat from a high-yield weapon, delivered over a longer period of time, will produce somewhat *less* severe burns than the same quantity of heat from a nominal detonation.

PROTECTION AGAINST BLAST AND HEAT

10. The hazard from both burn and blast effects in the *outer* affected areas would be reduced greatly by shelter. Clothing or almost any kind of shelter would reduce the danger of direct burns, although there might be some danger of clothing and structures becoming ignited. Also, shelter would materially reduce the hazard of blast injury by affording protection against flying or falling debris. The Federal Civil Defense Administration has made extensive studies of shelters and has issued plans for several simple and inexpensive types which can be utilized by householders. As is generally known, the shelter afforded by ordinary city buildings would not suffice within the central area surrounding the point of burst of a large nuclear weapon. For this reason, the Federal Civil Defense Administration recommends evacuation of the central areas of target zones on early warning of approaching attack.

RADIATION EFFECTS

11. The immediae nuclear radiation, i. e., the neutrons and gamma rays released instantaneously with the explosion of a large weapon on or near the ground, does not present a serious hazard beyond the area where heat and blast are of great concern.

FALLOUT RADIATION

12. However, particles with residual radioactivity produced by a detonation (as opposed to the immediate nuclear radiation) may fall out over an area much larger than that affected by blast and heat, and over a longer period of time. All nuclear detonations produce radioactive materials, but the nature and extent of the radioactive fallout depends on the conditions under which the bomb is fired. The main radioactivity of a bomb's fallout increases very rapidly with time—for the most part, within the first hours after the detonation.

FALLOUT FROM IN-THE-AIR DETONATIONS

13. In an in-the-air explosion where the fireball does not touch the earth's surface, the radioactivity produced in the bomb condenses only on solid particles from the bomb casing itself and the dust which happens to be in the air. In the absence of material drawn up from the surface, these substances will condense with the vapors from the bomb and air dust to form only the smallest particles. These minute substances may settle to the surface over a very wide area—probably spreading around the world—over a period of days, or even months. But they descend extremely slowly with the result that, by the time they have reached the earth's surface, the major part of their radioactivity has been dissipated harmlessly in the atmosphere, and the residual contamination is widely dispersed.

FALLOUT FROM SURFACE DETONATIONS

14. If, however, the weapon is detonated on the surface or close enough so that the fireball touches the surface, then large amounts of material will be drawn up into the bomb cloud. Many of the particles thus formed are heavy enough to descend rapidly while still intensely radioactive. The result is a comparatively localized area of extreme radioactive contamination and a much larger area of some hazard. Instead of wafting down slowly over a vast area, the larger and heavier particles fall rapidly before there has been an opportunity for them to decay harmlessly in the atmosphere and before the winds have had an opportunity to scatter them.

15. The area of hazard from radioactive fallout from a surface or near-surface explosion of a thermonuclear weapon is much larger than the areas seriously affected by heat and blast. The large radioactive cloud of a thermonuclear explosion rises with great rapidity to the highest levels of the atmosphere

and spreads over hundreds of square miles in the first hours. During this time the winds toss the extremely radioactive particles about and the pattern of the radioactive fallout is determined by the size of the particles and by the direction and velocities of the winds, including those up to 80,000 feet and above. The nature of the surface of the earth on which the bomb is fired also must be taken into consideration. Because of these variables, it is impossible to apply a single fallout pattern to all thermonuclear detonations, even test explosions conducted under selected conditions. However, with adequate knowledge of atmospheric conditions, including wind directions and velocities up to high levels and meteorological reports, the fallout region for any detonation usually can be predicted with considerable accuracy. In general terms, the region of severe fallout contamination from the detonation of a thermonuclear weapon fired on or near the surface can be described as an elongated, cigar-shaped area extending down-wind from the point of burst.

FALLOUT PATTERN OF 1954 TEST IN THE PACIFIC

16. The very large thermonuclear device fired at the Bikini Atoll on March 1, 1954, was exploded on a coral island. Coral consists of calcium carbonate, thus the detonation's radioactivity was spread by particles consisting largely of unslaked lime which, during the hours of descent, was slaked by moisture in the atmosphere. These particles ranged between 1/1000th and 1/50th of an inch in diameter and were, on the average, somewhat adhesive. The prevailing winds were westerly so the bomb cloud moved generally to the east and deposited the radioactive particles in varying amounts over an elliptical or cigar-shaped area. About 160 (statute) miles down-wind from the point of burst the early fallout was observed in the form of fine particles which looked like snow. Fallout began there about eight hours after the detonation and continued for several hours.

17. The roentgen is the commonly accepted unit of measurement of radiation dosage. A dose of about 25 roentgens of radioactivity received by a person over a brief space of time will produce temporary changes in the blood. A dose of some 100 roentgens received in a short interval may produce nausea and other symptoms of radiation sickness. About 450 roentgens delivered over a day or so might be fatal to approximately half of the persons so exposed. However, because of the body's repair processes, a total radiation dose which would be serious if incurred in a few minutes would produce much less effect if spread over a period of years. These statements may be helpful in understanding the data which follow.

18. The test explosion, at ground surface, contaminated a cigar-shaped area extending approximately *220 statute miles down-wind and varying in width up to 40 miles.* In addition, there was a contaminated area up-wind and cross-wind extending possibly 20 miles from the point of detonation. Data was collected from 25 points on 5 atolls located from 10 to 330 miles down-wind (generally east) from Bikini Atoll. Due to an unexpected shift in the direction of the prevailing winds in the higher altitudes, the fallout missed the observation rafts that had been placed farther north previous to the test firing. The estimated contour of the pattern of fallout is, therefore, based only in part on data obtained from actual measurements and partly on extrapolation, i. e., calculations based on known data, including factual information obtained during previous tests of smaller devices.

19. Data from this test permits *estimates* of casualties which would have been suffered within this contaminated area if it had been populated. These estimates assume: (1) that the people in the area would ignore even the most elementary precautions; (2) that they would not take shelter but would remain out of doors completely exposed for about 36 hours; and (3) that in consequence they would receive the maximum exposure. Therefore, it will be recognized that the estimates which follow are what might be termed *extreme estimates* since *they assume the worst possible* conditions.

20. On the basis of our data from this and other tests, it is estimated that, following the test explosion on March 1, 1954, there was sufficient radioactivity in a downwind belt about 140 miles in length and of varying width up to 20 miles to have seriously threatened the lives of nearly all persons in the area who *did not take protective measures.* During the actual tests, of course, there were no people in this zone. Inside Bikini Atoll at a point 10 miles downwind from the explosion it is estimated that the radiation dosage was about 5,000 roentgens for the first 36-hour period after the fallout. The highest radiation

measurement outside of Bikini Atoll indicated a dosage of 2300 roentgens for the same period. This was in the northwestern part of the Rongelap Atoll, about 100 miles from the point of detonation. Additional measurements in Rongelap Atoll indicated dosages, for the first 36 hour period, of 2000 roentgens at 110 miles, 1000 roentgens at 125 miles, and, farther south, only 150 roentgens at 115 miles from Bikini.

21. Some distance farther from the point of detonation, at about 160 miles down-wind and along the axis of the ellipse, the amount of radioactivity would have seriously threatened the lives of about one-half of the persons in the area who *failed to take protective measures.* It is estimated that the radiation dosage at that point was about 500 roentgens for the first 36-hour period.

22. Near the outer edge of the cigar-shaped area, or approximately 190 miles down-wind, it is estimated that the level of radioactivity would have been sufficient to have seriously threatened the lives of 5 to 10 percent of any persons who might have remained exposed out of doors for the first 36 hours. In this area the radiation dosage is estimated at about 300 roentgens for the first 36 hour period.

23. Thus, about 7,000 square miles of territory down-wind from the point of burst was so contaminated that survival *might* have depended upon prompt evacuation of the area or upon taking shelter and other protective measures.

24. At a distance of 220 miles or more down-wind, it is unlikely that any deaths would have occurred from radioactivity even if persons there had remained exposed up to 48 hours and had taken no safety measures.

25. The estimates cited above do not apply uniformly throughout the contaminated area inasmuch as the intensity of radioactivity within a region of heavy fallout will vary from point to point due to such factors as air currents, rain, snow, and other atmospheric conditions. Because of this and because most persons, if given sufficient warning, probably would evacuate the area or take shelter and other precautionary measures, the actual percentage of deaths could reasonably be presumed to be considerably *smaller* than these extreme estimates.

PROTECTION AGAINST FALLOUT

26. In an area of heavy fallout the greatest radiological hazard is that of exposure to *external* radiation. Simple precautionary measures can greatly reduce the hazard to life. Exposure can be reduced by taking shelter and by utilizing simple decontamination measures until such times as persons can leave the area. Test data indicate that the radiation level, i. e., the rate of exposure, indoors on the first floor of an ordinary frame house in a fallout area would be about one-half the level out of doors. Even greater protection would be afforded by a brick or stone house. Taking shelter in the basement of an average residence would reduce the radiation level to about one-tenth that experienced out of doors. Shelter in an old-fashioned cyclone cellar, with a covering of earth three feet thick, would reduce the radiation level to about 1/5000, or down to a level completely safe, in even the most heavily contaminated area. Designs of shelters of simple yet effective construction have been prepared by the Civil Defense Administration and are available to the public.

27. Radioactive material deposited during fallout may or may not be visible but would be revealed by radiation detection instruments such as Geiger counters. Any falling dust or ash that can be seen down-wind within a few hours after a nuclear explosion should be regarded as radioactive until measured by a radiation detection instrument and found to be harmless.

28. Care should be taken to avoid the use of solid foods or liquids that may contain fallout particles.

29. If fallout particles come into contact with the skin, hair or clothing, prompt decontamination precautions such as have been outlined by the Federal Civil Defense Administration will greatly reduce the danger. These include such simple measures as *thorough bathing of exposed parts of the body and a change of clothing.*

30. If persons in a heavy fallout area heeded warning or notification of an attack and evacuated the area or availed themselves of adequate protective measures, the percentage of fatalities would be greatly reduced even in the zone of heaviest fallout.

FALLOUT FROM NEVADA TESTS

31. Only relatively small nuclear test explosions are conducted at the Nevada Test Site, in contrast to the tests of high-yield thermonuclear devices at the Pacific Proving Grounds. In Nevada, as well as in the Pacific, all tests are

planned for times when forecast weather conditions minimize the possibility of fallout hazard. Methods of forecasting weather patterns in these areas are improving steadily. High air bursts at the Nevada Test Site have produced no significant fallout; heavy fallout from near-surface explosions has extended only a few miles from the point of burst. The hazard has been successfully confined to the controlled area of the Test Site. The highest actual dose of radiation at an off-site community has been estimated to be *less than one-third of the greatest amount of radiation which atomic energy workers are permitted to receive each year under the Atomic Energy Commission's conservative safety standards.*

INTERNAL RADIATION EFFECTS

32. Several basic facts should be kept in mind in evaluating the hazard from fallout radiation. First, radiation is not a new phenomenon created by the explosions of fission and thermonuclear weapons. Since the beginning of life, living things have been exposed constantly to radiation from natural sources. Cosmic rays from space constantly pass through our bodies. We are exposed to "background" radiation from radium and radon in the soil, water and air. Our bodies have always contained naturally radioactive potassium and carbon.

33. As pointed out earlier, detonations of all atomic weapons produce radioactivity, a portion of which is carried to high altitudes and over great distances in the form of fine particles. The percentage of this radioactivity which travels beyond the relatively near area of the explosion depends largely on the conditions under which the bomb is fired, the percentage being higher for in-the-air bursts where the fireball does not touch the earth's surface. The most widespread radioactivity is produced only by the longer-lived fission products, since the radioactivity of the shorter-lived products decays and disappears before the particles come down to earth in a matter of days, weeks, months, and even years. The longer-lived radioactive products may be distributed over the entire earth. However, as the particles are carried farther and farther to remote areas, the possibility of significant amounts of fallout decreases.

RADIOSTRONTIUM FALLOUT

34. One of the most biologically important radioactive substances found in fallout is strontium–90. It has a long lifetime—nearly 30 years on the average. Radiostrontium has a chemical similarity to calcium and, therefore, when taken into the body it has a tendency to collect in the bones. Radiostrontium can enter the body in two ways—by inhaling or by swallowing. Normally, the amount inhaled would be small compared with the amount one might swallow. Fallout material deposited directly on edible parts of plants may be eaten along with the plants, but washing the plants before they are eaten would remove most of this radioactive material. However, rainfall carrying the radiostrontium down to earth may deposit it in the soil where it can be taken up, in part, by plants and incorporated into plant tissues, later to be eaten by humans or by grazing animals which, in turn, provide food for humans.

35. Since the start of nuclear tests, careful measurements have been made of the distribution of radiostrontium over the earth's surface, in the soils, in plants and animal tissues, in the oceans, in rain, in the atmosphere and in all forms in which it might be expected to occur. The results of this study are reassuring. The amount of radiostrontium now present in the soil as a result of all nuclear explosions to date would have to be increased many thousand times before any effect on humans would be noticeable.

RADIOIODINE FALLOUT

36. Among the shorter-lived fission products involved in the study of internal radiation, the most biologically important is radioiodine–131 with an average life of only 11.5 days. Even though this product may be widely spread after a nuclear explosion, the possibility of serious hazard is limited by its relatively short life. Like the nonradioactive form of the element, it concentrates in the thyroid gland and, in excessive quantity, conceivably could damage the thyroid cells.

37. Scientists of the Atomic Energy Commission have estimated that the average exposure of people in the United States from radioiodine in the fallout from the entire series of tests in the spring of 1954 was only a few percent of the annual dose that can be received year after year and still have no noticeable effects.

38. These two isotopes—radiostrontium and radioiodine—constitute the principal internal hazards from the radioactivities produced by the detonations of atomic weapons, both fission and thermonuclear. The Atomic Energy Commission has been engaged for three years in a broad study of the radioactive forms of these isotopes and conducts year-round monitoring of these radioactivities in many locations. Any accumulation of these materials can be detected with great sensitivity so that ample warning of potential hazard could be given long before any actual danger occurred from test detonations. The amounts of radiostrontium and radioiodine which have fallen outside the areas near the test sites as a result of all atomic tests up to now are insignificant compared to concentrations that would be considered hazardous to health.

GENETIC EFFECTS OF RADIATION

39. One other effect of radiation must be considered in evaluating the long-range possibilities of hazard from nuclear detonations. This is the possible genetic effect upon the germ cells which transmit inherited characteristics from one generation to another. At our present stage of genetic knowledge, there is a rather wide range of admissible opinion on this subject.

40. In general, the total amount of radiation received by residents of the United States from all nuclear detonations to date, *including the Russian and British tests* and all of our own tests in the United States and the Pacific, has been about one-tenth of one roentgen. This is only about 1/100th of the average radiation exposure inevitably received from natural causes by a person during his or her reproductive lifetime. It is about the same as the exposure received from one chest x-ray.

41. The medical and biological advisers of the Atomic Energy Commission believe that the small amount of additional exposure of the general population of the United States from our nuclear weapons testing program will not seriously affect the genetic constitution of human beings. Nevertheless, we are continuing our thorough study of the entire question and will continue to report our findings to the American people.

SUMMARY

42. The Atomic Energy Commission hopes that the information on nuclear weapons effects contained in the foregoing report will never be reflected in human experience as the result of war. However, until the possibility of an atomic attack is eliminated by a workable international plan for general disarmament, the study and evaluation of weapons effects and civil defense protection measures must be a necessary duty of our government.

43. Inevitably, a certain element of risk is involved in the testing of nuclear weapons, just as there is some risk in manufacturing conventional explosives or in transporting inflammable substances such as oil or gasoline on our streets and highways. The degree of risk must be balanced against the great importance of the test programs to the security of the nation and of the free world. However, the degree of hazard can be evaluated with considerable accuracy and test conditions can be controlled to hold it to a minimum. None of the extensive data collected from all tests shows that residual radioactivity is being concentrated in dangerous amounts anywhere in the world outside the testing area.

44. In the event of war involving the use of atomic weapons, the fallout from large nuclear bombs exploded on or near the surface would create serious hazards to civilian populations in large areas outside the target zones. However, as mentioned in the foregoing Report, there are many simple and highly effective precautionary measures which must be taken by individuals to reduce casualties to a minimum outside the immediate area of complete or near-complete destruction by blast and heat. Many of these protective measures, such as shelter and decontamination procedures, have been detailed by the Federal Civil Defense Administration.

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington 25, D. C., August 8, 1956.

Hon. CLINTON P. ANDERSON,
Chairman, Joint Committee on Atomic Energy,
Congress of the United States.

DEAR SENATOR ANDERSON: Enclosed for the information of the Joint Committee on Atomic Energy are the Atomic Energy Commission's analyses on the following:

1. *The Biological Effects of Atomic Radiation*, a study by the National Academy of Sciences, 1956.
2. *The Hazards to Man of Nuclear and Allied Radiations*, a report by the United Kingdom Medical Research Council of June 1956.
3. *Race poisoning by Radiation*, an article by H. J. Muller, appearing in the Saturday Review, June 9, 1956.

Primary attention has been devoted to the two basic documents—the reports of the National Academy of Sciences and the United Kingdom Medical Research Council. These are competent, well written reports and we trust that an increased public understanding of the effects of atomic radiation will result from their publication. We note, however, that with the exception of the article "*Radioactive Fallout through September 1955*" by Eisenbud and Harley to be published this month in SCIENCE, there were no major data presented in either the National Academy of Sciences' report or the United Kingdom Medical Research Council report not already known to the Atomic Energy Commission and previously reported in open literature.

Except for some difference in the Strontium–90 data, the data, conclusions and recommendations of both reports were in good agreement considering the complexity of the problems and the independence of the two studies. The reports recommended an additional restriction as to the total radiation exposure to be permitted over a number of years. It is not anticipated that the reports will create any major change in our position regarding our weapons testing position or the Atomc-for-Peace program.

Or did they merely get to the point of recognizing a genetics hazard?

Both the NAS and the UK reports consider the genetics aspect of radiation as being paramount. It is with this factor principally in mind that upper limits of whole bodily exposure over a long period of time were recommended. Based on these recommendations and those forthcoming from the International Commission on Radiation Protection, the AEC may consider placing an upper limit of yearly exposure for atomic energy workers. However, the average exposure to atomic energy workers during past operations has been so far below the maximum permissible level that the placing of a yearly upper limit would not be expected to impose any major restriction.

The NAS report recommended an upper limit of 50 roentgens for *individual* exposure up to age 30, and 10r during the like period for the *general* populace. Except in the case of the March 1, 1954, incident involving intensive fallout in the Marshall Islands area, no individual outside the testing areas has been exposed to even the 10r maximum recommended for the populace as a result of fallout from the U. S. nuclear testing program. The NAS report estimates that if the nuclear weapons tests were continued at the present rate the average exposure for the general population of the United States over a 30-year period would be about one-tenth of a roentgen. In summary, the report was reassuring as regards nuclear weapons testing; it did not attempt to face up to the problems of an atomic war; and, finally, it was preoccupied with the potential hazards inherent in a developing era of large scale atomic power.

As to the Strontium–90 accumulating in the biosphere, the AEC will continue its extensive program of maintaining collection stations throughout the world and of analyses of the samples. This close and continuing checking system will provide ample warning of any significant upward trends in the Strontium–90 content of the biosphere before hazardous levels would be approached. It is indicated in the NAS report that the highest levels observed throughout the world are about 1/100 of the Academy's estimate of permissible concentration for the population as a whole. Furthermore, our knowledge of present pollution from radiostrontium is more exact and more extensive than that with respect to any other atmospheric pollution.

Sincerely yours,

K. E. Fields, *General Manager.*

Enclosures:
1. Critique of NAS Report
2. Critique of British Medical Research Council
3. "Race Poisoning by Radiation"—Comments on

(Enclosure 1)

Critique of the Report of the National Academy of Sciences

THE BIOLOGICAL EFFECTS OF ATOMIC RADIATION BASED ON (1) "A REPORT TO THE PUBLIC," AND (2) "SUMMARY REPORT."

To understand and best evaluate the implications of this report it is important to bear in mind the background of the individual scientists who made the study and their relationship to the National Academy of Sciencies-National Research Council and to the Government.

The NAS-NRC is not a Government organization. True, it was established by President Lincoln in order to have a distinguished body of scientists with whom the Government could consult at the time of the Civil War. On the other hand, it is a self-perpetuating body of free American scientists who control the membership of the Academy without any Government appointments. While various Federal agencies may appoint representatives to the various divisions of the National Research Council (the operating body of the NAS), they serve to bring problems to the Council for advice, and not to control the actions or the opinions of Council.

In the case of this study, the President of the NAS, Dr. Detlev W. Bronk, called together some 100 American scientists to carry out the study as individual citizens. While some of the scientists were Government employees and top advisers to Government on scientific matters, they were not acting in these capacities in their participation in the study.

The study was undertaken largely as a result of the concern felt throughout the country following the March 1, 1954 thermonuclear test explosion at Bikini, as a result of which a number of Marshall Islanders and Japanese fishermen were irradiated by fallout debris from the explosion. Subsequently, a number of scientific bodies in the U. S. passed resolutions requesting that a study be made of the possible effects on the human race of continued nuclear weapons testing.

In April, 1955, the Rockefeller Foundation provided the NAS with funds for undertaking a very broad study of the effects of atomic radiation. The subject reports are the final fruits of this study, which will be a continuing one.

Whereas the AEC has always been aware of the possible hazards from fallout from surface bursts of atomic weapons (see "Effects of Atomic Weapons," 1952), it had been even more aware of possible hazards to nearby livestock and the public generally from serious accidents which could conceivably occur to large production reactors such as those at the Hanford Works. The Bikini fallout incident made it abundantly clear that fallout *was* important from the standpoint of continued weapons testing and as a factor in civil defense planning. The problem of radiation effects has been under continuing review by the AEC and by the joint U. S., U. K. and Canada Tripartite meetings. In addition, the AEC has contributed a major portion of the basic scientific data for the deliberations of the National Committee for Radiation Protection and the International Commission for Radiation Protection.

A few words are in order on the general approach of the NAS study committees. They did not include an evaluation of the effects of an atomic war. As Dr. Bronk stated in the press conference of June 12, 1956, he could not define an atomic war so he asked the committees to limit themselves to peacetime atomic energy activities including weapons testing.

In the Foreword to the Summary Report, Dr. Bronk stated: "The use of atomic energy is perhaps one of the few major technological developments of the past 50 years in which careful consideration of the relationship of a new technology to the needs and welfare of human beings has kept pace with its development. Almost from the very beginning of the day of the Manhattan Project careful attention has been given to the biological and medical aspects of the subject. By contrast, the automobile revolutionized our pattern of living and working, but we are only now beginning to appreciate the problems of safety, urban congestion, nervous tension and atmospheric pollution which have accompanied its development. In the same way, the development of the aircraft industry outran our knowledge of how to meet the environmental needs of the human beings it intended to transport through the skies."

The scientists, save for the geneticists, were all person who had actively participated in the past in the efforts to reduce industrial toxicological hazards, air pollution, stream and harbor pollution, and soil and crop pollution, and de-

struction which has occurred with developing industries largely uncontrolled until serious damage had already taken place. They are determined that with a much greater body of knowledge to draw on concerning radiation effects, similar situations will not arise as a result of the rapidly growing atomic energy industry with its even greater potential dangers.

Consequently, once they had assured themselves on two points, namely: weapons testing at the present rate and with present safeguards was not a present menace, and the safety precautions of our present atomic energy operations were indeed effective, they became preoccupied with pointing out the problems inherent in a greatly expanded atomic energy industry. There constantly recurs through the report the idea that all is well today but for the future let us be very careful indeed.

In summary, the report was totally reassuring as regards nuclear weapons testing, it did not attempt to face up to the problems of an atomic war, and finally it was preoccupied with the potential hazards inherent in a developing era of large scale atomic power.

Summary Report of the Committee on Genetic Effects

This Committee consisted of geneticists, one authority on radiation pathology, one authority on radiological physics and radiation hazard control, and a mathematician, Dr. Warren Weaver of the Rockefeller Foundation, who chaired the group.

They considered the genetic effects against the background of present knowledge concerning radiation as a cause of mutations in microorganisms, plants, insects, and mice, bearing in mind the tendency of modern civilization to conserve all human life whether perfect or imperfect. They call attention to the perhaps greater importance of mutations which are relatively inapparent such as defects in resistance to disease processes, decreased fertility and curtailed life span, and impaired physical and mental vigor. The more dramatic mutations, monsters, still births, and early developmental defects leading to abortion and miscarriage are not apt to be passed on to another generation. The apparently relatively negative results of the genetics survey of the survivors' first generation at Hiroshima and Nagasaki serve to emphasize the validity of this point of view. This study demonstrated that with the methods used and the radiation dosages received, the heavily irradiated surviving population was not sufficiently large for it to be possible to demonstrate a statistically significant difference in the number of mutations in the offsprings of irradiated parents as compared with offsprings of non-irradiated control parents. It did not prove in any sense of the word that there was no genetic effect.

Following a general discussion of the mechanisms of genetic change especially as produced by radiation, both natural and artificial, the committee made certain recommendations. In doing so they used natural background radiation exposure (i. e., radiation from cosmic rays, igneous rocks, radium and radiopotassium in our bodies, etc.), and the so-called spontaneous mutation rate as base lines. In addition they were unanimous that no increase in the spontaneous mutations rate was desirable and that all radiation exposure to the germ cells at whatever rate of exposure did indeed increase the mutation rate in proportion to the total exposure received at the time of conception. Consequently they stated that all radiation exposure to the gonads was detrimental and consequently radiation exposures should be kept at the minimum consistent with the overall needs of a society.

They then observed that half of the American children were born of parents approximately 30 years of age or less. They noted that by the age of 30 the average American would receive germ cell exposures as follows:

1. Background or natural radioactivity---------------------------------- 4.3r
2. Medical X-rays--
3. Fallout from weapons testing if continued at rate for the past 5 years-- 0.1r (0.02 to 0.5r)

They then estimated that the exposure necessary to double the mutations rate in humans lay between 52 and 150r, more likely 30r to 80r, but also that different gene loci were quite different in their sensivity to radiation. Taking these observations into consideration they felt that if the population as a whole were to receive no more than 10r man-made exposure to radiation to the germ cells prior to the age of thirty no serious consequences would result. They, therefore, recommended that no one should receive a total accumulated dose to the repro-

duction cells of more than 50r prior to the age of thirty without clear cut medical reasons, and that in any event the average exposure of populations as a whole should not exceed 10r by the age of thirty. They point out that at present about ⅓ this figure is already being used up by medical x-ray exposures many of which could with proper precautions be greatly reduced.

As to occupational exposures the Committee considered this to be a limited group—no estimates were made as to its actual or potential size.

As finalized in the report the recommendations are:

1. There should be a national system of keeping radiation exposures on all persons as is now practiced at AEC establishments.
2. Medical exposures to the germ cells should be reduced.
3. No more than 10r by age thirty for the population as a whole.
4. The subject should be reviewed periodically with a view to possible further reduction in exposure.
5. No body, however, employed, should receive more than 50r of exposure prior to the age of 30.
6. For special activities inherent in which are a greater liability to over-exposure individuals who for one reason or other are unlikely to procreate should be selected.
7. The state of knowledge in the field of genetics has been outrun by our knowledge in the field of physics.
8. Keep all exposures to the germ cells as low as possible for radiation exposure is generally detrimental to living cells.

In essence, this Committee formalized the current thinking on the subject. It did not come up with any new or startling conclusions or recommendations.

The Committee on Pathologic Effects of Atomic Radiation

This Committee was composed of scientists well versed in radiation pathology and chaired by Dr. Shields Warren, Director of the Cancer Research Institute of the New England Deaconess Hospital, Boston, Massachusetts, and was for five years—1948 to 1952—Director of the Division of Biology and Medicine of the Atomic Energy Commission.

This group and subcommittees on blood, lung, delayed effects, and toxicity of ingested radioactive materials reviewed the present state of knowledge and found that our knowledge of immediate effects was much greater than for delayed effects. They observed a five year lessened life span for American radiologists, estimated to have received from a few roentgens to 1000r of exposure as compared with physicians not using radiation—and agreed that until we had more precise knowledge of the cumulative effects of repeated small exposure of the whole body to radiation the rule of thumb recommended by the Genetics Committee could equally well apply to medical effects. That is, no one should receive more than 50r total accumulated dose to the reproductive cells by age 30—and no more than 50r for each decade thereafter. This, they felt, would assure that any life expectancy curtailment would be exceedingly minor, and the likelihood of induced leukemia minimal. They noted that as far as effects on the blood-forming organs, the intestinal tract, etc., are concerned, none of these effects have been detected among those who have adhered to present permissible dose levels.

As for the hazards from ingestion and radioactive materials, they confirmed the validity of existing National Committee for Radiation Protection and International Commission for Radiation Protection recommendations and as for the most important of the fission products in fallout, namely Strontium–90 they stated "there seems to be no reason to hesitate to allow a universal human strontium burden of 1/10 of the permissible yielding 20 rep in a lifetime. * * * Visible changes in the skeleton have been reported only after hundreds of rep were accumulated and tumors only after 1,500 or more." The permissible level referred to is that recommended by the NCRP for industrial workers. The Committee noted that although "some children have accumulated a measurable amount of radioactive strontium in their bodies, the amount is quite small—a thousandth of what is considered a permissible dose. The Committee concluded, "then, that Strontium–90 is not a current threat, but if there were any substantial increase in the rate of contamination in the atmosphere, it could become one."

Committee on Meteorological Aspects of Atomic Radiation, Chairman Harry Wexler, U. S. Weather Bureau

In this part of the report there is the fullest discussion of fallout from nuclear weapons. They distinguish between kiloton bursts when the cloud does not penetrate to the stratosphere and megaton bursts where the cloud does. They estimate that with surface bursts, i. e., where the fireball touches the ground 70–80% of the residual radioactivity falls out nearby, i. e., with small weapons a few miles, with larger ones up to 300 miles or more. They emphasize the ease of predicting this "nearby" fallout pattern after the fact and the problem of predicting its precise pattern prior to detonation.

They speak of intermediate fallout, i. e., material of small particle size released below the stratosphere and some 80% of which falls out within three weeks in the same hemisphere in which it originated and tending to uneven distribution associated with rainfall and wind patterns along a broad band in the same general latitude as that of its origin. Finally, they refer to delayed fallout of material which has gained entry into the stratosphere. It is slow with an average storage time in the stratosphere of 10 years, plus or minus 5 years. This delayed fallout tends to distribute itself more or less uniformly over the surface of the earth over the years.

They state that "at present, the amount of Sr 90 in the stratosphere from nuclear weapons tests is far too small to approach maximum permissible concentration even if it were all deposited now." They urged a continuing program to check on the amount of radioactivity in the stratosphere as necessary so that if there were to be a greatly increased rate of thermonuclear weapons testing activities we would know at the earliest moment when it was time to slow down in terms of potential hazard from Sr 90 to man.

There is also a discussion of the radioactivity from fallout of the intermediate and delayed variety. They point out that it is usually too feeble to measure with a hand monitor—that air sampling does not give precise results as the amount of the passing air does not bear a direct relationship to what falls on the ground. The best measure of the actual fallout available to date are laboratory analysis of fallout on gummed paper, in collecting pots, and actual analysis of the soil.

There is a discussion of atmospheric radiocontamination as a result of uncontrolled release of materials such as radio-krypton and radioiodine from power reactors and processing plants. They point out that continued control over release of these products as is now done is essential. Control is by permitting a "cooling" time for short-lived radioactive materials to decay away, by off-gas cleaning, and by scheduling release of materials with due regard to meteorological conditions at the time.

There is a section on possible uses of radioactive materials in the study of the science of meteorology. Natural radon gas in the air can be helpful in understanding vertical movements of air from the land. Weapons tests have taught much with respect to lateral spread of air masses at various altitudes—how rain scavenges the atmosphere of particles—the rate of transport from the stratosphere to the troposphere and the removal time for water from the atmosphere. Experiments could be conducted using introduced radioactive materials under controlled conditions to study airflow and diffusion rates, hydrometeorology, i. e., condensation, precipitation and avaporation, and to study electricity of the atmosphere especially the possible relationship of electrical fields to the weather.

As to effects of nuclear weapons testing on the weather the committee stated:

1. Nuclear Weapon debris was not effective as a seeder for rain.
2. The amount of ionization produced is insignificant in meteorological terms.
3. There has been no measurable decrease in the amount of direct sunlight reaching the earth whereas volcanoes have been known to decrease it by as much as 10–20% for appreciable periods of time.
4. The apparent recent increase in severe storms is probably the result of "improved methods of reporting."

Committee on the Effects of Atomic Radiation, Oceanography and Fisheries, Chairman Roger Revelle, Scripps Institute of Oceanography

This group viewed the past record of this country with respect to pollution of streams, waterways and harbors with extreme repugnance. They point out

that 71% of the earth's surface is ocean and that eventually everything gets into the oceans.

They note that the sea as compared to the land is relatively nonradioactive. Natural radioactivity of the seas is 1/100 that of igneous rocks. As a result of weapons tests they report the following: two days after Operation Castle was over in the spring of 1954 there was a millionfold increase in radioactivity of the surface waters near Bikini; that after four months 1500 miles away it was three times the normal amount and that at 13 months are area of surface water contamination had spread over a million square miles, and that at a distance of 3500 miles from Bikini the "artificial" radiactivity was ⅕ the natural.

They concluded that to date there has probably been no damage to life in the sea except that at the test site proper. They call attention to concentration of radioactivity by plant forms in the sea and warn repeatedly against indiscriminate dumping of radioactive wastes into the sea. They discuss the "flushing time" of the Black Sea 2500 years as compared with perhaps 100 or 200 years for the shelf-deeps of the Atlantic and Caribbean. They stress they need to know much more about the ocean depths and their movements. (The International Geophysical Year has a very large-scale study of the depths planned for 1957–58.) This committee would apparently permit "controlled" sea disposal especially of short-lived radioactive materials. They recommend that "Industrial agencies formulate conventions for the safe disposal of atomic wastes at sea, based on existing knowledge." This would seem to be a very logical and necessary move. To date, except for small amounts of short-lived material, the U. S. has not dumped any radioactive wastes in the sea. We are still storing all process wastes in tanks.

They further recommend collaborative studies of the oceans and their organisms and though a beginning has been made urge a greater effort. Finally, they contend that in ten or twenty years certain radiatracer experiments will not be possible because of widespread low level contamination of the seas. This may well be true.

Committee on the Effects of Atomic Radiation on Agriculture and Food Supplies, Chairman Prof. A. G. Norman, University of Michigan, Ann Arbor, Mich.

This group first discussed the application of atomic energy techniques to the agricultural sciences. They feel great advances will be forthcoming, but perhaps not as soon as some claim. They note the value of radioactive tracer studies in improving our knowledge of how most economically to apply fertilizers, and to improve plant nutrition. They note the great potential value of ionizing radiation to induce mutations in speeding up crop improvement programs. They point up the invaluable contribution tracer studies can make to our understanding of animal nutrition. They touched on the problem of radioisotopes as possible contaminants in food products and point out that present law classes radioisotopes of any sort or in any amount as poisons. They urge a more realistic approach to this inasmuch as no food product is or ever has been literally free of radioactivity.

There is a general discussion of possible effects of fallout and the like on the ecology of the country. The committee recommends that it may well be in the public interest to expand the present programs to a continuous study of the changes in levels of background radiation and the movements of radioactivity in the system. (This is in essence an activity that the AEC has already underway and is expanding very much along the lines recommended.)

Finally, there is a statement concerning use of radiation for food processing. They note that relatively low exposure will destroy parasites in meat and inhibit sprouting in potatoes and onions. They also note that for sterilization extremely large doses are required (millions of roentgens). They felt this area of development was moving as rapidly as warranted and that the interest of the consumer will be adequately protected. They expect at a later date to review the evidence for wholesomeness and acceptability of irradiated foods.

Committee on Disposal and Dispersal of Radioactive Wastes, Chairman Abel Wolman, Johns Hopkins University

This group considered the magnitude of the problem not as it is today but as it will become with full scale production of power by nuclear reactors. They note that to date essentially none of these wastes has been returned to the environment. It is being stored in tanks. They point out the importance of developing

more economic methods of handling these wastes to the total development of atomic power. They have no quarrel with present practices but are concerned at the future magnitude of the problem. They estimate that by 1980 there will be 20×10^7 gallons of wastes to deal with. These must, they say, be contained in some form or other. AEC has a large program to cope with this problem on two fronts—one, to produce perhaps by sintering a non-leachable stable mass and, two, to remove by separation the worst offenders, Sr^{90} and $Cesium^{137}$.

They note present practices with regard to radioisotope production, transportation and utilization are sound, but suggest review from time to time as this very rapidly expanding activity cotninues.

The discussion of reactor accidents as a hazard is quite general. They urge continued requirement of containment of the reactor itself for all but small research reactors as practiced today in this country. They urge constant vigilance and conclude that the extreme hazard—total vaporization of a reactor—is unlikely.

In other words, this entire study adds up to reassurance for the present, and repeated urgings to keep vigilant lest this new technology needlessly get out of hand.

Enclosure 2

Critique of British Medical Research Council, the Hazards to Man of Nuclear and Allied Radiation

A report to the British Medical Research Council

The British Medical Research Council is a governmental body and was directed by the Prime Minister on 29 March 1955 to appoint a committee under the chairmanship of Sir Harold Himsworth to review the existing scientific evidence on the medical aspects of nuclear and allied radiation.

This report consists of eight chapters. The first four chapters deal with basic understandings of radiation and its biological effects, the fifth chapter with existing and foreseeable exposures due both to peacetime uses of atomic energy as well as to nuclear detonations in testing and in warfare, the sixth part with recommendations of permissible exposure and the seventh and eighth parts with summaries and conclusions.

Chapter I is an introduction to the report.

Chapter II discusses in simple terms the nature of radiation and its action on living cells. It deals with well known units, methods of measurement and biological effects.

Chapter III discusses the effects of radiation on the health of the individual. It includes discussions of the early effects upon the Japanese at Hiroshima and Nagasaki and the later development of an increased incidence of leukemia among the survivors. The British state they have demonstrated an increased incidence of leukemia in patients with arthritis of the spine treated with x-rays. They cite also American statistics on the increased evidence of leukemia in radiologists. They conclude that radiations can induce leukemia but do not quantitate the expose necessary for such an effect short of large single doses as at Hiroshima and Nagasaki.

There follows a discussion of radiation as an inducer of cancer and a conjecture that 1000r exposure to radon gas and its daughter produces induced lung cancer in the Schoneberg and Joachimsthal mines. Paradoxically, they go on to say that there is no evidence that external x- or gamma rays can cause lung tumors in man.

There is a discussion of radiation as a cause of bone tumors drawn principally from the reports of cancer of bones in radium dial workers and individuals given radium therapeutically. Most of this is American data. They feel there is not much of a factor of safety in the present maximum permissible concentration for radium. They indicate the risk of development of bone cancer from x-ray or gamma exposure in industry is insignificant. There is brief mention of skin cancer as induced by radiation, and thyroid gland cancer. Again the likelihood of this sort of thing from industrial exposure under modern controlled conditions is insignificant except, of course, in the event of accidental overexposure.

Radiation cataracts are mentioned as a hazard subject to ready control.

This report seems to understate effects of radiation on life span which has been so clearly proved in experiments with animals at, to be sure, radiation doses somewhat above permissible levels. The National Academy of Sciences report emphasizes this effect and cites the reduced life expectancy of American radiologists.

Both reports mention effects of radiation on developing fetuses, and the temporary sterility in males exposed to a few hundred roentgens at a single exposure. The British report is totally reassuring on the effects of occupational exposures on fertility.

Chapter IV is a very lengthy genetics effects discussion with many figures, tables and calculations and a critique of the Atomic Bomb Casualty Commission genetics stud: in Japan. This is a highly technical discussion and comes out with the same conclusions as does the National Academy of Sciences, namely that a dose of radiation which would double the mutation rate of a relatively small group of prospective parents would produce no noticeable effects. "For levels of radiation up to the doubling dose, and even some way beyond, the genetics effects of radiation are only appreciable when reckoned over the population as a whole and need cause no alarm to the individual on his own account."

Chapter V discusses natural radioactivity—radiation from appurtenances of civilization and occupational exposure to radiation. The report concludes that diagnostic medical X-rays produce exposures to the germ cells of the order of 22 percent that of background and constitute the most important source of man made irradiation. It is estimated that the United Kingdom Atomic Energy Authority's employees receive an average of 0.4 r per year.

The estimated external radiation exposure to people in Great Britain from fallout from all past nuclear tests has been quite minimal. "* * * Including all ordinary atomic bombs exploded before December 1955, and calculating all the radioactivity which they have contributed and will contribute over the next 50 years, it is found that the total dose which a man, continuously out of doors, night and day, would receive is 0.005 r. To this dose from ordinary atomic bombs must be added the dose of thermonuclear weapons. For these latter the dose from the radioactivity still to be deposited is more important. It can be estimated that the accumulated dose from thrmonuclear weapons is 0.002 to 0.003 r with another 0.027 r still to come. All these doses together add up to about 0.035 r from weapons already exploded. This is a maximum dose. The loss of radioactivity from weathering has not been taken into account, nor has the protection afforded by buildings in and around which most people in this country spend a large part of their lives. It would be realistic to divide the dose by three for weathering and by seven for protection afforded as a result of time spent in houses. The average inhabitant of this country may therefore receive in the next 50 years between 0.001 and 0.002 r from this fallout, or 0.02 to 0.04 percent of the radiation that he will receive during the same period from natural surroundings."

The report has this to say about the effects of a continuing program of testing: "* * * if the firing of both types of bomb were to continue indefinitely at the same rate as over the past few years, there would be a buildup of activity gradually reaching a plateau in about a hundred years time which, on the same basis of calculation, would give the average individual a dose over a period of 30 years of 0.026 r or about 0.9 percent of what he would receive in the same period from natural sources."

An important radioactive component of fallout material is Strontium–90. This isotope may be deposited in the bone and when present in sufficient quantities can cause bone cancer. The United Kingdom Medical Research Council report estimates that to date about 0.011 curies of Strontium–90 per square mile has fallen and that future deposits from past tests may produce a maximum of 0.045 curies of strontium-90 per square mile by 1965. These data are immediately evaluated in the report, "* * * these figures should be viewed against the background of the fact that the top one foot of soil has always contained on the average about one curie per square mile of the equally, if not more, dangerous naturally occurring radium."

They estimate the hazard from plutonium in fallout as very small. They feel Cesium 137, Iodine 131 and Barium 140 are of very little significance outside a nearby area of very heavy contamination. They estimate the gonadal dose as 1 percent of natural background and diagnostic radiology as 22 percent. The discussion of atomic warfare is too scant to consider here.

Chapter VI, Assessment of the Hazards of Exposure to Radiation, is in essence a summary of the foregoing—pointing out the differences between effects on the individual and genetic effects. They conjecture that no "authoritative recommendation will name a figure for permissible radiation dose to the whole population additional to that received from natural sources, which is more than twice that of the general value for natural background radiation." This is

estimated by the British at 0.1 r per year, hence 3 r in 30 years and 7 r in 70 years. The National Academy of Sciences estimate is an average 4.3 r in 30 years from natural background exposure and they recommend 10 r as the top figure for average exposure of the population as a whole before age 30.

As to the hazard from strontium[90] the report states "if the concentration in human bones showed signs of rising greatly beyond one-hundredth of that corresponding to the maximum permissible occupational level" they would feel that immediate consideration were required. This figure is 10 times the highest they report in man today. The National Academy of Sciences report states "It appears, then, that strontium[90] is not a current threat, but if there were any substantial increase in the rate of contamination of the atmosphere, it could become one."

The conclusions are to all intents and purposes identical to those of the National Academy of Sciences report.

1. Adequate justification should be required for the employment of any source of ionizing radiation on however small a scale. This is not explicitly stated in the National Academy of Sciences report but is inherent in it.

2. Dose levels to the individual—0.3 r per week—200 r in a lifetime for occupational exposures and no more than 50 r the first 30 years of life.

3. No more than twice natural background from man made sources for the population as a whole.

4. The present and foreseeable hazards from external radiation due to fallout at present rate of testing is insignificant. As to internal hazards from strontium[90] at its present level no detectable increase in the incidence of ill-effects is to be expected. "Nevertheless, recognizing all the inadequacy of our present knowledge, we cannot ignore the possibility, that if the rate of firing increases and particularly if greater numbers of thermonuclear weapons are used, we could within the lifetime of some now living, be approaching levels at which ill-effects might be produced in a small number of the population." This is a rather roundabout way of saying, "let's be careful."

5. a. All sources of radiation should be under close inspection. A personal record not only of doses of radiation received during occupation but also of exposures from all other sources such as medical diagnostic radiology should be kept for all persons whose occupation exposes them to additional sources of radiation. The National Academy of Sciences report would seem to include the whole population in its similar recommendations.

b. Present practices in medical diagnostic radiology should be reviewed with the object of clarifying the indications for different special types of examination now being carried out and defining more closely, both in relation to the patient and to the operators, the conditions which should be observed in their performance. This says, in effect, "let's tighten up on unnecessary exposures."

c. The uses of radiotherapy in non-malignant conditions should be critically examined—again, a warning to tighten up on unnecessary exposures.

d. The small amouts of irradiation from miscellaneous sources, such as x-ray machines used for shoe fitting, luminous watches and clocks, and television apparatus should be reduced as far as possible.

6. They end with a plea for better vital statistics. No comparable recommendation appears in the National Academy of Sciences report.

(Enclosure 3)

Comments on "Race Poisoning by Radiation"

By H. J. Muller

Professor Muller's remarks in regard to mutation changes resulting from nuclear warfare are in conformity with generally held views of geneticists. It is noted that Dr. Muller is a member of the National Academy of Sciences Study Committee on Genetics and the report issued by the Committee was unanimous.

With regard to the peacetime use of nuclear energy, Professor Muller presented estimates of life shortening based on two assumptions, i. e., that an atomic energy worker would receive the maximum permissible exposure every week for a 40 year working period and that the life shortening would be proportional to the total radiation dosage received. As indicated in Professor Muller's article and by figures released by the Atomic Energy Commission, the exposures to atomic energy workers have been considerably less than the maximum permissible amounts ("relatively few workers receive more than a fifth of this amount").

The possible effect of life shortening was considered by the Committee on Pathologic Effects of the National Academy of Sciences study on the biological effects of radiation. The Committee made the following statements:

"The shortening of life correlates roughly with dose of radiation, but has not yet been demonstrated at low doses."

"As the permissible dose level which they (Genetics Committee of the N. A. S.) have hypothesized as desirable for large populations were to be applied there would be no demonstrable somatic effect, although a theoretical minor shortening of life span could not be ruled out."

We are in complete agreement with Professor Muller's remarks that atomic energy "operations must be carried on with such rigorous safeguards that those working on the projects will feel no fear for themselves or their descendants."

In this connection, the AEC may consider placing an upper limit of yearly exposure for atomic energy workers. The average exposure to atomic energy workers during past operations, however, has been so far below the maximum permissible level that the placing of a yearly upper limit would not be expected to impose any major restrictions.

UNITED STATES SENATE,
COMMITTEE ON ARMED SERVICES,
January 31, 1957.

Hon. LEWIS L. STRAUSS,
Chairman, United States Atomic Energy Commission,
19th and Constitution Avenue, Washington 25, D. C.

DEAR ADMIRAL STRAUSS: Attached for your information is copy of letter just received which, as you will see, is signed by five professors of Yale University. The points brought out in this letter are to me extremely important, and coming from the source they do, I feel deserve most careful attention by the officials of our Government, particularly those working with and deciding the policy on development of nuclear weapons.

With kind regards, I am
Sincerely yours,

PRESCOTT BUSH,
United States Senate.

Enclosure.

JANUARY 21, 1957.

The Honorable Senator PRESCOTT BUSH,
United States Senate,
Washington, D. C.

DEAR SENATOR BUSH: At your request we are sending you an outline of the conversation you had with the members of the Yale Department of Biophysics in Woodbridge Hall on December 18th, 1956 on the subject of radiation hazards. The discussion covered the following points which we wished to emphasize to you.

1. The effects produced by nuclear weapons are world-wide. The radiation from our tests will not only be felt elsewhere, but similar weapons tested in Russia, England, or Australia will produce effects in our country as well as their own. The radiation effects are world-wide because most of the radioactivity is carried up into the stratosphere where it remains for such a long time (around ten years) that it becomes distributed over the whole world before the radioactive particles fall to earth or decay.

2. The effects produced by radiation are not reversible. If we were to find, ten years from now, that the people of this country had received too much radiation, we would be at an impasse because the residual effects would already have been produced. If we were to find that the population of this country, or of any country, or of the world for that matter, had received too much radiation, it would be too late to do anything about it. It seems clear that the present rate of world-wide weapons testing is bound to increase and therefore any margin of safety based upon our present rate of testing is sure to be wrong.

3. The effects of radiation are not completely understood. At the present time one of the big areas of disagreement among scientists is on the amount of strontium 90 (one of the radioactive elements produced only in the nuclear weapons tests) which should be permitted to accumulate in the world. Everyone's calculations of the amount of strontium 90 now at large are based on the AEC's data as presented by Commissioner Libby. The disagreement is on how much strontium 90 the human body is able to tolerate. In the twenty years since levels were set up for the maximum amount of radiation to which people

should be exposed, tolerance levels have been revised downward periodically and drastically. For example, the following table indicates the steady downward trend:

Estimated permissible level for occupational risk (in milliroentgens per week)

Year:	
1930	1,000
1936	500
1949	300
1956	100

We feel that there is a good chance that the tolerance levels will be still further revised downward in the future, as it has been in the past. If this were to happen, we certainly shall find ourselves in the tragic situation of having already exposed our population to radiation levels higher than those considered safe.

4. Because radiation effects seems to be cumulative, we may end up by exposing the population of this country to so much radiation from medical x-rays and nuclear weapons testing that we have no safety margin for radiation produced by nuclear power plants. At the present moment, the people of this country receive more radiation through medical x-rays (which of course are usually justified medically) than most of the other countries of the world. We, therefore, would be the first to suffer from the effects of nuclear weapons testing.

5. Because of the points mentioned above, we feel that the decision as to the extent of nuclear weapon testing should be made by a larger group of people than is at present making this decision. Obviously involved in the whole matter of weapons testing is the relative importance of the radiation danger about which we have spoken versus the useful information to be gathered for defense purposes. We do not advocate prohibition of nuclear weapons testing, but we do wish to see discussed the important point of the possible danger from such tests because we feel that the danger is real and that the people should not be misled about what is involved.

Yours sincerely,

(Signed:) ERNEST C. POLLARD,
Chairman, Biophysics Department.
RICHARD B. SETLOW,
Associate Professor Biophysics.
FRANKLIN HUTCHINSON,
Assistant Professor Biophysics.
WALTER R. GUILD,
Assistant Professor Biophysics.
HAROLD J. MOROWITZ,
Assistant Professor Biophysics.

APR 6, 1957.

Hon. PRESCOTT BUSH,
United States Senate

DEAR SENATOR BUSH: I am writing in response to your letter of January 31, 1957 forwarding a copy of a letter of January 21, 1957, signed by five members of the Yale University Department of Biophysics: Ernest C. Pollard, Richard B. Setlow, Franklin Hutchinson, Walter R. Guild and Harold J. Morowicz. I appreciate your patience in permitting the staff to take the necesary time to study the issues presented by the authors which as you know involve broad national defense policy and highly technical genetical and biophysical considerations. As was pointed out in the interim acknowledgment of the General Manager of March 13, 1957 and in earlier telephone discussions with your staff, the questions raised required the participation of several AEC divisions in the preparation of the final reply.

The letter of Professor Pollard and his colleagues, as is to be expected from their scientific standing and their familiarity with certain aspects of the problem, displays a high degree of appreciation of some of the factors involved in the limitation of human exposure to radiation from nuclear weapons test programs and from other sources. These and other factors upon which decisions regarding our various programs must be based are not only under constant study by members of our staff and by our consultants, but those factors involving biological effects of radiation and the exposure of persons to radioactive fallout from weapons tests are the subject of a substantial portion of our research programs in the fields of biology and medicine.

Our staff is in general agreement with the basic scientific statements made in the letter described by the authors as an outline of their conversaion with you on December 18, 1956. Because of the complexity of the subject, a brief summary of the considerations involved cannot be very definitive. I regret that we do not have available for comment a more complete statement of the authors' views.

I have asked our Division of Biology and Medicine to make an analysis of the various points briefly expressed in the letter in the light of our current thinking on problems of radiation protection. I believe you will be interested in their report, copies of which I enclose.

My own comments on specific statements contained in the letter are limited to a consideration of paragraph 5, which appears to summarize the conclusions of the authors, and which reads,

"Because of the points mentioned above, we feel that the decision as to the extent of nuclear weapon testing should be made by a larger group of people than is at present making this decision. *Obviously involved in the whole matter of weapons testing is the relative importance of the radiation danger about which we have spoken versus the useful information to be gathered for defense purposes.* We do not advocate prohibition of nuclear weapons testing, but we do wish to see discussed the important point of the possible danger from such tests because we feel that the danger is real and that the people should not be misled about what is involved."

I have underlined one sentence of paragraph 5 to emphasize that a basic consideration in decisons involving weapons testing is the relationship of the test programs to our national welfare. While the Atomic Energy Commission has certain responsibilities for the development and production of nuclear weapons appropriate to the estimated military needs of the nation, there are involved here, as you are of course aware, questions of national policy which extend far beyond the specific responsibilities of the Commission.

In the quoted paragraph above, the authors refer to "useful information to be gathered for defense purposes." Knowledge gained from past weapons tests have made it possible for us to increase reliability and safety, to improve the yield of explosive devices while reducing the amounts of material used, and to provide us weapons capable of being used tactically. One of the principal objectives of recent tests has been to develop weapons which may be used in defense against attacks and to reduce the radioactive fallout from weapons. As has been announced, the most recent weapons test series in the Pacific was planned and carried out with yields of explosive forces much less than those of the 1954 series.

In reaching decisions to recommend test programs for the approval of the President, the Atomic Energy Commission and the Department of Defense attempt to take advantage of all available information. The number of persons who can participate in the total of all of the considerations involved is necessarily limited. On those facets of the problem which do permit wider participation, we have sought and received counsel from many sources. In addition to persons with whom we have formal relationships of one kind or another, there is constant give and take with a great number of scientists and other professional persons here and abroad.

The total of our contacts represents, we believe, a good cross section of informed opinion throughout the world.

We share the author's view that possible dangers from weapons tests should be discussed and that the people should not be misled about what is involved in exposure to radiation from weapons tests or from peaceful uses of nuclear energy. The potential hazards should neither be minimized nor exaggerated. We believe that the extensive data which have been collected, particularly in programs sponsored by the United States and by the United Kingdom, demonstrate that the degree of potential hazard to the public is very low.

Much of the pertinent information is summarized in reports published last year on studies made concurrently but independently by the National Academy of Sciences and by the British Medical Research Council. Copies of these reports are enclosed for your convenient reference.

I hope that you will find the information which we are submitting helpful. If I can be of further aid to you, by arranging for discussions with appropriate Commission personnel or by other means, please call on me.

Sincerely yours,

W. F. LIBBY, *Acting Chairman.*

Enclosures:

Comments by Division of Biology and Medicine.

Reports of National Academy of Sciences and British Medical Research Council.

REPORT ON QUESTIONS RAISED BY YALE DEPARTMENT OF BIOPHYSICS IN LETTER TO SENATOR PRESCOTT BUSH DECEMBER 18, 1956

Prepared by Division of Biology and Medicine
U. S. Atomic Energy Commission March 27, 1957

The following report discusses a letter from members of the Department of Biophysics, Yale University, to Senator Prescott Bush, January 21, 1957. The letter is signed by Ernest C. Pollard, Richard B. Setlow, Franklin Hutchinson, Walter R. Guild, and Harold J. Morowitz. The letter was transmitted by Senator Bush January 31, 1957, with the following note:

"Attached for your information is copy of letter just received which, as you will see, is signed by five professors of Yale University. The points brought out in this letter are to me extremely important, and coming from the source they do, I feel deserve most careful attention by the officials of our Government, particularly those working with and deciding the policy on development of nuclear weapons."

The letter is described by the authors as an outline of a conversation on the subject of radiation hazards between Senator Bush and members of the Yale Department of Biophysics, December 18, 1956. The letter includes five numbered paragraphs, each of which discusses a point introduced by the initial sentence. For convenience in the following discussion, reference to each of these paragraphs is by number, with the point stated first, followed by our comment.

Because our comments may appear to be rather critical, we wish to state in advance that the letter is unusually well written considering the brevity with which the authors attempted to express their ideas. Thoughtful treatment of the subject is, of course, to be expected from a group of such competence and professional standing. The comments are as follows:

Paragraph 1. The paragraph is limited to factual statements on which there is general agreement.

Paragraph 2. "The effects of radiation are not reversible." The authors apparently have in mind some very specific effects of radiation, such as genetic effects, which are generally considered to be not reversible. We do not believe that, in general, the statement can be supported. For example, the transient effects characteristic of radiation sickness are appropriately described as reversible. Whether or not those effects of exposure of an individual to radiation which may, but not necessarily do, result in leukemia or cancer are to be considered as reversible or not reversible may be a question of semantics. For example, if a number of persons receive equal doses of radiation and one contracts leukemia as a result, it would appear that in the case of the one person some of the effects of the radiation were not entirely reversible. It is not apparent, however, that this is necessarily true in the cases of those who failed to contract the disease.

"If we were to find, ten years from now, that the people of this country * * * or of any country, or of the world * * * had received too much radiation, it would be too late to do anything about it." This is true, of course, whether the consequences implied by "too much radiation" are extremely serious or of relatively minor import from the point of view of their total impact on the population as a whole. The nature of the biological effects of radiation is such that it is impossible to categorically divide levels of exposure to radiation into *safe* and *unsafe* values, if one uses the term *safe* in the sense of absolute freedom from risk.[1] This is because the severity of biological effects, or the probability of a serious effect, increases from very low values for low exposures to radiation to high values for high exposures to radiation.

For example, it is generally considered that any exposure to radiation, no matter how small, makes a correspondingly small contribution to the probability of genetic mutation. On page 22 of the National Academy of Science Summary Reports it is stated that "U. S. residents have, on the average, been receiving from fall-out over the past five years a dose which, if weapons testing were continued at the same rate, is estimated to produce a total 30 year dose of about one tenth of a roentgen * * *" By comparison, it is stated, p. 21, that each person in the United States receives from natural background

[1] While the word *safe* is defined as freedom from risk, it is commonly used to describe situations in which the risk is not zero but is too low to be of concern or, possibly, to be recognized.

radiation "a total accumulated dose of about 4.3 roentgens over a 30 year period" and, from medical x-rays, "on the average a total accumulated dose to the gonads which is about 3 roentgens of x-radiation during a 30 year period." The dose rate required to double the natural rate of genetic mutation, if continued over a period of many generations, is estimated, p. 24, to lie in the range of 30 roentgens to 80 roentgens. On the basis of these estimates, if weapons tests were continued at the present rate, the effect of radioactive fallout from the tests on the genetic mutation rate would be to increase it by less than 1% of its natural value. A very small fraction of the population would be affected and a much smaller fraction would be seriously affected. The risk to the average individual, and the impact on the population as a whole, would be extremely small. Yet, since some individuals might be seriously affected, we must assume that even at these low levels of exposure people are receiving "too much radiation" unless we are convinced that the probable benefit to each person outweighs the risk.

Some perspective on relative values may be obtained by observing, on the basis of estimates quoted above, that exposures to radiation from natural sources (radioactive materials naturally in the earth's crust and cosmic rays from outer space) are more than ten times as high as those estimated to result from weapons testing if continued at the present rate. These are levels of radiation in which the human race has developed and prospered. Whatever risk there may be to the individual member of the race as a result of these relatively low additional exposures, it is quite low compared to many common hazards of life.

It appears that the authors are conscious of the smallness of the risk involved at the levels of exposure under discussion, since in paragraph 4 of their letter they restate their fear in the words, "* * * we may end up by exposing the population of this country to so much radiation from medical x-rays and nuclear weapons testing that we have no safety margin for radiation produced by nuclear power plants." That they also recognize the element of balance of risk against probable benefit is reflected in the statement in paragraph 5 of their letter, to wit: "Obviously involved in the whole matter of weapons testing is the relative importance of the radiation danger about which we have spoken versus the useful information to be gathered for defense purposes."

Summarizing our discussion of paragraph 2, it appears that whether or not ten years from now the population will have received too much radiation as a result of weapons tests will depend not only on the number of persons who may have suffered as a consequence of our weapons tests but also upon the number who may have been enabled to avoid suffering as a result of the contributions of the tests to our defense efforts.

Paragraph 3. "The effects of radiation are not completely understood." It would, perhaps, be more appropriate to say that the processes of life are not completely understood. We probably know more about the biological effects of radiation than those of any other element in our environment; certainly more than we know about the effects of many drugs and organic chemicals developed during the past few years, and perhaps more than about such factors as psychological stress, diet, exercise, etc.

"At the present time one of the big areas of disagreement among scientists is on the amount of strontium 90 * * * which should be permitted to accumulate in the world. Everyone's calculations of the amount of strontium 90 now at large are based on the AEC's data as presented by Commissioner Libby. The disagreement is on how much strontium 90 the human body is able to tolerate." The AEC has been supporting a broad program of investigation of strontium 90 in the atmosphere, soils, plants, animals and humans for more than three years. Most of the investigators are employees of other government agencies, universities, hospitals, etc. A similar program has been under way in the United Kingdom for about the same length of time, and other countries are undertaking studies of this kind. The best data available on the human content of strontium 90 is that in the report, "Strontium 90 in Man" by J. L. Kulp, W. R. Eckelmann, and A. R. Schulert of Columbia University, published in *Science*, February 8, 1957. The skeletal concentrations observed by Kulp, et al, are generally less than 0.01 of those considered acceptable in general population groups by such scientific groups as the International Commission on Radiological Protection, the National Academy of Sciences, and the Medical Research Council of the United Kingdom.

We believe that the principal area of disagreement between scientists well informed in this field is not on how much strontium 90 the body can tolerate,

in the usual sense of the word, but whether or not there may be a very small probability that a low concentration of strontium 90 in the skeleton of an individual will result in bone cancer. There is no experience of production of bone cancers in humans by strontium 90. Experience in humans is limited to the effects of radium as, for example, in the case of the luminizing industry. Estimates of the effects of strontium 90 to be expected in humans are based on comparative studies of strontium 90 and radium in animals and comparative studies of radium in animals and humans. Because bone cancers occur normally in animals (and in humans), and because cancers induced by radioactive materiels cannot be distinguished from those which occur from other causes, the relationship between concentrations of strontium 90 in the skeleton and the resultant probability of bone cancer can be determined only by conparing rates of occurrence in animals containing strontium 90 with rates of occurrence in normal animals. The increase in rate of occurrence that can be measured with confidence depends upon the numbers of animales under observation.

From studies made over the past ten years, it is estimated that from 1,000 to 10,000 times the concentrations of strontium 90 currently found in the skeletons of young humans would be required to produce a detectable increase in the normal rate of bone cancer. Some of our best informed radiobiologists are confident that somewhere below these concentrations the probability of a resulting increase in the rate of bone cancer would be zero, while others believe it possible that even at the very lowest concentrations there may exist correspondingly low probabilities of a bone cancer resulting from strontium 90. In the latter case, the situation is similar to the genetic case discussed in connection with paragraph 2. The question then becomes not how much can the human body tolerate but what is the risk at low levels of exposure in relation to the probable benefits associated with the exposure.

While informed radiobiologists disagree on the subject of whether or not relatively low concentrations of strontium 90 in the skeleton might conceivably result in small increases in the rate of occurrence of bone cancer, they generally agree that is such increases in rate do occur, they will not be large enough to be detectable, even at concentrations much higher than those currently observed. The degree to which speculation on the possibility of an increase in the rate of bone cancer too small to observe has become a matter of public concern is in striking contrast to the apathy with which the public accepts the fact that the rate of lung cancer has increased over the past 20 or 30 years by a factor of about ten.

"In the twenty years since levels were set for the maximum amount of radiation to which people should be exposed, tolerance levels have been revised downward periodically and drastically. . . . We feel that there is a good chance that the tolerance level will be still further revised downward in the future, as it has been in the past. If this were to happen, we certainly shall find ourselves in the tragic situation of having already exposed our population to radiation levels higher than those considered safe." In our discussion of paragraph 2, we have indicated that we do not consider that any level of radiation, no matter how low, can be considered absolutely "safe". If the exposure of the whole population to radiation at a certain level were to result in the premature death of one person, the result would be a tragedy. If it resulted in the death of several persons, it would be a greater tragedy. But, if one is realistic, one must admit that risk to human life is involved not only in other phases of our defense effort but, indirectly, in many decisions involving our foreign policy. In the case of radioactive fallout, the significant risk is not from weapons tests, but from the possibility of a nuclear war in which levels of radiation exposure may be higher by factors ranging upward to tens of thousands or hundreds of thousands. Obviously, this would be a much greater tragedy.

Paragraph 4. "Because radiation effects seem to be cumulative, we may end up by exposing the population of this country to so much radiation from medical x-rays and nuclear weapons testing that we have no safety margin for radiation produced by nuclear power plants." Estimates quoted in discussion of paragraph 2 indicated that at present rates of weapons testing, anticipated exposures to radiation from fallout are about one-thirtieth of current average rates from medical x-rays. At these relative magnitudes, a four percent increase in the use of medical x-rays would wipe out any gains resulting from the elimination of fallout from weapons testing. However, the relative merit of exposing persons to radiation from medical x-rays, nuclear power reactors, and fallout from weapons tests must depend upon the contribution that each may make to per-

sonal and national welfare. The primary consideration with respect to radiation from fallout is, how important to our national welfare is our nuclear weapons program?

Paragraph 5. "Because of the points mentioned above, we feel that the decision as to the extent of nuclear weapons testing should be made by a larger group of people than is at present making this decision." Decisions as to the extent to which weapons tests shall be conducted involve highly sensitive information including weapons design and capabilities, military strategy, and national policies. This limits participation in these decisions to persons officially responsible for our national security.

In evaluation of the radiological hazards associated with weapons tests for use in national planning, the Atomic Energy Commission not only seeks existing information from all available sources but supports extensive research on the production, dissemination, biological uptake, and effects of radioactive materials released by nuclear detonations. The number of persons with whom the Commission has contact on various phases of the question is very large.

"Obviously involved in the whole matter of weapons testing is the relative importance of the radiation danger about which we have spoken versus the useful information to be gathered for defense purposes." We believe this to be the primary consideration.

"We do not advocate prohibition of nuclear weapons testing, but we do wish to see discussed the important point of the possible danger from such tests because we feel that the danger is real and that the people should not be misled about what is involved." The discussions contained in reports issued last year by the National Academy of Sciences and the British Medical Research Council not only explain the possible dangers from weapons tests quite thoroughly, but present differences in the views of various radiobiologists. These reports were written by study panels composed of leading scientists in the respective countries, and it is our belief that they present a good cross section of scientific opinion on these subjects. Copies of the following reports are provided herewith for transmission to Senator Bush:

(1) National Academy of Sciences, "*The Biological Effects of Atomic Radiations, A Report to the Public.*" It is recommended that this brief, non-technical survey of the subject be read before referring to the other reports.

(2) British Medical Research Council, "*The Hazards to Man of Nuclear and Allied Radiations.*" This report has been made more readable than many scientific reports without sacrifice of technical accuracy. It is suggested that Chapter VII, pages 70 ff., summarizing the discussion, be read first. The summary statements refer to paragraphs of the text by number, facilitating the selection of portions of principal interest.

(3) National Academy of Sciences, "*The Biological Effects of Atomic Radiation, Summary Reports.*" This is a summary of individual reports by six committees. While it covers much of the same ground as the report of the British Medical Research Council, views expressed in the two reports involve some basic differences. For example, the American report considers it most probable that below certain levels of exposure to radiation, the contribution of the exposure to the probability that an individual will develop cancer or leukemia is absolutely zero; while the British report considers it likely that even at the lowest levels of exposure there are correspondingly small but real contributions to the probability that the individual will develop one of these diseases.

(4) National Academy of Sciences, "*Pathological Effects of Atomic Radiation.*" Appendix II of this report summarizes the considerations related to the irradiation of persons and populations by radioactive materials within the body.

It is desirable that the general public be as well informed as is practical on all phases of radiation hazards. We know of no information on this subject which is withheld from the public, but because of the complexity of the subject, it is difficult to transmit it comprehensively in a form or forms generally usable by individual laymen. Introduction of even the primary facts into the day-to-day thinking of a substantial proportion of the general public is a problem in education of which the solution may require many years.

SUMMARY

At the present time, it cannot be stated categorically that there is a level of radiation which is absolutely safe. Mainly because of genetic considerations,

there exists the possibility that some biological effect—perhaps difficult to detect—will be produced at very low doses. The degree of risk, however, would be correspondingly low.

The probability is remote that any individual would be seriously affected by low level radiation contributed by fallout from weapons tests. Unless, however, the risk is absolutely zero, a low individual probability when extrapolated to a large population will result in the statistical possibility that some members of the population may be affected. Because the risks here are very much smaller than the normal hazards of life, it is impossible to estimate the maximum number of persons who might be affected.

The dose of radiation outside the body resulting from fallout contributes about 1/30 of the dose to the population from either natural sources or from medical X-rays. Current concentrations of strontium 90 in the skeleton as a result of fallout are substantially below (about 0.01) levels considered acceptable by recognized independent scientific bodies.

The important question then is whether or not the discontinuation of weapons tests by the United States for the sole purpose of reducing the presently small risk from fallout would not be more than offset by the loss of data, vital to the national security, which weapons tests yield. In the absence of an international disarmament agreement, freezing our weapons technology at the present level may constitute a far greater risk. In such an event it becomes necessary to speak of radiation risk in terms of lethal doses to large segments of the population in the event of nuclear attack, in contrast to the present discussion which is in terms of fractions of permissible levels and statistical possibilities.

WASHINGTON, D. C., *April 25, 1957.*

Dr. ALBERT SCHWEITZER,
Lambarene Hospital, Lambarene, Gabon, French Equatorial Africa.

DEAR DR. SCHWEITZER: I am writing you as a scientist, to present data bearing on a scientific fact: The degree of hazard to humanity from radioactive fallout from nuclear weapons tests.

In the press on April 24, I read your statement from Oslo on the hazards of nuclear weapons testing, and in this way learned of your fears that the present testing program may be dangerous. Since I have spent much time during the past several years in the study of this question, I am taking the liberty of writing you. Also, since your statement was issued to news media and received wide public attention, I am making this letter public in the belief that every possible action should be taken to increase public understanding on the important question of weapons testing.

Your belief in the sanctity of life, and the dedication with which you have devoted your own life and talents to unselfish causes, have made a deep impression on the minds of persons throughout the world. Your concern over the possible effects of nuclear tests is characteristic of the humane and sensitive qualities which you always have displayed, and for which you are justly honored. Along with these qualities, I know you have the intellectual strength and integrity to seek the truth wherever it lies. It is in this spirit that I write you, believing that you will welcome whatever facts I may be able to provide regarding radioactive fallout from weapons testing.

I do not know what data you have utilized in studying this question, but I seriously doubt, from the evidence of your statement, that you have had access to the most recent information. Immediately after reading your statement, I sent you a copy of a speech which I gave recently regarding what we know from scientific studies on fallout radiation and its effects. I am enclosing with this letter a copy of a paper which I am presenting on April 26 before the American Physical Society. I hope these documents will be of use to you. They demonstrate that an intensive effort has been made to calculate on theoretical grounds, and to determine from sample collections, the actual levels of radioactivity in the soil, in water, in food products, and in human bodies as a result of weapons tests.

If you have gained the impression that United States official statements do not take into account the possible hazard from internal radiation—and I fear from your statement that you have—I hasten to assure you that this is not the case. Government statements have dealt extensively with this matter. It has likewise been considered at length in a report prepared by scores of eminent scien-

tists for the National Academy of Sciences, and in England by the British Medical Research Council, both reports appearing in June of last year.

Particularly since the summer of 1953, the Atomic Energy Commission has conducted an intensive study of worldwide fallout which has revealed most of the information now available on this subject. These studies have included analysis of soil, plants, foods and other materials from many parts of the world. The United States Government has furnished this information without reserve to the United Nations Scientific Committee on Atomic Radiation, which was established at the recommendation of the United States and which has studied data provided by other countries.

Although there are some differences in the findings of scientists in this country and abroad, there is general agreement upon the approximate magnitude of the fallout and the rate at which it is descending from the stratosphere. Perhaps there is less agreement about the magnitude of the physiological effects which can be expected to result from fallout radiation. Nevertheless, it is very generally agreed, among those who have studied the question, that the radiation exposures from fallout are very much smaller than those which would be required to produce observable effects in the population. The U. S. Government agencies have been continuously concerned with maintaining this condition of very small test radiation hazard and have never neglected study and action to reduce it.

I do not mean to say that there is no risk at all. What I should like to demonstrate to you is that the risk is extremely small compared with other risks which persons everywhere take as a normal part of their lives. At the same time, I ask you to weigh this risk against what I believe would be the far greater risk—to freedom-loving people everywhere in the world—of not maintaining our defenses against the totalitarian forces at large in the world until such time as safeguarded disarmament may be achieved. Of course, a workable, safeguarded system of international disarmament is a paramount objective of the United States Government, and one which we must work for and hope and pray will be achieved.

To go into more detail on the question of risk from worldwide radioactive fallout, there are two possible hazards. The first is the genetic hazard due to radiation of the reproductive organs by penetrating gamma radiation, and the second is the hazard due to the irradiation of the bones by assimilated strontium-90, taken up largely through food. These two possible hazards should not be confused; there is no reason to fear genetic hazard from strontium-90, since it accumulates in the bones and does not appreciably irradiate the reproductive organs.

In order to understand the degree of these hazards, it is necessary to compare the amount of radiation dosage received from fallout with the amount of radiation dosage normally received by all living things because of the natural radioactivity in the environment. In this way, it is possible to put the hazards from weapons testing into the context of normal human experience.

When this kind of comparison is made, it becomes apparent that we all carry in our bodes, and have in our surroundings, amounts of radioactivity very much larger than those derived from radioactive fallout.

Cosmic rays, which come from outer space, have their radiation effect progressively diluted as they pass through the atmosphere. Thus, a person living at an altitude of about one mile above sea level receives a dosage of cosmic rays approaching double that of a person who lives at sea level. There are other variations in the natural "background" dosages. For example, people living in certain localities of uranium or thorium mineralization will receive much more radiation than the average, and their ancestors have received these much higher doses over centuries in many parts of the world. Living in a brick house, rather than in a wooden house, will, with certain kinds of bricks in certain parts of the world, increase radiation exposure many times over that from test fallout.

The additional radiation dosages which persons receive from fallout are small compared to these natural dosages and even the variations in the natural dosages. To be specific, the dosage to new bone as in children which results from strontium-90 at present is about the same as the additional dosage which a resident at sea level would receive from cosmic rays if he moved from a beach to the top of a hill a few hundred feet high.

There is no question that excessive dosages of radioactive strontium can cause bone cancer and leukemia in animals, so we should not casually dismiss

the possibilitiy of harmful results from test fallout. However, keeping in mind that populations are exposed to natural radiations considerably greater than the fallout dosages, we can attempt to determine whether these have caused any detectable effects. We can examine, for example, whether there is any obvious increase in the rate of occurrence of bone cancer and leukemia in populations living at higher altitudes or in regions of uranium mineralization, etc.

Examination of available records does not disclose any such effects. However, vital statistics have not always been carefully kept, and further studies are being carried on under the aegis of the United Nations Committee to determine whether any such effects can be detected. One fact is apparent, however—it certainly is not our normal experience that people can appreciably increase the occurrence of these dread diseases by moving to a higher altitude or by moving from a sedimentary soil, where the uranium content is low, to an igneous or granitic surface, where the uranium content is very much higher, or by moving from a wooden to a brick or concrete house.

Another way of evaluating the possible risk from strontium-90 in fallout is through comparison with the permissible concentration of strontium-90 recommended by authoritative groups. The permissible amount of strontium-90 for atomic energy workers in the United States is about 2,000 times the present strontium-90 content of new bone in the United States resulting from fallout. (Strontium-90 concentrations in the rest of the world are generally lower than those in the United States.) Authoritative groups have recommended that, on grounds of general prudence, the permissible limit for whole populations be one-tenth of that for atomic energy workers. On this basis, the present level for new bone, that is, in children, in the United States is somewhat less than one percent of the maximum permissible concentration for the population.

Perhaps a word of explanation should be given regarding these maximum permissible concentrations. As you know, scientists do not speak of "risks" or "hazards" in the sense that the words ordinarily are used. They try to measure possibilities almost to the limits of the finite; therefore, "risk" includes the possibility of effects far beyond the range of the probable or detectable. The maximum permissible concentrations are not safety limits, rather, they indicate that at considerably larger concentrations, perhaps tenfold greater, there would be definitely detectable effects.

So far, I have been discussing principally the possible risks from radioactive strontium. Radioactive fallout includes other materials which do not accumulate inside the body, but do not emit penetrating radiation which can irriadiate the sex organs and other parts of the whole body from the outside. Such radiations can produce genetic mutations.

Again, in evaluating the possibility of genetic effects from fallout, we should try to compare it with normal experience. The external dosages from fallout, that is, those which might cause genetic effects, have averaged between one and five thousandths of one roentgen per year in the United States during the last three or four years. This figure should be compared with a normal dosage of 150 thousandths of one roentgen per year from cosmic rays and natural radioactive materials in the environment. In other words, the external fallout radiation has been from 0.7 percent to about three percent of the natural radiation exposure.

As another example, in certain countries of the world a brick house might easily have enough natural radioactive material in the walls to give up to 40 thousandths of a roentgen more exposure per year than a wooden house and a concrete block house gives about 100 thousandths of a roentgen more annually. These dosages range between 8 and 100 times the dosage due to test fallout.

Obviously, the genetic effect of fallout radiation must be very small compared with the genetic effect of natural radiation.

As you pointed out in your statement, radioactivity from tests which already have been held is present in the stratosphere, from which it will descend for years to come. The radioactivity of this material constantly is decreasing through normal radioactive decay. The tiny radioactive particles fall so slowly from the stratosphere that the continuing fallout in the United States just about compensates for the radioactive decay of the radiostrontium already deposited. Therefore, the present level of radiostrontium in the soil is about as much as we shall ever have from tests already fired.

Continued testing would not increase radioactivity on a straight additive basis, since an equilibrium would be established between the added radioactivity and radioactive decay. If tests were to continue until 1983 at the rate of the past five

years, levels in the United States would be expected to reach about four times their present values. Levels about six times the present ones would be reached by the year 2011 if testing were to continue for that long a time.

I hope that I have provided enough information to demonstrate that the risk from nuclear testing at the present rate is small. Of course, a great amount of more detailed information is available, and I shall be glad to supply it to you if you wish. No scientist contends that there is no risk. We accept risk as payment for our pleasures, our comforts, and our material progress. Here the choice seems much clearer—the terrible risk of abandoning the defense effort which is so essential under present conditions to the survival of the Free World against the small controlled risk from weapons testing.

Sincerely yours,

/s/ W. F. LIBBY.

PERE LORENTZ ASSOCIATES,
New York City, N. Y., April 1, 1957.

The Honorable CLINTON P. ANDERSON,
United States Senate,
Washington, D. C.

MY DEAR SENATOR ANDERSON: As you know, almost a year ago the National Academy of Sciences issued Summary Reports on the Biological Effects of Atomic Radiation, in collaboration with the National Research Council and financed by a grant of one million dollars from the Rockefeller Foundation. The reports were signed by one hundred and ten doctors and scientists, grouped into six committees. I am at present engaged in writing a book about atomic energy, for which I am under contract, and I am writing to you to inquire as to whether your committee has knowledge of any action or any research that may have been in accordance with the recommendations made by the six committees. That is, has the Congress appropriated funds for the use of any government agency in instituting studies as have been recommended, or has the Congress directed the AEC to institute studies in accordance with the major recommendations of the National Academy Committees.

1. The Committee on Genetics recommended, among other things, "that, in view of the fact that total accumulated dose is the genetically important figure, steps be taken to institute a national system of radiation exposure record-keeping, under which there would be maintained for every individual a complete history of his total record of exposure to X-rays, and to all other gamma radiation. This will impose minor burdens on all individuals of our society, but it will, as a compensation, be a real protection to them."

"That every effort be made to assign to tasks involving higher radiation exposures individuals who, for age or other reasons, are unlikely thereafter to have additional offspring. Again it is recognized that such a procedure will introduce complications and difficulties, but this committee is convinced that society should begin to modify its procedures to meet inevitable new conditions * * *"

"One important lesson which results from this study is the following: The present state of advance in atomic and nuclear physics on the one hand, and in genetics on the other hand, are seriously out of balance. We badly need to know much more about genetics—about all kinds and all levels of genetics, from the most fundamental research on various lowly forms of life to human radiation genetics. This requires serious contributions of time, of brains, and of money. Although brains and time are more important than money, the latter is also essential; and our society should take prompt steps to see to it that the support of research in genetics is substantially expanded and that it is stabilized."

Has any agency of the government undertaken research in genetics on a substantial scale, as was recommended?

2. The Committee of Pathologic Effects noted that "The increasing contamination of the atmosphere with potential carcinogens, the widespread use of many new and powerful drugs in medicine and chemical agents in industry, emphasize the need for vigilance over the entire environment."

Have there been any directed studies by your committee or any other committee of the Congress, to any United States agency to examine the "entire environment" of a community, or a city, or a rural area, or of any group of controlled individuals, whereby an analyses of the total pollution and of the total contamination might be made?

3. The Committee on Meteorological Aspects stated among other things that "the operation of any significant fraction of the earth's nuclear reactors without proper safeguards would be of concern to all" * * * The report stated further that "it should be pointed out that the release of a hazardous substance by any country may affect other countries—particularly in the same latitude belt; international control to establish and maintain high standards of safe plant operation is essential". This committee also remarked that "as additional safety factors, meteorological research to locate plants in areas where unexpected releases will do the least damage is desirable." * * *

Has your committee or any other body in Congress directed any agency of the government to take steps toward establishing any sort of international control of nuclear reactor plants? Has any scientific organization satisfactorily refuted the recommendation of the Meteorological Committee? If no, why has the AEC issued licenses for the construction of large scale reactors near New York City, Chicago, Detroit and Pittsburgh, if it is desirable "to locate plants in areas where unexpected releases will do the least damage"?

4. The Committee on Oceanography and Fisheries. This committee indicated more than any other, urgent and immediate need for world studies. In their Conclusions and Recommendations they stated, "Within the foreseeable future the problem of disposal of atomic wastes from nuclear fission power plants will greatly overshadow the present problems posed by the dispersal of radioactive materials from weapon tests. It may be convenient and perhaps necessary to dispose of some of these industrial wastes in the oceans. Sufficient knowledge is not now available to predict the efforts of such disposal on man's use of other resources of the sea."

"We are confident that the necessary knowledge can be obtained through an adequate and long-range program of research on the physics, chemistry, and geology of the sea and on the biology of marine organisms. Such a program would involve both field and laboratory experiments with radioactive material as well as the use of other techniques for oceanographic research. Although some research is already underway, the level of effort is too low. Far more important, much of the present research is too short-range in character, directed towards *ad hoc* solutions of immediate engineering problems, and as a result produces limited knowledge rather than the broad understanding upon which lasting solutions can be based."

"We recommend that in future weapons tests there should be a serious effort to obtain the maximum of purely scientific information about the ocean, the atmosphere, and marine organisms. This requires, in our opinion, the following steps: (1) In the planning stage committees of disinterested scientists should be consulted and their recommendations followed, (2) funds should be made available for scientific studies unrelated to the character of the weapons themselves, and (3) the recommended scientific program should be supported and carried out independently of the military program rather than on a 'not to interfere' basis."

"Ignorance and emotionalism characterize much of the discussion of the effects of large amounts of radioactivity on the oceans and the fisheries. Our present knowledge should be sufficient to dispel much of the over-confidence on the one hand and the fear on the other that have characterized discussion both within the Government and among the general public. In our opinion, benefits would result from a considerable relaxation of secrecy in a serious attempt to spread knowledge and understanding throughout the population."

"Sea disposal of radioactive waste materials, if carried out in a limited, experimental, controlled fashion, can provide some of the information required to evaluate the possibilities of, and limitations on, this method of disposal. Very careful regulation and evaluation of such operations will, however, be required. We, therefore, recommend that a national agency, with adequate authority, financial support, and technical staff, regulate and maintain records of such disposal, and that continuing scientific and engineering studies be made of the resulting effects in the sea."

"We recommend that a National Academy of Sciences-National Research Council committee on atomic radiation in relation to oceanography and fisheries be established on a continuing basis to collect and evaluate information and to plan and coordinate scientific research."

"Studies of the ocean and the atmosphere are more costly in time than in money and time is already late to begin certain important studies. The problems involved cannot be attacked quickly or even in many cases, directly. The pollu-

tion problems of the past and present, though serious, are not irremediable. The atomic waste problem, if allowed to get out of hand, might result in a profound, irrecoverable loss. We, therefore, plead with all urgency for immediate intensification and redirection of scientific effort on a world-wide basis towards building the structure of understanding that will be necessary in the future. This structure cannot be completed in a few years; decades of effort will be necessary and mankind will be fortunate if the required knowlerge is available at the time when the practical engineering problems have to be faced."

"The world-girdling oceans cannot be separated into isolated parts. What happens at any one point in the sea ultimately affects the waters everywhere. Moreover, the oceans are international. No man and no nation can claim the exclusive ownership of the resources of the sea. The problem of the disposal of radioactive wastes, with its potential hazard to human use of marine resources, is thus an international one. In certain countries with small land areas and large populations, marine disposal of fission products may be essential to the economic development of atomic energy. We, therefore, recommend: (1) that cognizant international agencies formulate as soon as possible conventions for the safe disposal of atomic wastes at sea, based on existing scientific knowledge; and (2) that the nations be urged to collaborate in studies of the oceans and their contained organisms, with the objective of developing comparatively safe means of oceanic disposal of the very large quantities of radioactive wastes that may be expected in the future."

"Because of the increasing radioactive contamination of the sea and the atmosphere, many of the necessary experiments will not be possible after another 10 or 20 years. The recommended international scientific effort should be developed on an urgent basis."

Has your committee or any other committee in the Congress considered the establishment of a national agency such as is recommended in paragraph 6? Has any agency of the United States Government taken steps to meet with other government bodies in an effort to "formulate as soon as possible, conventions for the safe disposal of atomic wastes at sea"? Has your committee directed any agency of the government to collaborate with other nations "in studies of the oceans and their contained organism, with the objective of developing comparatively safe means of oceanic disposal of the very large quantities of radioactive wastes that may be expected in the future"?

5. The Committee on Agriculture and Food Supplies. This committee recommended, among other things, that "The Committee therefore urgently recommends that appropriate experimentation be immediately activated to provide specific information about possible total or cumulative biological effects that might follow the ingestion of such foods. It further urges that the planning of such experiments be broadly based, and that the development of the experimental designs and details of their subsequent execution be most carefully considered in order that the emerging data will be acceptable as a basis for the crucial decisions that ultimately will have to be taken, and directly of value to the regulatory agencies charged with the protection of the public interest * * *."

"Research activities might appropriately be carried out on areas near weapons test sites where substantially greater changes in background would be anticipated. The distribution in the environment, in the soil at various depths, in the vegetation, in the wildlife, in the streams, etc., would all be pertinent. The rate of accumulation in soil as affected by land use ought to be studied. Forested land, range land, rotation grassland, and plowland, irrigated and non-irrigated, may each present a different situation. It is possible that certain of the State Agricultural Experiment Stations might be in a position to undertake limited surveys of this type on areas likely to be under their control for some considerable time in the future."

Has your committee knowledge of any experimentation being undertaken by any agency of the government "to provide specific information about possible total or cumulative biological effects that might follow the ingestion of such foods"? Also has your committee recommended funds for the Department of Agriculture, or any other government agency, to be used in undertaking the research activities in areas near weapons test sites, as recommended?

6. The Committee on Disposal and Dispersal of Radioactive Wastes—This Committee listed the following items that were considered important enough to require further study:

(1) Geophysical and geochemical aspects of ultimate disposal of highly radioactive wastes.

(2) Site selection for various nuclear facilities, particularly chemical processing plants and their location with respect to suitable waste disposal areas.

(3) Transportation of highly radioactive materials.

(4) Relationship of introduction and development of nuclear facilities to basic public health, social and economic situations extant or resulting from such development.

The Committee on Oceanography would seem to have covered this field thoroughly.

In summary, although the 1954 Atomic Energy Commission Act would seem to vest all responsibilities concerning atomic matter in the hands of the AEC, there seems to be now a public health problem which involves the land, the oceans, the air and agricultural products, and I would be deeply grateful if your committee could provide me with any record of research projects that might have been approved by the Congress, and that might be carrying out some of the recommendations of the before-mentioned committees of the National Academy of Sciences.

Very truly yours,

PARE LORENTZ.

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington 25, D. C., April 9, 1957.

Mr. JAMES T. RAMEY,
Executive Director, Joint Committee on Atomic Energy,
Congress of the United States.

DEAR MR. RAMEY: This is to acknowledge Dave Toll's letter of April 6, 1957, enclosing a letter from Pare Lorentz Associates, Inc., addressed to Senator Anderson dated April 1, 1957.

Be assured the letter will receive our prompt attention, and a reply will be forwarded.

Sincerely yours,

BRYAN F. LAPLANTE,
Special Assistant to the General Manager (Congressional).

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington 25, D. C., May 22, 1957.

Mr. JAMES T. RAMEY,
Executive Director, Joint Committee on Atomic Energy,
Congress of the United States.

DEAR MR. RAMEY: This is in further reply to Mr. David Toll's letter of April 6, 1957, requesting the Commission's comments on a letter dated April 1, 1957, Senator Anderson had received from Pare Lorentz Associates, Inc. To the extent we are knowledgeable we are happy to provide answers or comments in connection with the questions raised by Mr. Lorentz:

1. Congress has not appropriated funds specifically directed toward the establishment of a national system of radiation exposure record keeping, nor has it directed the Atomic Energy Commission or any other agency to institute such a system. Some study has been given this subject by the Commission's Division of Biology and Medicine, by the Advisory Committee for Biology and Medicine and by a number of national and international bodies including the National Committee on Radiation Protection, the International Commission on Radiation Protection, the United Nations Scientific Committee on the Effects of Atomic Radiation, and the World Health Organization. A complete system of record keeping would involve either (a) the necessity for each individual in the population to carry a card upon which would be entered the radiation exposure received at each medical examination and treatment and the radiation received from occupational exposures, or (b) the reporting of each such exposure to a central bureau by radiologists and health physicists, together with some means of accurately identifying the individual's exposure. The first of these methods, to be successful, would require the cooperation of all individuals in the population. Either method would be unwieldy. As a result of increasing interest in radiation exposures, radiologists are voluntarily making more accurate determinations of exposures actually given during examinations and treatments and are keeping better records of such exposures. A number of studies to obtain more accurate estimates of average exposures incidental to medical diagnosis and treatment in representative hospitals are under way. Records of occupational exposure are kept on individuals employed in AEC establishments. Persons

using sources of radiation licensed by the AEC are also required to keep records of personal exposure to demonstrate compliance with regulations covering such use. It may be expected that most private industries employing persons subject to radiation exposure will maintain records of exposure for purposes of legal protection.

As noted in the Commission's 21st Semi-annual Report to Congress, research in genetics was initiated by the Manhattan Project and has been supported on a greatly expanded scale by the AEC. Research in genetics is also supported independently by the National Science Foundation and by the National Institute of Health. There has been no large increase in the amount of Government-supported genetic research as a result of NAS–NRC report. While more rapid expansion could take place with a greater expenditure of Government funds, there is also a need for a large number of competent scientists interested in carrying out the studies.

2. The Congress is well aware of the problem of the increasing contamination of the atmosphere and its possible relationship to the increase in lung cancer, and has set up under the Department of Health, Education, and Welfare the Interdepartmental Committee on Community Air Pollution.

This Committee has official representation from the Department of Agriculture, the U. S. Atomic Energy Commission, the Department of Commerce, the Department of Defense, the Department of Health, Education, and Welfare, the Department of Interior, and the National Science Foundation.

The Committee holds regular meetings in order to discuss in detail the problems connected with air pollution. In addition, there is a budget supplied to DHEW through Congressional appropriation which is being used for research and survey work in connection with air pollution.

3. Some degree of international control of nuclear reactor plants is to be expected from the International Atomic Energy Agency. Such controls will apply only to reactors built and operated by members of the agency.

The Atomic Energy Commission has issued "construction permits" for power reactors at Indian Point, New York, approximately 24 miles from New York City, at Dresden, Illinois, approximately 50 miles from Chicago, and at Lagoona Beach, Michigan, approximately 30 miles from Detroit. In addition, the Commission is constructing a Pressurized Water Reactor at Shippingport, Pennsylvania, approximately 25 miles from Pittsburgh. In the case of the Pressurized Water Reactor the AEC undertook the construction of this reactor after a careful survey of the conditions at the site and the type of reactor which was to be built. It was concluded that the public would be adequately protected by the type of reactor selected and also by providing for a tight container around the reactor to contain radioactive materials in the unlikely event of a reactor accident severe enough to disrupt the heavily constructed primary reactor container system. In the case of the three privately-owned reactors, the construction permit was issued for these only after careful consideration of the general type of reactor plan and with provisos that these too would be surrounded by safety containers to protect the public in the case of an accident. As you are no doubt aware there is now in progress a hearing on the Power Reactor Development Corporation Lagoona Beach reactor.

4. A scientific committee initiated and financed by the Atomic Energy Commission, Office of Naval Research, Fish and Wildlife Service, and with the participation of the National Science Foundation has recently been established by the National Academy of Sciences. The scope of the committee is to:

1. Survey and evaluate the state of knowledge and activity in the various branches of oceanography and recommend broad programs and specific tasks that might be undertaken to advance the oceanographic sciences.
2. Facilitate joint planning among those responsible for the support and conduct of research in oceanography.
3. Stimulate coordinated studies on problems which overlap the traditional boundaries of specialized research, and identify opportunities for the application of knowledge and theory from other sciences and disciplines to problems of oceanography.
4. Develop a focal point for the compilation, exchange, and dissemination of information, and promote the efficient utilization of research personnel and facilities.
5. Provide forums for the discussion of problems of concern to all branches of oceanography (such as manpower, ship and laboratory facilities, instrumentation, data processing, etc.) and foster the search for solutions to those problems.

6. Provide for appropriate scientific representation in international meetings and furnish counsel regarding United States national interests in matters pertaining to the ocean.

It is anticipated that this committee will be a working group, as well as one to give advice which will enable the participating agencies to accomplish objectives even beyond those mentioned in Mr. Lorentz' question.

The Oceanographic Panel of the International Geophysical Year (U. S. participation financed through N. S. F.) is also actively studying phases of physical oceanography which will contribute to knowledge necessary for sea disposal of atomic wastes.

A number of research projects are supported by the Atomic Energy Commission which have a bearing on the biological effects of radioactive wastes which might be disposed of at sea. These are:

1. Woods Hole Oceanographic Institution "Biological and Radiochemical Studies of Coastal Plankton Populations"
2. Marine Biological Laboratory, Woods Hole "Studies on the Physiology of Marine Organisms Using Radiosotopes"
3. Applied Fisheries Laboratory, U. of Washington Radiobiological Surveys in the Vicinity of the Eniwetok Test Site. (This is not the exact title of any one project but is the general subject of their research.)
4. Naval Radiological Defense Laboratory, San Francisco "Study of Soil, Water, Flora and Fauna of the Marshall Islands"
5. The Fish and Wildlife Service, Beaufort, N. C. "The Accumulation of Fission Products by Marine Fish and Shellfish
6. University of Hawaii, Hawaii Marine Laboratory "Radioisotope Uptake in Marine Organisms with Special Reference to the Passage of Such Isotopes As Are Liberated from Atomic Weapons Through Food Chains Leading to Organisms Utilized as Food by Man"
7. University of Hawaii, Hawaii Marine Laboratory "Management of the Eniwetok Marine Biological Laboratory"
8. Stanford University, George Vanderbilt Foundation "Marine Biological Survey of Western Pacific"

In reference to question (6), geophysical and geochemical aspects of ultimate disposal of high-level wastes are being actively considered by a number of research groups and projects. These are described on pages 159–160 of the 21st Semi-annual Report to Congress (January 1957). In addition, a new project now in effect with Scripps Oceanographic Institute is concerned with studies of the circulation of elements of biological importance, and the dynamics of their dilution and concentration, throughout the entire geophysical environment.

5. The recommendation of the Committee on Agriculture and Food Supplies quoted here applies primarily to fallout from nuclear weapons, particularly strontium 90. The AEC has for several years been engaged in extensive research programs covering not only the biological effects that might follow the ingestion of such foods but also studies of the distribution of strontium 90 in the environment, in the soil at various depths, in the vegetation, and in animals under a variety of environmental conditions. The U. S. Department of Agriculture in certain state agricultural experiment stations has cooperated in some of these studies. The AEC has conducted studies of uptake of radioactive materials by plants and wild animals near the weapons test sites but has not found a favorable environmental condition for establishing a large scale agricultural research activity near the test site.

6. In this portion of his letter, after enumerating items considered by the Committee on Disposal and Dispersal of Radioactive Wastes as sufficiently important to require further study, we assume Mr. Lorentz intended to say that that Committee would seem to have covered this field thoroughly. These tiems have been under intensive study by the AEC but may be expected to represent basic problems in the development of nuclear energy for some years to come.

Geophysical and geochemical aspects of the disposal of highly radioactive wastes in soil have been under intensive study for more than ten years at our Hanford plant and to a somewhat lesser extent at Idaho Falls and at Oak Ridge. In these studies we have had the cooperation of the U. S. Geological Survey and the Earth Sciences Division of the National Academy of Sciences.

Studies of site selection for chemical processing plants with respect to suitable waste disposal areas are of course not made independently of the studies mentioned above.

Responsibility for the safe transportation of highly radioactive material is vested in the Department of Commerce, the Civil Aeronautics Board, and the

U. S. Coast Guard. The AEC has worked closely with these agencies and is presently engaged in studies designed to minimize hazards incidental to future transportation requirements of the nuclear energy industry.

The relationship of nuclear facilities to basic public health, social and economic situations is a broad question upon which many studies are being brought to bear; for example, the Joint Committee on Atomic Energy established in 1955 a panel of prominent persons from many professions to study the impact of the peaceful uses of atomic energy. The report of this panel was published in two volumes in January of 1956. The Joint Committee on Atomic Energy and other committees of Congress such as the Committee on Interstate and Foreign Commerce, House of Representatives; the Committee on Government Operations, House of Representatives; and the Senate Committee on Foreign Relations, to mention but a few, haxe from time to time held hearings on various aspects of this subject.

This subject has also been of interest to many other groups including the Atomic Energy Commission, the U. S. Public Health Service, the National Academy of Sciences, the World Health Organization, the United Nations, the International Labor Organization, the Rockefeller Foundation, state health organizations and many other groups. These interests are developing our knowledge of the relationship of nuclear facilities to public health more rapidly than any other aspect of public health. Social and economic impacts arise more spontaneously from economic interests.

In reference to the last paragraph of Mr. Lorentz' letter, we believe that he is in error in assuming that Congress approves individual research projects rather than approving budgets for research programs.

We trust the information supplied above will be of assistance to you in replying to Mr. Lorentz' letter. Mr. Lorentz' letter is being returned to you.

Sincerely yours,

R. W. Cook, *Deputy General Manager.*

Enclosure: Letter dated April 1, 1957.

United States Atomic Energy Commission,
Washington 25, D. C., July 9, 1957.

Hon. Carl T. Durham,
Chairman, Joint Committee on Atomic Energy,
Congress of the United States.

Dear Mr. Durham: The Atomic Energy Commission approved for unclassified publication the rewritten handbook entitled "The Effects of Nuclear Weapons," copies of which are attached.

New information has been developed since "The Effects of Atomic Weapons" was last published. At the request and with the assistance of the AEC, the Armed Forces Special Weapons Project of the Department of Defense prepared the new edition. The services of Dr. Samuel Glasstone were secured as editor and the material was reviewed by the Federal Civil Defense Administration, by cognizant contractors of the AEC, and by the Department of Defense.

"The Effects of Nuclear Weapons" updates information appearing in the 1950 handbook and the February 15, 1955 release on Effects of High-Yield Weapons. It includes an expanded coverage on fallout referred to in our letter to Senator Anderson dated November 16, 1956. It bears a foreword signed by the Secretary of Defense, Charles E. Wilson, for the Department of Defense, Administrator Val Peterson for the FCDA, and the undersigned for the AEC. The handbook has been printed by the Government Printing Office which also prepared the advanced page-proof copies of those chapters dealing with fallout which were furnished to Representative Holifield of your Committee for the recent hearings on fallout.

Approval of the text by the Commission was unanimous except with respect to a number of points on which Commissioner Murray desired wording or action differing from that believed most appropriate or factual by the other members of the Commission. Commissioner Murray asked that you be informed of the points concerned; these are listed in the second attachment and comment thereon will be provided if requested.

These copies of the Effects of Nuclear Weapons handbook are being sent in advance of the release for the book which is set for publication in afternoon newspapers of Friday, July 12, 1957. When these copies were received it was

noted that the printer had omitted six lines from paragraph 10.24 on page 454. A correction sheet for that page has been included in each book.

Sincerely yours,

LEWIS L. STRAUSS, *Chairman.*

Attachments: As stated above.

Listed below are seven suggestions regarding "The Effects of Nuclear Weapons" handbook, which were made by Commissioner Thomas E. Murray but were not accepted by the Commission.

(1) That Chapters 9, 10, and 11 be submitted to the Advisory Committee on Biology & Medicine for review prior to approval for publication.

(2) That there be inserted in the Handbook a detailed Appendix of latest statistics and opinions relative to world-wide fallout.

(3) That in the last sentence of para. 9.49 which reads: "In fact the external radiation produced by the fallout from a weapon with a fission yield in the megaton range would be extremely small in comparison with the natural background radiation," the word "extremely" be deleted and, following the word "small," (of the order of —— percent)" be added.

(4) That in para. 9.94 of the Handbook, the words "can under some conditions" be replaced with the words "could be expected to" in the sentence which reads: "One is that the residual nuclear radiation can under some conditions represent a serious hazard at great distances from a nuclear explosion, well beyond the range of blast, shock, thermal radiation and the initial nuclear radiation."

(5) That in para. 10.1, the word "will" be used instead of "may" in the sentence which reads: "During the first few days or weeks after the detonation, the radiation levels may be high enough to represent a danger to exposed persons."

(6) That Chapter 10 entitled "World-Wide Fallout and Residual Radiation" give added coverage to the long-term strontium–90 hazard from local fallout.

(7) That in para. 11.122 the word "may" be deleted and the word "find" be changed to "finds" in the second sentence which reads: "The strontium may then find its way, mainly through milk products, into the human body."

APPENDIX 7

1. INFORMATION FURNISHED TO THE JOINT COMMITTEE BY STANLEY H. CLARK, BALTIMORE, MD.

2. INFORMATION FURNISHED TO THE JOINT COMMITTEE BY ALDEN A. POTTER, BETHESDA, MD.

8420 LOOKOUT MOUNTAIN AVENUE,
LOS ANGELES 46, CALIF.,
February 22, 1957.

Mr. GRAHAM DUSHANE,
Editor, Science,
1515 Massachusetts Avenue NW.,
Washington 5, D. C.

DEAR SIR: The article on Sr–90 content in human beings by J. L. Kulp, et al. (*Science*, February 8, 1957) may possibly be interpreted by some readers as indicating that there is little danger at this time from Sr–90. Actually that is very far from the truth.

First, consider the International Commission of Radiological Protection's recent (November 1956) reduction of the maximum permissible dose. It is now considered to be one-third (⅓) of the former value or five roentgens per year (this is occupational). Next we must always consider the occupational maximum permissible concentration separately from the maximum permissible concentration for the entire populace. The accepted reduction factor is ten—that is, the populace maximum permissible concentration should not exceed 0.5 roentgens per year (5×0.1). Applying to the Sr–90 maximum permissible concentration, we see that the populace m. p. c. is 33.3 micromicrocuries gram of calcium rather than the 1,000 micromicrocuries/gram of calcium as stated in the above-mentioned article. In the light of this value let us look at the actual Sr–90 content in human beings from different locations on the earth.

We see that the *average value* which is present in human beings today is only 1/330 of this m. p. c. and will become 1/33 (to 1/16) of the populace m. p. c. by

1970 even if no further nuclear devices are exploded! Now let us look more carefully at the concentration of Sr–90 in some of the 600 specimens analyzed. We find that 13 out of these 600 specimens actually exceed 1 micromicrocurie per gram of calcium *now* (through 1955). Or that they will have 10 to 20 micromicrocuries per gram of calcium by 1970 even though no further tests are conducted! These 13 specimens represent about 2 percent of the specimens which, if applied to the world population, is about 50,000,000 people! It will be noted from Fig. 2 of the *Science* article that these are mostly young people, 20 years or younger.

Of course, if tests are conducted at the same rate as in the past ten years, these 50,000,000 people on the surface of the earth would almost certainly be carrying an amount of Sr–90 equal to or even greater than the populace maximum permissible concentration of 33.3 micromicrocuries/gram of calcium!

Finally, we should note on page 68 of the British Research Council's Report "The Hazards To Man Of Nuclear And Allied Radiations" the following paragraph "In the light of knowledge at present available, we should feel that immediate considerations were required if the concentration (of Sr–90) in human bones showed signs of rising greatly beyond *one-hundredth* of that corresponding to the maximum permissible occupational level."

I believe that this information merits the attention of scientists and public alike.

Sincerely,

STANLEY H. CLARK,
Medical Physicist, Cedars of Lebanon Hospital, Los Angeles, Calif.
NUCLEAR DIVISION, GLENN L. MARTIN CO., BALTIMORE 3, MD., *March 15, 1957.*

10 EDGEVIEW RD., BALTIMORE 4, MARYLAND,
June 10, 1957.

Re hearings on the problem of radicactive fallout from nuclear weapons explosions.

Representative CHET HOLIFIELD,
Chairman, Subcommittee of the Joint Committee on Atomic Energy,
Room F–88, Capital Building, Washington, D. C.

DEAR MR. HOLIFIELD: I would like to submit for inclusion in the Congressional Record my feeling, as a scientist-citizen, on the subject of fallout and radiation injury. I would also like to make a suggestion for resolving the current conflict regarding testing of nuclear devices.

*   *   *   *   *   *   *

Although the medical genetic radiation exposures do exceed the fallout genetic radiation exposures in the United States, these medical exposures are knowingly received under medical supervision. This is not the case with world populace when we consider exposure from radioactive fallout. This, I feel, is the main point—we cannot expect all people to accept biological damage no matter how small or well justified we feel as a nation in exposing ourselves. *If there was general agreement* in the scientific community that there was negligible damage then such exposure would probably be acceptable.

Since the government has clearly stated that the testing of nuclear devices must be continued if our national security is not to be jeopardized, then we should look for solution to this problem which will not appreciably hinder future testing programs. The currently considered solution, that of reducing world nuclear testing to a few megatons per year, obviously does not fulfill the national security need. We must provide a means for testing nuclear devices by other nations as well as ours since world wide development of nuclear devices seems inevitable.

Probably the only solution to this problem is to make tests outside of the earth's atmosphere, i. e., in space. Today, 1957, this is technically feasible. We are now in a position to launch such nuclear devices on missiles that go well above the atmospheric envelope. I will not belittle the test difficulties; the difficulties in obtaining detailed test information; however, I feel certain the problems in this area can be resolved. Certainly the magnitude of an explosion and most of its physical characteristics can be ascertained during detonation in space.

The point I would like to stress technically is that it is completely safe from the radiological hazard standpoint to test weapons in space. (Probably the most serious hazard would be the great light intensity produced during such

tests.) What happens to the radioactive fission products when a nuclear device is exploded, for example, at 100 kilometers? Particles of 2 microns diameter (1 micron equal 1/10,000 centimeter) take a little over three years to fall to the earth's surface from this altitude. Particles of greater diameter take much less time to come to earth (at 8 microns diameter a particle takes about 130 days to reach the earth's surface). However, when the particle diameter approaches 0.2 micron or less, a new phenomena begins to occur. The light from the sun actually will push the particles out of the earth's gravitational field and, in fact, out of our solar system. Thus, for nuclear explosions in space with all particle sizes probably occurring of less than 1 micron, we have a means of cleaning up the fission products; in fact, sweeping them out into space. Surely this is the ultimate in a radiologically safe testing program. It is my hope that this means of resolving the nuclear testing debate will be investigated. If greater costs are necessary for such tests I feel certain that they will be jusified in the light of human well being.

STANLEY H. CLARK.

MEDICAL X-RAY EXPOSURES—NATIONAL VARIATIONS, INTEGRAL DOSES, ETC.

Stanley H. Clark,[1] National Biophysics Conference, Columbus, Ohio, March 4–6, 1957

INTRODUCTION

The development of the uses of atomic energy has focused greater and greater attention on the biological effects, particularly, the effects on man, of all ionizing radiation. The relative furor over the biological effects on man of radioactive fall-out has led to the refocusing of attention on existing and accepted uses of ionizing radiation. Thus, the pressures exerted due to new problems associated with uses of nuclear materials has caused a re-evaluation by a U. N. Committee, by Government bodies, scientific organizations, and individuals, of man's total radiation environment. Most, or all of these studies have indicated that of all the "men created" radiation exposures, that the uses in medicine are currently the most significant. Thus, we find that in the United States National Academy of Science Report, "The Biological Effects of Atomic Radiation" and in the British Medical Research Council Report "The Hazards to Man of Nuclear and Allied Radiations," as well as the U. N. Report on the biological effects of radiation that the medical uses of radiation have been subject to rather critical analysis.

Here is a comparison of the genetic exposure values in the medical uses of radiation as stated in the various national reports.

Values are for the first 30 years of life, and assume exposure at the same rate as at present and in the immediate past.

1. The United States as given in the National Academy of Sciences Report—3 roentgen.
2. The British, as given in the Medical Research Council Report—0.6 roentgen.
3. Sweden—Sivert's Study—0.78 roentgen.
4. Australia—Martin's Study—0.304 roentgen.
5. The U. N. Radiation Studies Committee Report—not yet completed wide range of values for different countries.

Let us now see what factors could account for these differences. (There is no particular order in this listing of factors.)

(*a*) Differences in actual number of diagnostic exposures to the populations of the various nations is one of the most important factors. Since each report sums medical exposures and then divides this lumped sum (in roentgens) by the numbers of people it makes considerable difference how many people in a populace have how many exposures. Related to the number of exposures per populace is no doubt standard of living of that particular nation.

(*b*) We might also ask, what medical uses are included in each nation's report. This calls attention to the fact that only the United States value includes therapeutic uses of X-rays (for non-malignant conditions), and uses of radioisotopes in medicine. These uses account for about 15% of the 3 roentgen value in the National Academy figure.

---

[1] Cedars of Lebanon Hospital, L. A., Consultant, Medical Division, Oak Ridge Institute of Nuclear Studies, Physicist in Radiology, Medical School, University of Southern California, now with the Glenn L. Martin Company, Nuclear Division, Baltimore 3, Maryland.

(*c*) Difference in physical factors and techniques represents a significant variable. There is considerable more control of physical factors in England and in Sweden due to a combination of factors—socialized medicine; physicists have a closer relationship with radiologists; and early recognition of radiologic physics as a profession. In the United States one finds the private physician performing fluroscopy as well as other medical specialists, dentists, chiropractors, general practitioners, etc., using X-rays with few exceptions without any consultation with a radiologic physicist.

(*d*) Another factor that should be remembered is that of the origin of the various data which were used for averaging.

1. The National Academy averaged from a few United States hospitals and medical groups, howèver, correlating this data with nationwide uses of X-ray film, etc.

2. The British used hospitals in England and Wales which have hospital physics, groups and extrapolated for the rest of England. "Gonadal doses are based primarily on 1500 patients at one large hospital, (where incidentally particular care is taken to reduce the gonad dose to the minimum)."

3. In Sweden, most of the medical uses of radiation were considered in obtaining the average genetic exposure, no doubt the study was strongly influenced by the Institute of Radiopsysique in Stockholm where procedures are under direct supervision of a physics group.

4. In Martin's report on gonadal exposure due to diagnostic uses of X-rays he uses almost all available literature and the ratios of various diagnostic procedures in one hospital are the basis for his dosage estimates.

(*e*) Additional explanatory notes on factors which contribute to the range of exposure values.

1. The British Medical Research Council figure is really not comparable with the United States value since—to quote from that report—"The value of 22% (0.6 roentgen) should be regarded as a probable lower limit rather than as an estimate. A realistic estimate of the radiation contribution from diagnostic radiology might be considerably greater than this figure". They also discuss the possible factors of 2, 3, and even 10 times this lower limit.

2. In the Australian exposure analysis, J. H. Martin states "that the turn over of patients in the X-ray diagnostic department had already doubled" at the time of his presentation (November 1954), thus his values when brought up to date would be similar to, or greater than, the values for Great Britain and Sweden.

3. S. B. Osborn, whose work forms part of the basis for the British Medical Research Council's estimate discusses in his analysis in Lancet that examinations of the hip and lumbar spine, pyelograms and pelvimetry, although they constitute only 7% of the total number of examinations, none-the-less contribute 75% of the total genetic dosage. Obviously, even minor variations in these procedures from one hospital to another, would cause significant differences in the genetic exposure values. It is interesting to note that Osborne finds that 26.3% of the total populace genetic exposure takes place during radiographic exposure of the fetus in pregnant women.

What incident values mean in terms of whole body effect—the induction of cancer and the shortening of life span.

(*a*) There is need for the integral dose concept when evaluating the medical uses of X-rays. Discussion of radiation exposure of the gonads in man and the ovaries in women require measurements in the vicinity of the reproductive organs and some correction in women for the depth of the ovaries, however in this case we are concerned with the dose at essentially a point and need not be concerned with the energy absorbed in the entire body. However when considering the hazard from diagnostic uses of X-rays which may produce damage in terms of more general tissue damage we need a different concept. The concept most appropriate for evaluating such general body injury is that of the integral dose, or absorbed dose. Specifically, the two important effects related to integral dose are increased incidence of leukemia and shortening of life span. Both are known to be associated with exposure to ionizing radiation.

It is generally agreed that shortening of life span is strongly correlated with the amount of radiation energy abosrbed in the body. This effect then can be most appropriately discussed in terms of the integral dose—a concept originating in England, I believe—it is simply the product of the number of grams or cubic centimeters irradiated, and the number of roentgens to each of the cubic centimeter volumes. Such values are calculated from either X-ray distribution patterns known as isodose patterns, or from an equation developed by Johns. (6)

In general, the first technique is the more accurate. However, for the sake of our calculations, the John's equation has been used to obtain integral doses. When isodose curves were used the area between each isodose line was measured, using a planimeter. This area was then multiplied by the field height and the average roentgen dose to that volume to give the integral dose for that segment. These segment values were then summed to give the total integral dose.

Before comparing particular values of diagnostic integral dose, let us consider how the integral dose changes with change in the energy of the incident X-ray. Assuming that field size and exit dose-rate is kept constant how does the integral dose vary?

The integral dose decreases rapidly with increased X-ray kilovoltage assuming a constant exit dose.

Now turning to the diagnostic usages, we note that the patient exit dose must be essentially constant, regardless of KV, in order to produce useful film darkening or fluoscopic light intensity. This fact when considered in conjunction with the area under the depth dose curve (which is proportional to the integral dose) shows very clearly that in the range from 40 KV to 1 mev that the integral dose decreases substantially with increase KV—thus, we might ask why the diagnostic radiologist does not continue to raise the X-ray KV and thereby reduce the integral dose. There are at least two physical reasons why this is not wholly feasible:

1. The absorption coefficients for different elements in the body, i. e. bone and tissue become so similar at higher kilovoltage that very little contrast is obtained at energies above a few hundred kilovolts.

2. The response of film (and fluorescent screens) decreases with increase KV for a given incident intensity. It becomes evident that there are optional KV values for various parts of the body, depending strongly on what information one is most interested in and the thickness of the particular body cross section. It should be remarked that if too much filtration is used at high energies there will be no reduction of integral dose. There will be a decreased incident dose, but the exit dose must be increased because of the decreased film response at higher KV.

(*b*) Since the increased incidence of cancer, primarily leukemia, is related to the actual bone dosage as well as to the integral dose, it is important to note the depth dose distribution as well in evaluating this particular hazard. It has been pointed out by Hardin Jones and others, that the dosage to long bones, i. e., the dosage to the rib cage, in chest X-rays would be particularly important insofar as the production of leukemia is concerned. It would be well to mention at this point the studies that indicate leukemia is produced by even small amounts of radiation. The best evidence is from the following studies:

1. Studies by Alice Stewart, J. Webb (7, 8), etal in England which indicate an increase in incidence of several malignant diseases including leukemia due to diagnostic X-ray exposures of the pregnant mother, particularly abdomenal exposures. Secondly studies of the survivors of the Hiroshima and Nagasaki nuclear explosives (9, 10), and thirdly exposure of radiologists (11) in the course of their professional activities.

2. Studies which indicate that somewhat larger radiation doses will produce leukemia include a series of infants treated with X-rays for thymus condition (12) and secondly X-ray therapy of anklosing spondylitis (13).

The statistical analysis of this information, as well as the linear relationship between incidence of leukemia and integrated dose is given strong support by Hardin Jones of University of California, in material which will be published in the near future. Shortening of life span as caused by radiation has been borne out by many animal experiments by the shortening of life span of radiologists, and by patients who have been treated with X-rays. Here again, the dose is linear with respect to shortening of life span. According to Jones about 10 days should be subtracted for each roentgen received of whole body radiation. The integral dose ranges from about 11,300 to 56,000 gram-roentgens for 1 roentgen incident dose. This assumes a range of X-ray energies corresponding to 2 mm of aluminum half value layer to 2 mm of copper half value layer.

In the course of using X-rays for treatment of malignant diseases, we encounter total integral dosages ranging from 1 million gram-roentgens to many million (30 or 40) gram-roentgens. This wide range is due primarily to the variation of field sizes that are used for lesions of different dimensions, and to some extent of KV. These large integral doses are actually admin-

istered in daily doses—as many as 30 or 40—in the course of a single treatment. To cite a specific example—using a 5 cm circular field at a focal skin distance of 80 cm with a half value layer of 4.00 mm of copper. The daily dose of 100 roentgen produces an integral dose of about 19,800 gram-roentgens, (remember that one roentgen incident dose produces about 11,300 gram-roentgens). Now let us compare this therapeutic value with some common diagnostic procedures. A film of the lumbar spine taken through the AP direction with the following physical factors—AP thickness 20 cm F. S. D.–71 cm, KV–70, MAS 160. Focal film distance 91.44 cm (36 in.) produces an incident dose per exposure of 4.3 roentgen. This produces an integral dose of 28,810 gram-roentgens. If we take a lateral film of the same person, we will obtain an integral dose of about 150,000 gram-roentgen or the equivalent of some 7½ times the daily integral dose cited for the above cancer therapy! Or to make another comparison, about 3 times the present maximum permissible exposure for one year period! This value would be greatly increased for a heavier person. The explanation for the larger integral dose as compared to the therapeutic dose is almost entirely due to the increase in field size. In the therapeutic example, the field size used was about 20 cm^2 in the diagnostic case it was 1487.5 cm^2 (a standard 14x17 inch film). Of course, the actual X-ray field size as determined by the cone used was circular and somewhat larger even than the area used. The integral dose in chest photofluoraphy would be about 10,000 gram-roentgens.

In fluoroscopy, the literature cites incident dosage rates from 5 to 20 roentgens per minute, depending undoubtedly on the amount of filteration and body thickness. This dosage rate would produce integral dose values from about 30,000 gram-roentgen per minute to about 120,000 gram-roentgen per minute. Frequently fluoroscopies last as much as 5 or 10 minutes which means the absorbed energy approaches that which is actually used for some small field cancer therapy. What does this mean in terms of shortening of life span and increased incidence of cancer? In the case of the shortening of life span we found that a 1 roentgen incident dose (which amounts to about 11,000 gram roentgen would decrease life span statistically by about 10 days. Thus, a single fluoroscopic examination would shorten the life span by as much as 500 days (at a 20 r per minute dosage rate). Or the lateral pelvic radiograph would shorten the average life span by about 140 days! But let us see how these figures compare with some other factors that are known to shorten life span (unpublished, Hardin Jones-University of California).

	Years
25 percent overweight group	3.6
Heart murmur	11
Rapid pulse	3.5
Varicose veins	0.2
Trace of albumin in urine	5.0
Epilepsy	20.0
Skull fracture	2.9

It should be noted that when applied to the total populace millions of man-years of life are lost due to medical radiation exposure (about 7,000,000 man-years each 30 years at the current exposure rate).

With respect to increasing the incidence of cancer particularly myeloid leukemia I would simply like to make a general statement regarding the increased incidence with respect to chest photofluography as a significant example. We find that 1r exposure per year gives a probability of 1 in 100,000 to 1: 1,000,000 of developing leukemia per average individual (7) (14) (15). This probability applied to the 15,000,000 people in the United States who have chest X-rays (in mass chest X-ray surveys) annually would amount to an increased number of cases of myelogenous leukemia; due to these chest X-ray exposures alone 15 to 150 cases per year and each year thereafter. There is in addition speculations that some individuals are genetically more asensitive to the induction of radiation leukemia. Thus a particular sub-population might have a considerably higher probability of the occurrence of radiation induced leukemia.

MISCELLANEOUS-CONCLUSION

Finally let us consider the exposure to X-ray technicians in the course of their training, there are some 40,000 in the United States. They divide up into pairs

and go through the entire radiographic series of exposures normally encountered in diagnostic X-ray work. The genetic exposure for 35 procedures amounts to about 5.6 roentgens for the male technicians and 6.3 roentgens for the female technicians. This exposure corresponds to perhaps 20 to 30 roentgens incident dose (not greater) or about 10 to 15 roentgens of whole body radiation (in terms of integral dose). Thus in this group of technicians due to their X-ray exposure during training we might expect (using a probability of 10^{-5} per roentgen per year) some four cases of radiation induced leukemia per year. The additional exposure in the course of their work is not considered. Even so, since X-ray technicians are generally young people (at the time of training) we would expect this increased incidence of radiation induced leukemia to amount to about 160 additional cases (normally one would expect about 240 cases of both lymphatic and myelogenous leukemia). Hardin Jones states that the leukemia doubling rate is about 30 roentgens full body exposure. Which would give a figure of about 120 cases of radiation induced leukemia. It is hoped that this estimate could be confirmed from actual death statistics of X-ray technicians.

REFERENCES

1. The Biological Effects of Atomic Radiation available from The National Academy of Science, 2101 Constitution Avenue, Washington 25, D. C.
2. The Hazards to Man of Nuclear and Allied Radiations available from the British Information Services, 30 Rockefeller Plaza, New York.
3. To be published in late 1958.
4. Sievert, R., (1952) "Tolerance Levels and Swedish Radiation Protection Work", in "Biological Hazards of Atomic Energy", Clarendon, Oxford, 181.
5. Martin, J. H., "Radiation Doses to Gonads in Diagnostic Radiology and Their Relation to the Long Term Genetic Hazard" Medical Journal of Australia November 12, 1956, Page 806.
6. The Physics of Radiation Therapy, H. E. Johns, 1953 page 133.
7. A. Stewart et al. The Lancet, Sept. 1, 1956 P. 447
8. Osborn, S. B., Smith, E. E., Lancet 1956 p. 949
9. Lange, R. D., Moloney, W. C., Yamanwaki, T., Blood 9: 574 (1954)
10. Moloney, W. C., and Kastenbaum, M. A., Science 121: 308 (1955)
11. March, H., Amer. Jour. Med. Sci. 220 p. 282 (1950)
12. Simpson, C. L., et al., Radiology 640 p. 840 (1955)
13. Pages 87–89 Reference (22)
14. W. M. Court Brown, Richard Doll, Lancet Jan. 12, 1956 p. 97, 98
15. Unpublished calculations
    Clark, S. H., Bulletin Atomic Scientists 12, 14 (1956)
    Martin, J. H., (see reference 5) (Martin's report contains a comprehensive reference list)
    Stanford, R. W., and Vance, J., (1955) Brit. J. Radiol. N. S., 28, 266
    Weens, H. S., Clements, J. L. and Tolan, J. H., (1954) Radiol. 62: 745

BETHESDA 14, MD., *July 5, 1957.*

Hon. W. STERLING COLE,
Joint Committee on Atomic Energy,
Washington, D. C.

MY DEAR CONGRESSMAN COLE: Through your kind services I beg to submit the enclosed "Critique of Scientific Knowledge" for entry, as the Committee may see fit, in the record of the hearings on The Nature of Radioactive Fallout and Its Effects on Man, held in May-June.

The Kremlin seems to have shifted its position, softened its opposition to "western" genetics which they have ardently rejected—until it came handy in their cold war in Japan against *our* bomb tests.

At the suggestion of the Committee Technical Advisor I am asking the following geneticists with whom I am acquainted to comment, to the committee on copies sent them.

Dr. James F. Crow, National Institute of Genetics, Mishima, Japan.
Dr. Carl C. Lindegren, Southern Illinois University, Carbondale, Ill.
Dr. Stanley H. Emerson, Atomic Energy Commission, Washington, D. C.

Very truly yours,

ALDEN POTTER.

A Critique of Scientific Knowledge

Keep that which is committed to thy trust, avoiding profane and vain babblings, and oppositions of science falsely so called (I Timothy 6.20).

The evidence adduced in these hearings on the effect of radioactive fallout on human and other life has shown that even a considerable enlargement of test explosions will not increase the incidence of radioactivity materially beyond what all forms of life have experienced from time immemorial.

A prime consideration is the growing belief that most of the radioactivity that is injurious can be eliminated from military weapons by tests now being planned and conducted, so that possible danger to future generations in case of war will be correspondingly reduced. The larger, fusion bombs are less radioactive in proportion to the success that may be attained in eliminating fission, as in "triggering" the explosion. This may explain the Kremlin's current readiness to suspend tests so they can catch up with our "classified" progress toward clean bombs, while shedding crocodile tears over the genetic horror which they have subtly helped "western" geneticists implant at the Japanese National Institute of Genetics at Mishima.

The attempt to make medical science "objective" has turned on statistical theory and practice which is leading to a great deal of confusion as to the applicability of degrees of probability thus determined. This source of misinformation is by no means confined to research in radioactive fallout as presented in these hearings, but extends over the whole field of science. It will therefore be necessary to attempt some clarification of these basic disputes as they affect the argument over whether observed effects may be linearly extrapolated (whether the extent of biological injury by radioactivity is directly proportional to the extent of such activity, however small), as against the existence of a "threshold" or range of radiation that is either beneficial or at worst not injurious to any life. This critique will contend that thresholds are the rule, not the exception, in nature. Moreover, insofar as a threshold is biologically determined, we shall argue that it cannot be a fixed factor in the survival of any species because of adaptive changes in the organisms involved.

Senator Anderson has cited such an adaptation in the case of the resistance of insects (flies) to DDT; and there are many other such cases in recent experience with insecticides. This factor of adaptive change has been so widespread in medical research that in 1954 the University of Pennsylvania's medical school cooperated with Naval Research in organizing a symposium on drug resistance at the Statler Hotel in Washington; and thereby hangs a tale. The honor guest and speaker at dinner was Dr. Cecil P. Martin of McGill University in Montreal, Canada. He was called upon to speak because of an essay, "A Non-Geneticist Looks at Evolution," published in the American Scientist shortly before, in which he assailed "Western" genetics on the ground that it has produced an untenable theory of evolution, that is, the mutation-selection theory.

This view has gathered some support even among geneticists; witness the recent note (Science, May 10, 1957) of Lindegren and Braun criticizing the views of Dr. George Beadle of the California Institute of Technology as given in his presidential address to the American Association for the Advancement of Science, for his too confident proposal to call nucleic acid a "living" molecule because it is a "carrier" of genes in heredity. The essence of this question lies in the way the species pattern is carried. Is it by a template—a minute replica as Dr. Glass contends—or is the system like our cultural information system, linguistic and therefore *meta*physically informational like an engineer's handbook?

It should be unnecessary to record the views of Dr. Martin here for they have been available in a treatise published last year by the eminent medical publishing house of C. C. Thomas of Springfield, Illinois, entitled "Psychology, Evolution, and Sex." But the evidence adduced in these hearings by the panel of geneticists from the National Academy of Science has studiously omitted any reference whatever to these conflicting opinions, quite as they have been omitted from any and all proceedings and reports of the Academy and, as to the Martin treatise, also from the pages of Science where the book has not been reviewed after almost a year since its publication.

It will be necessary, therefore, to try to clarify the relation between the Darwinian views of Dr. Martin and some few geneticists whom our Academy has deliberately ignored, and those set forth by the Soviet scientists at a recent conference in Japan on genetics where they spoke in the terms of Mendelian theory for the first time since the days of Trofim Lysenko. Is it indeed a mere coincidence that the mechanistic materialism of our Academy with its pretense of

"objectivity" now serves in furthering the curtailment of bomb testing just as the Russian cold war is so intensifying its peace offensive as to produce riots before the American embassy in Tokyo? Are we not ourselves so supersaturated with the prevalent anti-Darwinian philosophy that we refuse to examine such a scholarly treatise as that of Cecil Martin which counters the false alternatives of an alleged conflict between military and genetic security? Only the mutation-selection doctrine with its elmination of competitive stratagems in organic evolution can serve the communistic pacifism of Soviet propaganda; and it is with a sudden acceptance of this idea that they carry the war into Tokyo!

A clarification of these issues on an honestly open-minded, objective basis entails a thorough reconsideration of the basic tenets of *scientific empiricism* which define "the scientific method" ("Scientism" and "positivism" are other terms for this "unity of science" philosophy) as purely inductive. Such a reconsideration is supported by the rising tide of literature indicating the imminence of a conceptual revolution in science. This deductive revolt is renewing the conflict between science and the anthropocentric bigotry which gave rise to the Scopes trial in Tennessee instigated by William Jennings Bryan. This court case sought to ban the teaching of a theory of evolution that does not admit the uniqueness of man which is today propounded by the chemical theory of genetics and the origin of life because, very obviously, *human affairs are certainly not chemically explicable.* Man does not live by bread alone; *but neither does any other form of life.*

The bigotry of this anthropomorphic theology has unmistakably injected the pontifical edict that an "emergent" evolutionary theory may be anthropomorphic upward *to* divinity, but not upward *from* monkeys, in its "extrapolations." Our Fundamentalists thus create a self-idolatry—a God in the image of man; an authoritarian threshold that may not be crossed even by the authorities. This prideful conceit that man is peculiarly unique not only has no support in the Christian Gospels but it seems to beget a peculiar predilection for statistical extrapolation, while banning biological analogy, as scientific evidence.

Mice are not men; so science must perforce extrapolate averages (statistical data) as a "first approximation" or "educated guess," the while indulging in crash projects, not to test empiricism against an alternative postulate, but to "prove" that extrapolations (predictions) are correct and so justify, by pragmatic "proof", the public policy already adopted in "playing safe" with probabilities. This is called "verifying the hypothesis" with "observational evidence." Its successes are proclaimed from the housetops; its failures suppressed like a Nobel Prize gone sour.

In assailing this materialistic biology it would be well to cite further evidence, from outside these hearings, that it does exist and is, indeed, censoriously entrenched in scientific literature and training for "academic freedom" in research. The popularity among biological empirics of a small book written by Prof. Erwin Schrödinger, author of the principal equation of the so-called "quantum mechanics" of atomic physics, "What Is Life?", with its attempt to forecast a physical answer, is attested not only by numerous reprints (including a paperbound, popular edition) but also by the widespread opinion, urged most recently by Linus Pauling in a lecture at the National Institutes of Health in Bethesda, that the genesis and genetics of life can and ultimately must be defined in purely chemical terms. Any other outcome would, *ex cathedra*, be a betrayal of progress in "science."

Since these hearings were recessed early in June, an article has appeared in Science (June 7) based on another lecture at CIT (alphabetics for California Institute of Technology) by an Iowa biophysicist (Robert Sinsheimer) on "First Steps Toward a Genetic Chemistry." Here "steps" in progress *toward* the end *sought*—toward what "we *shall* discover" chemically—are recounted as if time were reversed by some extrasensory perception. The facts are "not a sufficient proof * * * but in a way it is *satisfactory* to believe that the physical basis of the gene—the factor that is passed on from cell generation to cell generation—is physically conserved * * *." (Just as it was satisfactory to believe that "parity" was "conserved" in quantum mechanics—until it was shown, not first by observation, but by straight thinking, that it was untenable however satisfying!)

To our biophysicist certain chemical correlations have provided "a *pleasing* confirmation of our hypothesis," even though inconsistent with other facts. To get rid of these embarrassing obstacles to *pleasure* "more refined techniques could prove to be of *great value*" in the "*hope* that the development of a genetic

chemistry will *help* * * *." Why, indeed, are "elegant tracer studies" credited with a "*pleasing* result"? Why do "we know *painfully* little"?

Obviously because "we" are extrapolating our pullets—counting them before they are hatched. Come what may, "we" are not going to hatch any cockerels as long as any refining of techniques (or elaboration of hypotheses) remains to renew the hope that springs eternal in the chemical breast that we shall soon know how to sex them, not by the Japanese manual methods after hatching, but chemically, before the eggs are laid. Some day there will be an RNA for sex, chemically the same for all life from virus to vireo to mastadon.

Here, indeed, is an endless frontier for scientists; a fiscal rathole down which appropriations and empirical data can be poured endlessely before reaching any conclusions; such as foreign-aid policy for Japan to provide a chemically immaculate conception to displace the recently legalized abortions that are keeping Japan (and China, too) from being any further "under-developed"; that is, overpopulated. Why, indeed, worry about poverty if all we need do in the matter of a trait called parental care is to take the long last step in genetic chemistry, to wit, find the proper, parental test-tube in the gene-bank kept under lead to exclude radioactive fallout? We are definitely assured that there *is* a chemistry of the gene (not yet correlated with DNA chemistry, but soon to be) which, *when* "we have become sufficiently advanced in our understanding" of the raveled sleave of mitosis, *will* enable us to "demonstrate the action of a gene *in vitro*"—so we can live *in vitro* for nine months and then have plenty of fresh milk.

To force a reconsideration of the empirical "unity of science" is more of a task than can be encompassed by this brief critique. Such a reconsideration might, however, be invited by a rather categorical review of the high spots in this conceptual revolution as dimly perceived despite the din of data in the process of erecting our scientific Tower of Babel. Conceivably we might thus stir the well-known, open-minded curiosity of scientists in their zealous search for ways to "ring out the old; ring in the new; ring out the false, ring in the true." For not one but many minds are needed in the process of dismissing all the facets of fallacy involved in a postivistic "idealism" which obscures reality by making it synonymous with actuality so that "objective" means a consensus of *subjective* judgments as "verified by experience," pragmatically.

Life on Earth, as a whole, has no opportunity for the unlimited expansion in which it seeks to indulge. For this reason what men, and all other organisms, have most to fear is not the physical environment and its accidents, but far more what other biological occupants ("fellow creatures") do to that environment in the struggle to survive. What any and all biological coalitions do is not purely physical or fortuitous. It is expediently adaptive in its strategic choices so that all free-will, animal life requires eternal vigilance to choose aright and avoid extinction at the hands of other predators who are also exploiting other organisms as "renewable resources," sometimes symbiotically.

Among men, in a civilized context, Disraeli put the case for vigilance against misinformation in a retort to "factual" argument by the Loyal Opposition in parliamentary debate: "There are three kinds of lies; white lies, damned lies, and statistics." Since then there have been frequent protests against empirical "objectivity" among "experts" who employ "factual" correlations to verify hypotheses. These have pursued the pattern, reflecting Christ's warnings against false prophets set forth by St. Paul to Timothy telling him to avoid "profane and vain babblings, and oppositions of science falsely so called."

There have been side-splitting satires, like Chick Sales' "The Specialist," and, less biting but more specific, Anthony Standen's "Science Is a Sacred Cow" featured by the author's review, with lampooning cartoons, in LIFE seven years ago. Lately a physical chemist turned social philosopher, Prof. Michael Polanyi of Manchester, England, has severely indicated *scientific empiricism* in America in a paper read before a symposium on "Fundamental Concepts and Units of Science," published in Science this past winter.

Most recent and satirical of all is a contribution from Cornell University by zoology professor LaMont Cole, "Biological Clock in the Unicorn" (Science, May 3, 1957). This paper so aptly caricatures statistical correlation in verifying an hypothesis that it is astonishing to find, following it by only two weeks in the pages of Science, the same "quantitative information about the effect of radiation on human health" as that presented before this committee by CIT's Prof. Edward Lewis on "Leukemia and Ionizing Radiation" which Dr. Shields Warren rejected as inadequate evidence of causation in leukemia, supported editorially as follows (DuShane, Science May 17, 1957, p. 963, "Loaded Dice"):

"E. B. Lewis shows that there is a direct linear relation between the dose of radiation and the occurrence of leukemia * * * The meaning of such findings is that any amount of radiation takes its toll of the population and any increase takes a greater toll * * * We are approaching the point at which it will be possible to make the phrase 'calculated risk' for radiation mean something a good deal more precise than the 'best guess.' It is apparent that the atomic dice are loaded. The percentages are against us and we ought not play (with bomb tests) unless we must to assure other victories."

The movement toward clean bombs for the West which was revealed in these hearings and later publicized by the President (and caustically condemned by the Kremlin!) has taken the wind out of the Schweitzer sails (and the Pauling Petition) as related to leukemia and strontium 90 so that what remains of the loading of atomic dice by statistical correlation is now confined to genetic effects as alleged by the panel from the National Academy of Science before the Committee on June 4.

If it is not true that (DuShane) "much can be learned about biological reactions (in man) by appropriate statistical and epidemiological studies," such as that of leukemia conducted by a geneticist and statistician (Lewis) who has the "quantitative information" but not the facts about the disease (the recent rise in the statistical incidence of leukemia has been due, not to improved methods of diagnosis, much less to any increase in casual factors, but to the use of penicillin and other "wonder drugs" to save the patients from infections to which bad blood makes them very susceptible), it is equally fallacious to conclude that a correlation between chromosomes and the statistical results of sexual crossing reveals the true character of survival values in organic evolution.

By the same token it is not competent to conclude from a "linear relation," as did Professor Crow before the Committee, that "death, disease, and misery" produce "natural selection" without considering the occasion for such phenomena; for example, war, famine, and pestilence among men, which would be extremely difficult to correlate with any genes without even greater stretching of hypotheses than has as yet been employed in genetics. ("To multiply auxiliary hypotheses is to goropise." See "The Principle of Simplicity" by Lewis Feuer, Philosophy of Science, April, 1957.)

It is not because fruit flies are not men that the monstrous effects of laboratory irradiation do not prove the thesis that the human race will suffer from bomb tests for centuries to come. It is because mutations are not adaptations that this conclusion is unwarranted, either for men or for fruit flies. Gene injuries are as possible as any other anatomical damage from irradiation. But that they persist as recessives throughout a population (without benefit of the dysgenic effects of medical science among men), is not proved by selected statistical averages that destroy the facts.

Mutations, when they do occur, are baneful, indeed; for they destroy the hereditary homeostasis of the species in much the same sense that a burning of books can destroy a cultural heritage. They are *devil*-utionary, not evolutionary. Unlike adaptations to meet competitive pressures they do not remodel the species for survival and become, as Martin contends, "lingering modifications" which do evolve new genes in the course of unobservable time—time which is measured, not in terms of sidereal or sexual events (years or generations) but in terms of marginal elements in interacting stratagems that cannot possibly be reduced to "quantitative information."

Adaptive changes do not occur in detectable form for easy correlation; but they are not therefore mere accidentally adapted mutations. They are teleological expedients, intentionally produced, in the unobservable realm of the microcosm. This is, indeed, an unverifiable hypothesis. But it is not a "goropised" set of hypotheses devised to verify each other in an endless tautology; nor is it untrue because it is unamenable to empirical confirmation by "the scientific method," as was the case with a similar hypothesis in quantum mechanics, to wit, the conservation of parity, which was found to be *predictably* and *testably* false because it pertained to purely physical, inorganic structure. Not observation, but theory, made this discovery!

In biology theory must perforce pursue analogy—the judging of all living processes by our own, in aspects which every man-on-the-street knows and which require no expertise explanations; and these processes are not predictable in their course. They are expedient; just for today. While they cannot violate the natural laws of the physical order, they are not governed by them. They

are "cybernetically" governed, by negative feedbacks that involve a correction in the current theory of information—a corrective which postulates the existence of a metaphysical factor, that is, a dialectical system of commication analogous to human communication in everyday affairs wherein *conditioned* reflexes are a matter of the most familiar processes of modern education. As Whitehead has pointed out, the more civilized we are, the more automatized is our behavior. (Cf. Hayek, "The Use of Knowledge in Society," Am. Econ. Review, September, 1945.)

Concurring with the Martin thesis, Lindegren and Braun (Science, May 10, 1957) cite literature which holds that gene mutation *per se* does not afford a satisfactory explanation of evolution; also that the gene, like sex, is not primitive; nor is its alleged stability a fact.

Pertinent to the fallacy of genetics as a physical science is my own senior thesis dated 1909 (Minnesota) on "The Cytology of Weismannism." This paper not only rejected the mutation-selection theory in almost the same terms as those of Dr. Martin's treatise of 1956, but in a full review of the evidence in the literature of genetic cytology, nucleic acid was twice mentioned as playing some part in the processes of heredity. Though this theme of the early twentieth century is "dated" in some particulars, there is no more reason now than there was then to believe, with Sinsheimer, that "this hard-won recognition of the role of DNA (nucleic acid) has brought us into a new era in genetics and biochemistry. The gene, once a formal abstraction," he concludes, "has begun to condense, to assume form and structure and defined reactivity."

That this is wishful thinking; that there is nothing new in the evidence at hand today which can change the conviction that a chemical explanation of life can never be adduced, can be seen by comparing the two papers. So that interested readers may do so, "The Cytology of Weismannism" is here submitted for its first printing.

To predict by extrapolation from statistical correlations is the unforgivable sin of biological empiricism. There are some contexts in medical practice having legalistic implications, where statistically "educated guesses" as to "calculated risks" form the basis of "expert" opinion used, for example, in judging degrees of disability and causes thereof, as in insurance payments, suits for damages, social "security", veterans compensation, and the like. But in a social context where the results of taking chances with probability are compounded by a multiplier or even exponentially, as in public health policies or in the anarchy of foreign relations where victory or defeat in global warfare is the risk involved, its success, if any, is at best illusory and its failure catastrophic. Biologicaly and culturally Operations Research is worse than futile; it is fraudulent. In medical practice or research epidemiological figures can at best furnish only clues, never evidence that confirms any hypothesis. If the patient is going to die anyway—a fix in which everyone finds himself sooner or later—an experiment may be in order. But when the fate of a people is in the balance and survival is at stake, in disarmament schemes for instance, or when the health of millions of children is concerned with the standardizing of a vaccine, then a trust in statistical probability as against a laissez faire policy, can be disastrous.

Such an error has just been courageously exposed, even in Science (May 31, 1957), namely, the fiction of the safety of the Salk polio vaccine; nor is this the first Nobel Prize in medicine that has not been rescinded in the light of the pitiless truth as to its falsity. In this exposure of the untruths broadcast by the National Foundation for Infantile Paralysis, falsehoods which are plainly proclaimed, there is a serious omission in failing to state the underlying occasion for them, to wit, the idea that the degree of probability can be scientifically measured in biology as in physics and make (to quote the DuShane editorial on dice again) "the phrase 'calculated risk' * * * mean something a good deal more precise than the 'best guess'," and afford a reliable basis for a public policy based on "appropriate statistical and epidemiological studies."

That hundreds of children were not paralyzed and crippled, or even killed, was the work, not of medical science, but of nature; for had the same children been vaccinated with unattenuated virus very few clinical cases would have developed. The probability that infection will cause the disease is so low that its incidence cannot be taken as a measure of the spread of the virus. Poliomyelitis is a relatively rare disease; so also is leukemia. Vaccines for all such afflictions—and they are multifarious and but dimly classified—would burden the community beyond endurance if promoted as a public charity. The treatment of hog chlorea by a similarly attenuated, living vaccine has admittedly kept the disease active instead of exterminating it; so at long last the live

vaccine is being eliminated by law. It has been unjustified also in poliomyelitis, despite the full responsibility of the National Institutes of Health for promoting its use.

The logical fallacy of statistical biology with its reliance on correlations can be shown by an "anthropomorphic" analogy such as was banned from Tennessee public schools by law and, right now, is "scientifically" tabooed by the "positivistic" ban on "anthropomorphic extrapolations."

The cultural DNA of modern civilzation is plainly *money*. That it is coined from gold and other "precious" metals is as irrelevant to its value as an informational guide for human exchanges and consequent behavior, as is the forming of genes (genetic prices) from nucleotides. It is quite as absurd to believe that the substance of DNA must differ in its physical structure in order to create a man instead of a monkey, as it ever was to believe that an ovum is simply a small edition of an embryo and has only to grow to produce an adult man or monkey.

The assertion of Dr. Glass before the Committee that something like a template (a "mould" or "replica") is involved in the embryonic developmental process, is just this sort of untenable belief for which there is no evidence at all. At fault is, basically, the treatment of "information" as a matter of signals as distinguished from signs or symbols with a metaphysically conditioned system of meaning. It is not at all necessary for the same word (or figure) to be used to convey the same meaning (or value); nor different words to appear when different meanings are conveyed. Ambiguity is rampant in language—as are paradoxes in mathematics. An assumption that there is a similar dialectic in genetics constitutes the only possible alternative for the empirical ambiguities which must be eliminated from science by sound reasoning from realistic principles.

The composition of chromosones, genes, alleles, genomes, nucleotides—or what-have-you in genetic equipment—can never determine what they signify in the organism's behavior and development, just as monetary units of various denominations cannot determine the real values underlying the judgments and consequent behavior of a business community as guided by intelligence (price relations); the values are the same regardless of the prices which the number of dollars or pounds determines. *Changing* prices do distort the normal course of judgment and action just as ambiguity distorts the influence of speech or literature in behavior. The fact that it is an artifice, not a reality, that creates the uncertainty of meaning and of biological behavior in general through *conditioned* reflexes (learned symbols) is the essence of the problem of living together by communicated information. There are no puns or paradoxes in nature's realities.

If a Man from Mars were to observe the number of pounds in a British pocket, and the number of dollars in a Canadian pocket, he would have no way of knowing that they differ in their value even though they look alike. So if he were to use Cartesian coordinates in two dimensions to correlate these numbers with the energy level of observed events (behavior) related to those numbers, would he be warranted in "discovering" that this graphic relation is a natural constant? What should he conclude if the British set up another Newtonian Recoinage Commission, or if an International Monetary Fund changed its "mind" on the proper exchange rate, so that the energy level of events departed from the previously observed, linear correlation? Should he call it a "spontaneous" mutation? Would not some Martian jester turn up to publish a report in a Saturnian Science on the rhythmic clock of *Unicornus martius*?

Of Dr. Bentley Glass discovers (as he has) that the correlation between the traits of *Homo insipiens* and the number of chromosomes is not 48 after all, shall he conclude that all the observers who counted 48 were cross-eyed, or that their staining techniques were defective? Or should he conclude, with an ancient inscription in the Libyan Desert, that "Life is change; to cease to change is to cease to live"? If the nucleotides-to-be-counted with electronic relays when genetic chemistry gets a big enough appropriation to buy a big enough computer from IBM, turn out, as all such research to date has done, only some more negative results in the attempt to correlate their chemistry with their behavior in heredity, will it at long last become reasonable to agree with Dr. Lindegren that genes are not stable after all? Will it finally be admitted that his charge that the evidence of such stability has been a matter of choosing only data that support such an assumption, is warranted?

The loophole in the "laws" of Mendelian inheritance as presented by the geneticists before this Committee, lies in their manipulation of "spontaneous"

cases of genetic change to avoid admitting that any of them are adaptive. Adaptation has been ruled out of the vocabulary of organic evolution so that life is today supposed to be one grand symbiotic brotherhood seeking a communistic goal in selfless abandon. Perish the thought of conflict of purposes acting incompatibly in a tooth-and-claw struggle, or of any innovation in survival that has been other than the result of rare accidents in throwing genetic dice that are *not* loaded! Those big claws on Alaskan king crabs turned pink by our loving kindness, are purely ornamental, love-patting appendages, swords beaten into plow shares like atoms-for-peace in Utopia.

The dispute in these hearings over the interpretation of data has hinged on the idea of a threshold as opposed to linear extrapolation, rather than on the evidence that has here been adduced as to the absence of biological constancy where genetic science has postulated it. What remains to be shown, therefore, is that nature consists of thresholds, not of linear correlations, and that research consists of discovering the status of thresholds, never in tracing imaginary linear relations. This would be obvious but for the linear predilections of the Euclidean influence in science for even in the inorganic realm of physics the field of macrocosmic relations is not universal; witness phase transformations such as melting points and boiling points and their energy relations. A jet plane passes from one range of speed into another with an explosive sound as it crosses the "barrier" into a very different set of relations. Physical constants established by instrumental measurements (as by the National Bureau of Standards), such as those of Hooke's Law on the strength of materials, which are inapplicable to very small sizes such as exist in one of the most recent developments in solid state physics (metallic "whiskers"), are another example.

In biology the operation of sensory perception, from which all knowledge proceeds, is replete with thresholds. The *source* of knowledge is the same for any and all of the creatures that inhabit the earth; it lies in a myriad of wave forms, or bands, filling all possible environments throughout the universe. But there are great areas of this spectral information that are extrasensory for any mammal. Some bands are accessible to insects but not to vertibrates; and some mammals such as rats, bats, cats, dogs, can sense ranges greater or less than other species, man included. Some men can sense a range wider than others; but none can be devoid of all sensation and still live.

Other sensations can be translated into tactual perception, as by Braille, to make individuals "literate", that is, to train their reflexes for communication purposes in guiding behavior. In this aspect of a cultural heritage as it operates genetically—and genocidally—in the human struggle to survive, telecommunication preforms transformations in and out of sensory ranges, with continual improvements for technically conditioned people.

Thus science as we know it is continually extending the range of sensation and communication to new bands and ranges; but these new, extrasensory signals have to be modulated into the normal sensory ranges or thresholds before they can affect behavior. No possible information can be had without reference to these physical wave bands as they have been affected by discontinuities (things) in the pyhsical universe. It is because the laws governing these spectral conditions never change anywhere in eternity, so that the sequence of events they reveal is absolute, that time cannot be reversed or events known that have not yet occurred. (Cf. Anthony Standen on "Causes and Effects" in Science, May 3, 1957, p. 900.) Thus no possible living creature can have any information available for conditioning its reflexes (guiding its behavior) that does not rest on these ordered discontinuities (waves and particles), in the physical environment, that are the source of all certainty in sensory experience.

But if sensory equipment has its thresholds, living conditions are also narrowly restricted to those prevailing in the so-called biosphere. Some heat is essential; much more or less, is lethal. It's a case of not crossing a *threshold* and getting *too much* of the good things of life; including life itself. To avoid too much or too little, animals have evolved sensory equipment which men are still evolving by mechanical instrumentation which facilitates motility and thus also serves predatory purposes in acquiring food. Then there are the "trace" elements, essential to plant growth, an excess of which is toxic. In short, there is no such thing as a "linear" relation between life and the elemental forms of matter and energy. A threshold is the very essence of life.

Linear projections or extrapolations are a semantic fiction originating in the Euclidean concepts that arose in the days when the earth was called flat and everything in geometry was worked out in terms of rectilinear and rectangular frames of reference, a state of affairs that still plagues the problems of solid state physics. Molecular engineering, such as is characteristic of this atomic age,

cannot be conducted in these all too familiar terms. The microcosm, in short, is not at all a replica of the macrocosm, as it was held to be by no less a scientist than Thomas Huxley, father of the equally mistaken geneticist, Julian Huxley. This false analogy is being gradually abandoned though the layman is still deluded by the clutter of orbits in pictures of a now discarded concept of the behavior of electrons in an atom. "Shells" (energy "levels") have become the "truth"; and just now there seems a prospect that "spin" will also be displaced, perhaps by a helical structure. The whole theory of atomic and molecular models seems to be in a highly fluid state of uncertainty that is semantic rather than realistic in character. The meaning of symbols, such as the linear representations of Euclidean geometry, is at stake.

The truth about mathematical semantics seems to be that the so-called "natural" numbers, said to be an infinite continuum, are actually *un*natural in being a progression from an arbitrary origin (zero) in two "linear" directions. When they represent objective measurements of physical realities (rather than "value judgments" which can never be referred to any standard, subjective unit to give commutative character to their meaning) they are dimensional and, however remotely derived by instrumentation, they do form an integrated whole or system through reference to the ultimate unit, an arbitrary standard of length, to wit, the yardstick at the National Bureau of Standards. The resulting figures, called measurements, are always, whether "first" or last, approximations and never fully commensurable.

Truly *natural* numbers are dimension*less*; they have no quantitative meaning. Their field seem to be derived from the symmetries of microcosmic, spherical packing which emerge into specific forms in the periodic table of elements and in crystalline structures. Their order, if any, is timeless and independent of any comparison in size and they omit all but the smallest prime numbers since they are exponential and are not amenable to decimal treatment. No zero, no signs (plus or minus), no incommensurables, approximations or probabilities, no statistical averages, are involved in these symmetrical realities that never signify values either measured or subjectively appraised. Incidentally, it is not true that ratios are independent of dimensional numbers.

Assuredly, these venturesome generalizations need to be as critically reviewed as do the accepted conventionalities of mathematics. They are entered here heuristically, to suggest a clear distinction between certainty and uncertainty, a definitive difference between the possible and the impossible, in order to realize that the element of uncertainty is injected as soon as the information existing in the microcosm is "perceived," that is, *after* the optical structure of the eye, for example, has detected the signals and started, not merely to amplify, but to "comprehend" or classify them relative to the purposive procedures implicit in the neural system of any animal, such as a man or an insect. From then on there is a degree of uncertainty paralleling the logical doubt as to the truth of such semantic signs as are used in communication, even by honeybees.

There is nothing more irrational than the bland assumption that what is not yet known to exist must be considered not to exist, scientifically speaking. As Professor Ballard of Tulane University expresses this in the July, 1955, issue of Philosophy of Science, there is every reason to suspect that a dialectic comparable to human (and apian) language is "also carried on within an individual between distinguishable parts of his organism," a phenomenon which obviously presents "metaphysical" aspects and therefore "problems which mechanics cannot solve." Explanations which are a matter of inference by analogy, and not a matter of observation psychologically, thus lose their aura of mysticism and become scientifically anti-empirical. Rational strategy often rests, not on experimental verification of a suspicion (hypothesis) but on an "anthropomorphic" inference or belief that *all* behavior derives from communicated intelligence; subconscious action (instinctive) is not altogether mechanistic or free from awareness, not even in hereditary phenomena.

So meaning in communication is metaphysically incorporated and only approximately true at "best", while at "worst" (these antitheses are reversible in their "value" judgment, depending on whose ox is being gored) it is deliberately deceptive in its strategy, even though only by the "humor" of a pun. The living, purposeful organism has to learn not to be naive but to correct such illusions as that of the asymptotic approach in the perspective of distance or that of change in the pitch of a sound as its source moves past to create a "Doppler effect."

Dr. Schweitzer to the contrary notwithstanding, John Gunther's medical treatise on cancer and death is grounded on falsehood; for it is Life, not Death,

that needs to "Be Not Proud!" Immortality is impossible. Racial survival is possible, but not certain. Indeed, it too is impossible if all life—all mankind—is to be the objective. A Communistic Utopia is entirely outside the pale of any Creation except that of a demagogic imagination. Humility toward our competitors (our "fellow men") can only be hypocritical. Only the Creator (if any; we can never know), or better the actual order of the universe (which is not beneficent but is the very paradigm of neutrality) can be an object of respect and faith on the part of any moral culture seeking a political order not grounded on the personal discretion of leadership (der Feuhrerprinzip) in determining its blindfolded justice.

Occasionally the life of the individual can and must be treated with the utter *sang froid* of statistical probability and war, for it is always subordinate to the higher order that can be immortal. We can be legally exempt from taxes, but not from death. The termination of the life cycle can only be postponed, even by "Atoms in Our Future," the pleasant author of this pleasant prophesy, Senator Anderson, to the contrary notwithstanding. Men may be blessed with "travel out among the stars" and "pushbutton weather" (which is not altogether lacking even in New Mexico without benefit of atomic energy). But any promise of "even eternal life" is out of the reach of either man or God or, even, of the Positivism of such "science" as that which Sir George Thompson has set forth *ex cathedra* in his "The Foreseeable Future."

In her youth Lily Pons popularized "I Dream Too Much!" Perhaps her vibrant voice could yet teach it to Sir George and the prophetic Senator and persuade them and their sycophants in science to think in terms of thresholds rather than extrapolations.

Appendix 8

Radioactive Fallout

A Short Selected List of References

(Prepared by Ruth A. Little, Legislative Reference Service, Library of Congress, June 30, 1957)

Atomic Scientists Association, Great Britain. Statement on strontium hazards. [London, England, 1957], 7 pp. Mimeographed.

Campbell, C. I. Radiostrontium fallout from continuing nuclear tests. Science, v. 124, November 2, 1956; 894.

Eisenbud, Merril. Global distribution of strontium-90 from nuclear detonations. Scientific monthly, v. 84, May 1957: 237–244.

——, and Harley, John H. Radioactive fallout in the United States. Science, v. 121, May 13, 1955: 677–680.

——, and Harley, John H. Radioactive fallout through September 1955. Science, v. 124, August 10, 1956: 251–255.

Genetics and radiation. Eugenics quarterly, v. 3, September 1956: 131–138; December 1956: 201–208.

Genetics and the atom. Bulletin of the atomic scientists, v. 11, November 1955: 314–343.

Great Britain. Atomic Energy Research Establishment. The radiological dose to persons in the United Kingdom due to debris from nuclear test explosions prior to January 1956, by N. G. Stewart [and others]. Harwell, Berkshire, England, 1956. 22 pp. (A. E. R. E. HP/R 2017).

——. Radiostrontium fallout in biological materials in Britain, by R. J. Bryant [and others]. Harwell, Berkshire, England, September 5, 1956. 44 pp. A. E. R. E. HP/R 2056).

——. Medical Research Council. The hazards to man of nuclear and allied radiations. London, Her Majesty's Stationery Office, 1956. 128 pp.

India (Republic) Defense Science Organization. Nuclear explosions and their effects. Foreword by Jawaharlal Nehru. [Delhi] Publications Division, Ministry of Information and Broadcasting, Government of India, August 1956. 184 pp.

International Conference on the Peaceful Uses of Atomic Energy. Proceedings of the International Conference * * * held in Geneva, August 8–August 20, 1955. New York, United Nations, 1956. 16 v. (Vols. 7, 11, 13 contain papers relating to radioactivity and fallout.)

Kulp, L. Laurence, and others. Strontium-90 in man. Science, v. 125, February 8, 1957: 219–225.
Commentary by Ralph E. Lapp. *Ibid.*, May 10, 1957: 933–934.
Reply by Kulp and others: *Ibid.:* 934.

Lapp, Ralph E., *jt. auth.* Radiation; what it is and how it affects you, by Jack Schubert and Ralph E. Lapp. New York, Viking, 1957. 314 p.

——. Strontium limits in peace and war. Bulletin of the atomic scientists, v. 12, October 1956: 287–289 ff. [Note: See main bibliography for further references to articles by Dr. Lapp in the Bulletin of the atomic scientists and elsewhere.]

Lewis, E. B. Leukemia and ionizing radiation. Science, v. 125, May 17, 1957: 965–972.

Libby, Willard F. Current research findings on radioactive fallout. Proceedings of the National Academy of Sciences, v. 42, December 1956: 945–962.

——. Dosages from natural radioactivity and cosmic rays. Science, v. 122, July 8, 1955: 57–58.

——. Radioactive fallout. Washington, United States Atomic Energy Commission, 1957. 24 pp., charts. Mimeographed. To be published in the Proceedings of the National Academy of Sciences.

——. Radioactive strontium fallout. Proceedings of the National Academy of Sciences, v. 42, June 1956: 365–390.

——, and others. Forum on the Schweitzer declaration. Saturday review, v. 40, May 25, 1957: 8–13, ff.

Machta, L. and Harris, D. L. Effects of atomic explosions on weather. Science, v. 121, January 21, 1955: 75–81.

——, and others. Worldwide travel of atomic debris. Science, v. 124, September 14, 1956: 474–477.

Maximum permissible radiation exposures to man; a preliminary statement of the National Committee on Radiation Protection and Measurement. A special report. A. M. A. Archives of industrial health, v. 15, Apr. 1957: 350–355.

Muller, Hermann. How radiation changes the genetic constitution. Bulletin of the atomic scientists, v. 11, November 1955: 329–338.

——. Race poisoning by radiation. Saturday review, v. 34, June 9, 1956: 9, ff.

National Research Council. The biological effects of atomic radiation. A report to the public. Washington, D. C., 1956. 40 pp.

—— ——. Summary reports. Washington, D. C., 1956. 108 pp.

——. Pathological effects of atomic radiation. Washington, D. C., 1956. Various paging. (Publication no. 452).

Russell, W. L. Radiation in mice—the genetic effects and their implications for man. Bulletin of the atomic scientists, v. 12, January 1956: 19–20.

Schweitzer, Albert. A declaration of conscience. Saturday review, v. 40, May 18, 1957: 17–20.

Strauss, Lewis L. The effects of high-yield nuclear explosions. Washington, United States Government Printing Office, February 1955. 19 pp.

Turekian, K. K. and Kulp, J. L. Strontium content of human bones. Science, v. 124, August 31, 1956: 405–407.

United Nations. World Health Organization. The effect of radiation on human heredity; report of a study group, Copenhagen, August 7–11, 1956. [Geneva] January 24, 1957. Various paging.

United States Atomic Energy Commission. Semiannual reports. Washington, D. C., United Government Printing Office, 1953. 13th–21st semiannual reports contain sections on fallout.

——. New York Operations Office. Health and Safety Laboratory. Annotated bibliography on fallout resulting from nuclear explosions; by Allen G. Hoard, Merrill Eisenbud, John H. Harley. New York, September 1956. 44 pp. (NYO–4753).

——. Annotated bibliography on long-range effects of fallout from nuclear explosions, by Allen G. Hoard, Merril Eisenbud, John H. Harley. New York, November 1956. 15 pp. (NYO–4753 Suppl. 1).

——. Background radiation; a literature search, by Wayne M. Lowder and Leonard R. Solon. New York, July 1956. 43 pp. (NYO–4712).

United States Congress. House. Committee on Government Operations. Civil defense for national survival. Hearings before a subcommittee, 84th Congress, 2nd session. Washington, United States Government Printing Office, 1956. 3145 pp. (in 7 pts.) Statements by Willard F. Libby, Willard Bascom,

Merle A. Tuve, Lester Machta, Lauriston B. Taylor, Ralph E. Lapp, Charles L. Dunham, Eugene P. Cronkite; H. Bentley Glass.

——. Joint Committee on Atomic Energy. Health and safety problems and weather effects associated with atomic explosions. Hearing before the Joint Committee on Atomic Energy. 84th Congress, 1st session, April 15, 1955. Washington, United States Government Printing Office, 1955. 60 pp. (Committee print).

United States National Bureau of Standards. Handbooks. Washington, United States Government Printing Office, 1949. Nos. 42, 48–62.

United States National Committee on Radiation Protection. Maximum permissible radiation exposures to man. Washington, United States Government Printing Office, 1957. 32 pp. (National Bureau of Standards, Technical News Bulletin, v. 41, no. 21).

APPENDIX 9

RADIOACTIVE FALLOUT

SELECTED LIST OF REFERENCES

(Prepared by Ruth A. Little, Legislative Reference Service, Library of Congress, June 30, 1957)

BIBLIOGRAPHIES

Argonne National Laboratory, Chicago, Ill. Annotated bibliography in radiobiology, by Sophie V. Stephens and Robert D. Boche. Chicago, Ill., 1953. 367 pp. (ANL–5111).

Bibliography containing 2,153 abstracts covering all phases of the biological effects of radiation.

Available from Office of Technical Services, United States Department of Commerce.

——— ——— The tolerance dose, by S. T. Cantril and H. M. Parker. Chicago, Ill., January 5, 1945. 23 pp. (MDDC–1100).

Atomic Bomb Casualty Commission. Bibliography of A-bomb material collected through April 17, 1952. Washington, D. C., 1952. 27 pp. (AEC File No. NP–4563).

List of 124 references covering both published and unpublished material on the effects of the atomic explosions at Hiroshima and Nagasaki.

Battelle Memorial Institute, Columbus, Ohio. A bibliography on "The effects of X-rays on bacteria," by R. F. Robinson [and others]. Columbus, Ohio, August 20, 1951. 18 pp. (AEC File No. NP–3610).

Includes published articles from 1896 through August 1951.

Brookhaven National Laboratory, Upton, N. Y. Conference on biological applications of nuclear physics, July 12–27, 1948. Upton, N. Y., 1948. 154 pp. (BNL–C–4).

Summary paper covering biological effects of radiations. Documented by lists of references.

——— ——— Effects of radiations on biological systems, by A. H. Sparrow and B. A. Rubin. Upton, N. Y., February 15, 1951. 94 pp. (BNL–97; AECU–1207).

California. University. University at Los Angeles. Atomic Energy Project. Recent attempts to alter the response to ionizing radiations, by Wilbur A. Selle [and others]. Los Angeles, Calif., September 15, 1953. 73 pp. (UCLA–264).

Previous reports reviewing literature dealing with attempts to alter the response of animals to ionizing radiations were published in 1949 and 1951. This report summarizes 118 additional papers published 1951 to 1953.

Carbide & Carbon Chemicals Co., New York, N. Y. Literature survey on skin decontamination, unclassified material, by Francis L. Sachs. New York, 1951. 2 parts.

Part II, 22 pp. (Y–B4–4).

Part III, 23 pp. (Y–B4–10).

——— ——— Radiation effects on the blood, by Francis L. Sachs. New York, July 23, 1951. 11 pp. (Y–B4–22).

Bibliography containing 51 titles selected from Nuclear Science Abstracts.

——— ——— Preliminary literature survey on skin decontamination, unclassified material, by Francis L. Sachs. New York, March 1, 1951. 10 pp. (M–4638; TEM–12317).

——— ——— Skin decontamination; a literature search, by Francis L. Sachs, New York, October 22, 1952. 26 pp. (Y–914).

63 annotated references on skin decontamination, radiation protection and treatment of radiation injuries.

Chicago. University. Metallurgical Laboratory. The effects of X-rays on immunity, by W. H. Taliaferro and L. G. Taliaferro. Chicago, Ill., June 1946. 42 pp. (CH–3891).

Survey of X-irradiation effects on: formation of specific antibodies after immunization with nonliving antigens; infection with animal parasites; infection with bacteria and viruses; and mechanisms of radiation effects in the host. 158 references.

Great Britain. Atomic Energy Research Establishment. Range-energy and range-momentum relationships for charged particles; a bibliography. Harwell, Berkshire, England, February 1951. 27 pp. (AERE–Inf/Bib–71).

222 references.

——— ——— Some papers on the effects of radiations of bacteria and moulds. Harwell, Berkshire, England, October 14, 1949. 7 pp. (AERE–Inf/Bib–51).

List of 69 papers covering 1930 to 1949.

——— ——— Some published reports on the effects of radiation on reproduction and on the genetic effects of radiation, by A. Lundie. Harwell, Berkshire, England, March 8, 1950. 10 pp. (AERE–Inf/Bib–61).

List of 69 references.

List, Robert J. Annotated bibliography on the transport and deposition of atomic debris. Washington, United States Government Printing Office, 1956. 27 pp. (U. S. Atomic Energy Commission TID–3061).

National Research Council. Division of Medical Sciences. Conference on radiation cataracts and neutron effects, by Philip H. Abelson. Washington, D. C., February 17, 1950. 100 pp. (AEC File no. NP–1826).

Report on the conference, composed of two parts: (1) a discussion of the production and detection of neutrons and of experimental neutron-irradiation of animals and (2) a discussion of various studies made on the lens of the eye and the effects of radiation thereon. 181 references.

Oak Ridge National Laboratory, Oak Ridge, Tenn. Bibliography of unclassified documents. Oak Ridge, Tenn., n. d. 10 pp. (ORNL–502, pt. 2).

Documents included cover the biological and medical aspects of radiation; radiation protection and health physics; and inhalation of toxic and radioactive materials.

United States Army Medical Research Laboratory, Fort Knox, Ky. Adaptation to ionizing radiation, by A. T. Krebs and John B. Storer. Fort Knox, Ky., February 2, 1955. 17 pp. (AMRL–175).

Reviews literature on radioresistance in mammalian systems. 40 references.

United States Atomic Energy Commission. Civil defense against atomic warfare; a selected reading list. Washington, D. C., United States Government Printing Office, March 1950. 24 pp.

List of references on radiation detection, protection and biological effects. Divided into USAEC and other Government agency reports.

——— ——— Radioactive waste disposal; a bibliography of unclassified literature, by R. L. Shannon. Washington, D. C., August 1950. 8 pp. (TID–375).

Abstracts of 49 unclassified reports.

——— ——— Division of Engineering. Waste materials in the United States atomic energy program, by Abel Wolman and Arthur E. Gorman. Washington, D. C., January 12, 1950. 20 pp. (Wash. 8).

——— ———, New York Operations Office. Health and Safety Laboratory. Annotated bibliography on fallout resulting from nuclear explosions, by Allen G. Hoard, Merril Eisenbud, John H. Harley. New York, September 1956. 44 pp. (NYO–4753).

——— ———, New York Operations Office. Health and Safety Laboratory. Annotated bibliography on long-range effects of fallout from nuclear explosions, by Allen G. Hoard, Merril Eisenbud, John H. Harley. New York, November 1956. 15 pp. (NYO–4753 Suppl. 1.)

——— ———, New York Operations Office. Health and Safety Laboratory. Background radiation; a literature search, by Wayne M. Lowder and Leonard R. Solon. New York, July 1956. 43 pp. (NYO–4712).

Available from Office of Technical Servces, United States Department of Commerce.

——— ———, Technical Information Service. Biological effects of neutrons, gamma radiation, and neutron-gamma combinations. A selective bibliography of unclassified literature, by Lore Rose David. Oak Ridge, Tenn., May 1954. 38 pp. (TID–3052).

United States Library of Congress. Technical Information Division. Radioactive manganese, iron, cobalt, copper, zinc and strontium. A selected bibliography of recent literature with special reference to biological and medical applications, by M. C. Lekind and M. H. Eller. Washington, D. C., February 24, 1948. 19 pp. (AEC File No. NP-232).
151 references.

United States National Bureau of Standards. The biological effects of deuterium compounds by Harold J. Morowitz and Lawrence M. Brown. Washington, D. C., January 14, 1953. 50 pp. (NBS-2179).
Literature survey consisting of 216 references.

United States School of Aviation Medicine, Randolph AFB, Tex. Mechanisms of hypersensitivity and their significance in relation to certain aspects of the radiation syndrome: a critical review. Report No. 1, by Kenneth L. Burdon, School of Aviation Medicine and Baylor University College of Medicine. August 1953. 44 pp. (NP-4978).
424 references.

Books and Parts of Books

Auerbach, Charlotte. Genetics in the atomic age. Fair Lawn, N. J., Essential Books, 1956. 106 pp.

Bale, W. F. Health hazards in the use of radioactive isotopes. In his The use of isotopes in biology and medicine. Madison, University of Wisconsin press, 1948. pp. 395-408.

Blair, Henry A. Biological effects of external radiation. New York, McGraw, 1954. 508 pp. (National nuclear energy series. Manhattan Project technical section. Division VI [University of Rochester Projects] v. 2).

Bugher, John C., and others, eds. Medical sciences. New York, McGraw, 1956– 1 v. (Progress in nuclear energy, ser. 7.)
Deals with medical radiology.

Cantril, S. T. Biological bases for maximum permissible exposures. In Industrial medicine on the plutonium project. New York, McGraw, 1951. Chap. 2. (National nuclear energy series, division iv, v. 20.)

Dean, Gordon E. Report on the atom; what you should know about the atomic energy program of the United States. Second edition with a new chapter covering recent developments. New York, Knopf, 1957. 359 p.
Discussion of fallout: pp. 348-351.

Fano, U. Principles of radiological physics, In Hollaender, Alexander, ed. Radiation biology, New York, McGraw, 1954, v. 1, pp. 1-144.

Great Britain Admirality. Notes on atomic energy for medical officers. New York, Philosophical Library, c 1956. 169 p.

Hachiya, Michihiko. Hiroshima diary; the journal of a Japanese physician, August 6–September 30, 1945. Translated and edited by Warner Wells. Chapel Hill, N. C., University of North Carolina press, 1955. 238 pp.

Hultqvist, Bengt. Studies on naturally occurring ionizing radiations, with special reference to radiation doses in Swedish houses of various types. Stockholm, Almqvist and Wiksell, 1956, 125 pp.

Lapp, Ralph E. The accident, radioactive fallout. In his Atoms and people, New York, Harper, 1956. Chap. 7.

———, joint author. Radiation: what it is and how it affects you, by Jack Schubert and Ralp E. Lapp. N. Y., Viking, 1957. 314 pp.

———, and Andrews, H. L. Nuclear radiation physics. New York, Prentice-Hall, 1954. 532 pp.

Lea, Douglas E. Action of radiations on living cells. Cambridge [England], University Press, 1955. 416 pp.

Muller, Hermann J. The nature of the genetic effects produced by radiation. In Hollaender, A., ed. Radiation biology, New York, McGraw, 1954, v. 1, pp. 351-453.

———, Radiation damage to the genetic material. In Science in progress. New Haven, Yale University Press, 1951. pp. 93-165, 481-493.

Nagasawa, K., and others. Results of investigations and examinations on the influences upon sea food in Japan caused by the hydrogen bomb experiments at Bikini Atoll, 1954. In research in the effects and influences of the nuclear bomb test explosions, Tokyo, Japan, Society for the Promotion of Science, 1956. p. 895.

Oughterson, Ashley W., and Warren, Shields. Medical effects of the atomic bomb in Japan. New York, McGraw, 1956, 477 pp. (National Nuclear Energy Series, Manhattan Project technical section. Division VIII, v. 8).

Spear, Frederick G. Radiations and living cells; an introduction to radiation biology, in which the action of penetrating radiations on the living cell is described, with special reference to the effect on cell division in human tissues. New York, Wiley, 1953, 222 pp.

Symposium on Radiobiology, Oberlin College, 1950. The basic aspects of radiation effects on living systems, edited by James J. Nickson. New York, Wiley [1952] 465 p.

Gene mutations caused by radiation, by H. J. Muller, pp. 296–332.

Analysis of mammalian radiation injury and lethality, by Austin M. Brues and George A. Sacher: 441–465.

Zirkle, Raymond E. Biological effects of external X and gamma radiation. New York, McGraw, 1954. (National nuclear energy series. Manhattan Project technical section. Division IV: Plutonium Project record, v. 22B).

———. Statistical analysis of the medical effects of the atomic bomb from the Report of the Joint Commission for the Investigation of the Effects of the Atomic Bomb in Japan, by Ashley W. Oughterson [and others]. Oak Ridge, Tenn., U. S. Atomic Energy Commission, Technical Information Service, 1955, 288 pp. (U. S. Atomic Energy Commission, TID–5252).

Renne, Harold S. Atomic radiation detection and measurement. Indianapolis, H. W. Sams, c. 1955, 198 pp. (A Howard W. Sams photofact publication, ADR–1.)

Schubert, Jack, and Lapp, Ralph E. Radiation, what it is and how it affects you. N. Y., Viking, 1957, 314 pp.

Trumbull, Robert. Nine who survived Hiroshima and Nagasaki; personal experiences of nine men who lived through the atomic bombings. New York, Dutton, 1957, 148 pp.

Technical Periodical Articles, United States

GENERAL SCIENTIFIC ARTICLES

A-blasted food supplies; foods containing phosphorus and sodium chloride as permanently radioactive. Chemical and engineering news, v. 34, October 29, 1956: 5244 f.

After the fall-out is over. Chemical week, v. 76, March 26, 1955: 72 f.

Amphlett, C. B. Behavior of radioactive contamination in the ground. World crops, v. 9, March 1957: 112–115.

Examines the role of the soil in limiting the spread of contamination.

———. Soil chemistry and the uptake of fission products. Research (London), v. 8, 1955: 335–340.

Traces the likely fate of radioactive contamination from an atomic bomb explosion or a nuclear reactor accident according to the nature of the ground contaminated, and discusses possible treatments of contaminated land.

Anderson, E. C., and others. Radioactivity of people and foods. Sciences, v. 125, June 28, 1957: 1273–1278.

Andrews, Howard L. Radioactive fallout from bomb clouds. Science, v. 122, September 9, 1955: 453–456.

General discussion of radioactive fallout in the vicinity of nuclear detonations, including mechanics of formation, amount of activity, etc. Discusses maximum permissible levels of gamma radiation for test series, estimates dosages near test site under certain assumptions. Discusses beta burn hazard and danger from retention of particles in lungs. Concludes only particles from 0.5 to 5 micron diameter are potentially hazardous to lungs, and fraction in this size range will be small. Also discusses long-lived fallout and potential hazard from Sr–90 and genetic effects. Concludes weapon testing program justified by defense effort and that radiological hazards have been minimized under the well-controlled conditions of weapon testing.

Answer sought to fallout hazards. Chemical and engineering news, v. 33, April 25, 1955: 1774.

Baker, W. K., and Von Halle, E. Production of dominant lethals in drosophila by fast neutrons from cyclotron irradiation and nuclear detonations. Science, v. 119, January 1, 1954: 46–49.

Bell, Carlos G., and others. Passage of nuclear detonation debris through water treatment plants. Journal of the American Water Works Association, v. 46, October 1954: 973–986.

After nuclear detonations, 2,200 samples taken from 3 water plants, were measured for beta-gamma radioactivity; within 2 weeks after detonation 45

percent of fallout radioactivity passed through rapid sand filtration plants; from 2 to 10 weeks after detonation, percentage increased to 53 percent, and 10 weeks after detonation all radioactivity passed through plant.

Blifford, Irving H. Collection of atomic bomb debris from the atmosphere by impaction on screens. Science, v. 123, 1956: 1120–1121.

——— and Rosenstock, Herbert B. Fallout dosages at Washington, D. C. Science, v. 123, 1956: 619–622.

Buckthought, K. Radioactivity and the hydrogen bomb. Canadian chemical processing, v. 38, April 1954: 78 ff.

Burnett, T. J., and Hatch, T. F. Estimating airborne radioactive particulate hazards—a review of sampling criteria. American Industrial Hygiene Association Quarterly, v. 17, March 1956: 85–88.

Methods for sampling airborne radioactivity are reviewed and evaluated.

Campbell, Charles. Radiostrontium fallout from continuing nuclear tests. Science, v. 124, November 2, 1956: 894–895.

Carter, T. C. Genetic problem of irradiated human populations. Bulletin of the atomic scientists, v. 11, December 1955: 362–363.

Caster, W. O. Strontium-90 hazard: relationship between maximum permissible concentration and population mean. Science, v. 125, June 28, 1957: 1291.

Chow, Tsaihwa J., and Thompson, Thomas G. Flame photometric determination of strontium in sea water. Analytical chemistry, v. 27, January 1955:18–21.

Clark, H. M. The occurrence of an unusually high-level radioactive rainout in the area of Troy, N. Y. Science, v. 119, May 7, 1954: 619–622.

Thirty-six hours after detonation of a nuclear bomb at the Nevada Proving Ground, an unusually violent electrical storm hit Troy, N. Y., 2,300 miles distant. The storm left in its wake an exceptionally high, though not hazardous, deposition of radioactive material.

Clark, Stanley H. Genetic radiation exposures in the field of medicine. Bulletin of the atomic scientists, v. 12, January 1956: 14–18.

Cohn, S. H., and others. Nature and extent of internal radioactive contamination of human beings exposed to fallout material in Operation Castle. Radiation research, v. 3, October 1955. Presented before the Radiation Research Society, New York, May 16–18, 1955.

The first instance of exposure of human beings to significant internal contamination with fission products occurred as a result of the ingestion and inhalation of fallout material from a nuclear detonation in the spring of 1954. An evaluation of the nature and extent of these internal radioelements excreted by the exposed human beings with data obtained from radiochemical analysis of the tissues and excreta of animals contaminated in the same event.

Cole, L. C. Biological clock in the unicorn. Science, v. 125, May 3, 1957: 874–876.

Comar, C. L., and others. Thyroid radioactivity after nuclear weapons tests. Science, v. 126, July 5, 1957: 16–18.

Conger, Alan D. The relative biological effectiveness of radiation from a nuclear detonation on tradescantia chromosomes. Science, v. 119, January 1, 1954: 36–42.

Tests at Oak Ridge on the flowering plant tradescantia to determine quantitative relation between radiation dose and biological effects of radiation resulting from nuclear detonation.

Coven, A. W. Evidence of increased radioactivity of the atmosphere after the atomic bomb test in New Mexico. Physical review, v. 68, 1945: 279.

A G.-M. counter and circuit (Rev. Sci. Instr. 13, 188 (1942)) was tested for background count near Annapolis, on July 12, 14, 15, 19, August 16 and October 31–November 3, all in 1945. The background count was 5.5 per minute. On July 16–18 the background count was 6.3, 7.7, and 10.7 per minute, respectively.

Cronkite, Eugene P., and others. Biological effect of atomic bomb gamma radiation. Science, v. 122, July 22, 1955: 148–150.

Mice were exposed to atomic bomb radiation at 28 stations no both sides of the established L. D. $_{50}$ distance to compare the biological effect with that produced by laboratory gamma and X-radiations. At doses up to 620 r, mortality was consistently at 3 percent but above this dosage, mortality increased rapidly to 950 r, which was approximately the absolute lethal dose. The established L. D. $_{50}$ was 759 r. The relative biological effects of X-ray to bomb radiation closely approached unity.

Cronkite, Eugene P., and others. The characteristics of fallout material and the effects of fallout radiation on human beings. Radiation research, v. 3., No. 2, 1955. Presented before the Radiation Research Society, New York, May 16–18, 1955.

Human beings were accidentally exposed to fallout radiation commencing approximately 5 hours after explosion of a large nuclear device. The fallout material was visible (snowlike). It contaminated skin, clothes, and surroundings, producing skin lesions, whole-body effects, and internal deposition of small amounts of radionuclides. Whole-body exposure occurred at four levels: 175 r, 78 r, 69 r, and 14 r midline dose of radiation.

Damon, P. E. and Kuroda, P. K. Artificial radioactivity of rainfall. Nucleonics, v. 11, December 1953: 59.

Fission products found in rain water during the period June 5–July 23, 1953, correlated with the origin of the prevailing air mass. The total fission activity fallout in the vicinity of Fayetteville, Ark., during this period was in excess of 10^{-8} curies per square mile.

Disposal and dispersal of radioactive wastes. Science, v. 124, July 6, 1956: 17–19. Part of National Academy of Sciences report.

Dunn, L. C. Radiation and genetics. Scientific monthly, v. 84, Jan. 1957: 6–10.

Dunning, Gordon M. Effects of nuclear weapons testing. Scientific monthly, v. 81, December 1955: 265–270.

Presents a summary of blast, thermal and radiation effects of atomic testing. Discusses internal radiation hazard from radioactive iodine, strontium and carbon hazard from contaminated foods. Concludes hazard negligible to date. Discusses genetic effects and possible increase in mutations. Computes the average radiation exposure to people in the United States from all nuclear detonations to date is 0.1 roentgen. Discusses possible effects on weather and nitric acid formation and concludes effect is negligible.

———. Thyroid dose from radioiodine in fallout. Nucleonics, v. 14, 1956: 38–41.

Eisenbud, Merril. The AEC fallout monitoring network. Journal of the Air Pollution Control Association, v. 6, November 1956: 144–146.

———. Global distribution of strontium-90 from nuclear detonations. Scientific monthly, v. 84, May 1957: 237–244.

———. Monitoring network for measuring radioactive fallout. Journal of the American Water Works Association, v. 48, 1956: 659–664.

The basic mechanics of radioactive fallout are discussed. The principal isotopes are listed, collection stations are tabulated, and methods of analysis are briefly outlined. The isotope Sr^{90} is considered the constituent of prime biological significance.

——— and Harley, John H. Radioactive dust from nuclear detonations. Science, v. 117, February 13, 1953: 141–147.

A network of 121 monitoring stations has been established in the United States to collect airborne and settled dust samples for radioactive assay. The results from 30,000 samples collected in conjunction with the 8 detonations in Nevada between April 1 and June 4, 1953, are given. For brief periods following an explosion the radioactive background can be increased in distant areas by fallout of airborne dust.

——— and Harley, John H. Radioactive fallout in the United States. Science, v. 121, May 13, 1955: 677–680.

Summarizes fallout in the United States from early in 1951 through 1954. Accumulated fission product activity in the United States, exclusive of the area within 200 miles of the Nevada test site, was 61 millicuries per square mile. Gamma radiation from this is of the order of 0.0010 mr/hr compared with normal background of 0.005 to 0.05 mr/hr. Measuring technique detects increase of 10^{-8} roentgens per hour, natural background 5×10^{-6} to 5×10^{-5} r/hr.

——— and Harley, John H. Radioactive fallout through September 1955. Science, v. 124, August 10, 1956: 251–255. Bibliography.

Eliassen, Rolf and Lauderdale, Robert A. Radioactive fallout in water supply at Portland, Maine. Journal of the American Water Works Association, v. 48, 1956: 665–670.

This work was done to determine any radioactive increase following atomic weapons tests during 1955. Nine objectives are outlined. Analytical procedures are listed, and the results discussed. Materials of all kinds were

found. It was found that Sr^{89} in the tap water could be increased by a factor of about 70,000 before exceeding the limits specified by the National Bureau of Standards.

Fafarman A. and Shamos, M. H. Effect of fallout from atomic blast on background counting rate. Nucleonics, v. 11, June 1953: 80–81.

Background measurements with a sodium-iodide scintillation detector at New York University show normal variation of ±5 percent. At 1030, March 19, 1953, the rate increased to 3850 cpm, 600 percent above background. A heavy rain preceded the measurement and presumably contained debris from the March 17 Nevada test. Removal of rainwater dropped rate to 1470 cpm. Curves of energy versus count before and after fallout are given.

Fallout detector. Military review, v. 35, March 1956; 69.

Fallout; new H-bomb peril? Chemical and engineering news, v. 33, February 28, 1955: 842–843.

Fallout warning signal blankets United States. Signal, v. 10, March–April 1956: 70.

Fearson, R. E., and others. Results of atmospheric analyses done at Tulsa, Okla., during the period neighboring the time of the second Bikini atomic bomb test. Physical review, v. 70, October 1 and 15, 1946: 564.

Radioactive concentrates were prepared from the atmosphere. Data of July 26 and August 30, 1946, represent the active deposits of Rn and Tn. The data of July 28, based on two samples with initial intensities of $\sim 5 \times 10^{-10}$ curie, are explained by assuming that the concentrate is the active deposit of a new rare radioactive gas of at. No. 86, with a halflife of 82 min.; it corresponds with at least two members of an unreported radioactive series.

Fields, P. R., and others. Transplutonium elements in themonuclear test debris. Physical review, v. 102, 1956: 180–182.

The isotopes of curium, berkelium, and californium found in the thermonuclear debris of the November 1952 thermonuclear test are discussed. The instantaneous buildup of the heavy elements in the thermonuclear device is compared with the buildup during pile irradiation. The alpha-particle energy (5.4 Mev) and the spontaneous fission half-life ($<1.2 \times 10^7$ years) of Cm^{246} are reported. The spontaneous fission halflife of Cf^{254} was found to be 55 days. No other mode of decay was observed for this isotope.

Genetic effects of atomic radiation. Science, v. 123, June 29, 1956: 1157–1164.

Text of the summary report of the Committee on Genetic Effects of Atomic Radiation, one of six reports prepared for the Study of the Biological Effects of Atomic Radiation by the National Academy of Sciences.

Genetics and the atom. Bulletin of the atomic scientists, v. 11, November 1955: 314–343.

Issue contains an editorial on genetics in Geneva, glossary of genetic terms and articles on the subject.

Greenfield, S. M. Ionization of radioactive particles in the free air. Journal of geophysical research, v. 61, 1956: 27–33.

In order to evaluate the possible role of radioactive particles from an atomic cloud as condensation nuclei, an analysis has been made to determine their degree of ionization. Individual radioactive particles become ionized owing to β-emission, and an estimate of the half-life of these ions has been made for various times in the life history of an atomic cloud. It is concluded that while there is a transient charge on these particles, its half-life is small compared to the disintegration rate, with the result that for all practical purposes radioactive particles in the free air are not necessarily preferred condensation nuclei.

——— Rain scavenging of radioactive particulate matter from the atmosphere. Journal of meteorology, v. 14, April 1957: 115–125.

Hahn, Richard B. and Straub, Conrad P. Determination of radioactive strontium and barium in water. Journal of American Water Works Association, v. 47, April 1955: 335–340.

Haldane, J. B. S. Genetical effects of radiation from products of nuclear explosions. Nature, (London) v. 176, July 16, 1955: 115.

The serious nature of genetical effects of radiation is argued. Upper and lower limits of radiation-induced human mortality are estimated.

Harris, D. Lee. Effects of atomic explosions on the frequency of tornadoes in the United States. Monthly weather review, v. 82, December 1954: 360–369.

The increase in tornadoes reported in the United States during the past few years is ascribed to better reporting procedures rather than the presence of atomic debris. Maps showing the distribution of fallout in the United States during the first and second halves of May 1953 are given.

Harris, William B., and LeVine, Harris D. Sampling and measurement of radioactive atmospheric pollution. Proceedings of the Air Pollution Control Association, 1953: 17–21.

Apparatus is described for continuously monitoring the alpha and gamma radiation from stack effluents. A storage-battery-driven dust collector can be used to collect dust away from power sources. An adhesive-coated film can be used to collect "fallout," at a distance from the source. An apparatus is shown for collecting samples nearer the source of contamination by the use of impactors, cyclones, etc.

Harris, D. Lee. Effects of radioactive debris from nuclear explosions on electrical conductivity of lower atmosphere. Journal of geophysical research, v. 60, March 1955: 45–52.

An increase in the ionization near the ground due to the fallout from a radioactive cloud formed by a nuclear explosion will increase the conductivity and lower the potential gradient in the lower atmosphere. Records of atmospheric conductivity and potential gradient from the Tucson Magnetic Observatory are compared with records of the deposition of atomic debris on the ground following the Nevada tests.

Herzog, G. Gamma-ray anomaly following the atomic bomb test of July 1, 1946. Physical review, v. 70, 1946: 227–228.

A recording gamma-ray meter (in Houston, Tex.) indicated a peak in atmospheric gamma rays, from 8 p. m., July 4 to 7 p. m., July 5, with a maximum at 3 a. m., July 5. The maximum increase was 77 percent of the background count.

Heslep, J. M., and Bellamy, A. W. Sampling for airborne radioactivity. Air repair, v. 5, May 1955: 1–4.

Potential sources of radioactive aerosols are discussed, and special attention is given to widespread contamination as might be expected from atomic weapons. There is no perfect sampling method, but rather good results have been secured by use of con. vacuum cleaners and Hollingsworth and Vose H–70 filter paper. Particle-size determinations are best made by the cascade impactor—this gives good characterization of particle size down to 0.3–0.6 μ

Hess, Victor F. and Luger, Paul. The ionization of the atmosphere in the New York area before and after the Bikini atom-bomb test. Physical review, v. 70, 1946; 564–565.

From June 29 through July 10, 1946, no atmospheric ionization due to the atomic bomb was observed.

Hollaender, Alexander. Modification of radiation response. Bulletin of the atomic scientists, v. 12, March 1956: 76–80.

Holzman, B. The effects of atomic bomb explosions on weather. Weatherwise, v. 4, February 1951: 3–4 f.

Holter, N. J. and Glasscock, W. R. Tracing nuclear explosions. Nucleonics, v. 10, August 1952: 10–13.

Airborne radioactivity precipitated in rain and snow has been measured by counter observations on samples concentrated by evaporation or filtration through cotton. A maximum half life of 10.6 hours (Pb^{212}) is associated with natural atomspheric activity. A number of samples collected (at Helena, Mont.) revealed activities of much longer decay periods. These are attributed to atomic explosions in Nevada and Russia. It is considered possible to assign a date to the occurrence of the explosion from observations of the decay curve.

Humphrey, Andrew J. Radiation injury: a technical and legal survey. Cleveland—Marshall law review, v. 6, May 1957: 171–188.

Examines the relations between various types of radiation as to sources and effects.

Hunter, H. F. and Ballou, N. E. Fission-product decay rates. Nucleonics, v. 9, November 1951: C–2–C7.

Jaffee, Gilbert, and others. Radioactive hailstones in the District of Columbia, May 26, 1953. Bulletin of the American Meteorological Society, v. 35, June 1954: 245–249.

At 2030 Greenwich civil time on May 26, 1953, hailstones ranging to the size of tennis balls fell in the District of Columbia, 29 hours after an atomic test in Nevada. Activity of 620 c/m as compared to a background of 20 c/m was measured in the stones. Meteorological analysis and decay curves confirmed the origin of the radioactivity as being from the atomic test.

Kellogg, D. A. Atomic defense in oil refinery. Petroleum engineering, v. 27, October 1955: C6–C8.

Methods of preventing continuous effects of radioactive fallout.

Kellogg, W. W., and others. Close-in fallout. Journal of meteorology, v. 14, February 1957: 1–8.

Keosian, John. Speculation on hazards of exposure to radiations. Science, v. 122, September 30, 1955: 586–587.

Question of maximum tolerance dose of radiation for man has not been satisfactorily determined. It may turn out that all high energy radiation, even of low intensity and brief duration must be considered as potentially dangerous to the exposed individual.

Kilcawley, E. J. Measurement of radioactive fallout in reservoirs. Journal of the American Water Works Association, v. 46, November 1954: 1101–1111.

An extensive survey of radioactivity in the Troy, Albany, Schenectady water supply system was made in an investigation following the heavy rainout which occurred in that area on April 26, 1953. Samples of water, soil, algae, plants, etc., were measured for radioactivity. Decay rates, effectiveness of filtration, rate of disappearance, etc., were studied. The general contamination of the ground at arrival time was $1\mu c/ft^2$. Surface water at Rensselaer Polytechnic Institute, Troy, N. Y., had 2.7×10^{-2} $\mu\mu c$/ml. Measurements were also made for subsequent bursts. Corrected to time of rainout, highest rainwater activity was 25.0 $\mu\mu c$/ml on June 9, 1953. Highest stream samples, 0.10–.13 $\mu\mu c$/ml. Alpha activity of same samples was also investigated.

Kirby-Smith, J. S. and Swanson, C. P. The effects of fast neutrons from a nuclear detonation on chromosome breakage in tradescantia. Science, v. 119, January 1, 1954: 42–46.

Physical determination of the fast neutron dose in nuclear explosions; supplementary experiments to those of Conger.

Krumholz, Louis A. Observations of the fish population of a lake contaminated by radioactive wastes. Bulletin of the American Museum of Natural History, v. 110, article 4, 1956: 281–367.

Kulp, J. Laurence, and others. Strontium–90 in man. Science, v. 125, February 8, 1957: 219–225.

To determine amount of radioactive strontium in human bones today, three scientists from Columbia University analyzed about 500 autopsy samples obtained from 17 stations in a worldwide network. Concludes that if bomb tests continue at their present rate the average worldwide concentration in 1970 will be 4 to 8 micromicrocuries of strontium–90 per gram of calcium. The upper figure approaches the significant level established by the British Medical Council in its June 1956 report.

Lacassagne, A. The risks of cancer formation by radiations. Bulletin of the atomic scientists, v. 13, April 1957: 135–136f.

Langham, Wright H., and Anderson, F. C. Strontium–90 and skeletal formation. Science, v. 126, August 2, 1957: 205–206.

Lapp, Ralph E. Strontium–90 in man. Science, v. 125, May 10, 1957: 933–934.

Commentary on Kulp article in Science, February 8, 1957.

Reply by Kulp and others: 934.

Lewis, E. B. Leukemia and ionizing radiation. Science, v. 125, May 17, 1957: 965–972.

Discusses incidence of leukemia in Hiroshima and Nagasaki, also cases among radiologists. Applied to radiostrontium exposures.

Libby, Willard F. Current research findings on radioactive fallout. Proceedings of the National Academy of Sciences, v. 42, December 1956: 945–962.

Speech given before American Association for the Advancement of Science, October 12, 1956.

———. Dosages from natural radioactivity and cosmic rays. Science, v. 122, July 8, 1955: 57–58.

Reprinted in Congressional Record (Daily ed.) July 14, 1955: A5165–A5166.

———. Genetic effects of atom bombs. Metal progress, v. 68, October 1955: 130–131.

Libby, Willard F. Radioactive strontium fallout. Proceedings of the National Academy of Sciences, v. 42, June 1956: 365–390.

Based on a speech given before Annual General Meeting of the American Philosophical Society, April 20, 1956.

Hazards from Sr^{90} deposited in fallout following nuclear explosions are reviewed. $Strontium^{90}$ is of particular interest among the fission products because of chemical similarity to Ca, an average life of about 40 years, and a low rate of skeletal elimination. The maximum permissible average concentration of Sr^{90} in the adult skeleton is calculated to be $1\mu c/1,000$ gm of Ca.

Data are summarized on Ca, Sr, and Sr^{90} concentration in samples of soil, animal, and plant material collected throughout the world before and after the thermonuclear explosions during Operation Castle. A Sr^{90} fallout probably derived from megaton weapons are nearly uniform over the world except for local effects due to rainfall variations and to fallout from submegaton weapons, was found to occur at least 1.7 years after the megaton test series. The average world-wide Sr^{90} fallout rate in the fall of 1954 and the spring and summer of 1955 was 1.2 mc/mi²/yr. An estimate is presented of fallout rate of Sr^{90} to be expected from weapons tests up to and including the Castle series. Factors influencing the transfer of Sr^{90} from soil to plants, to animals and milk produced by them, and finally to the human skeleton are discussed.

Lieberman, Joseph A. Disposal of radioactive wastes—a growing problem. Civil engineering, v. 25, July 1955: 44–47.

List, Robert J. On the transport of atomic debris in the atmosphere. Bulletin of American Meteorological Society, v. 35, September 1954: 315–325.

Describes results of 91 gummed paper and 51 air filter monitoring stations in the United States during Nevada tests in the spring of 1952. Detailed meteorological trajectories of each of the bursts of the series are given, together with a discussion of the meteorological aspects of transport and fallout. Excluding area within 200 miles of test site, highest gummed paper fission product beta activity was 8×10^{6} d/m/ft²/day at station 330 miles from test side. At distances over 2,000 miles, maximum activity was 1.7×10^{5} d/m/ft²/day, on sampling day. The two highest air filter activities were 1.3×10^{5} and 6.8×10^{3} d/m/meter³. Detailed discussion of fallout from 3 of the 8 bursts given, with daily maps showing isolines of activity and areas of precipitation for several days following the bursts.

———. On the transport of atomic debris in the atmosphere. Journal of the Air Pollution Control Association, v. 5, 1955: 153–156f.

The author correlated the meteorological trajectories of bomb debris following each of the eight nuclear detonations at the Nevada test site in 1952 with fallout in the United States. In most instances the predicted pattern of fallout was in agreement with fallout data.

Luntz, Jerome D. Radiation safety for a weapons test. Nucleonics, v. 10, May 1952: 10–13.

An eyewitness report on the elaborate system used to obtain detailed data on distribution of radioactivity from the atomic explosion on April 22, 1952.

Machta, L. and Harris, D. L. Effects of atomic explosions on weather. Science, v. 121, January 21, 1955: 75–81.

A study of temperature and rainfall for the United States does not indicate any departures from normal that are related to atomic explosions.

Machta, L., and others. Worldwide travel of atomic debris. Science, v. 124, September 14, 1956: 474–477.

Machta, L., and others. Airborne measurements of atomic debris. Journal of meteorology, v. 14, April 1957: 165–175.

Margolis, Emanuel. The hydrogen bomb experiments and international law. Yale law journal, v. 64, April 1955: 629–647.

Detailed consideration of influence of such international law doctrines as freedom of the seas and the illegality of the "pollution" of international waters on the H-bomb test in the Pacific.

McDougal, M. S. Hydrogen bomb tests and the international law of the sea. American journal of international law, v. 49, July 1955: 356.

——— and Schlei, N. A. Hydrogen bomb tests in perspective: lawful measures for security. Yale law journal, v. 64, April 1955: 648–710.

Discusses conflicting claims of the security of the United States and its allies and the principles of international law as interpreted in some quarters.

Meinke, W. Wayne. Observations on radioactive snows at Ann Arbor, Mich. Science, v. 113, May 11, 1951: 545–546.

Rigorous chemical separations performed on radioactivities found in snows around Ann Arbor, Mich., after the Las Vegas atomic test explosions on January 27 to February 6, 1951, have definitely established the presence of radioactive rare-earth isotopes and Ba and/or Sr isotopes and have shown the possible presence of I isotopes. The tests conducted on the samples are described. Because of the chemical distribution of the activities found in the Ann Arbor snows, these activities undoubtedly originated in the Las Vegas atomic test explosions.

Mesler, Russell B. and Widdoes, Lawrence C. Evaluating reactor hazards from airborne fission products. Nucleonics, v. 12, September 1954: 39–41.

Meteorological aspects of atomic radiation. Science, v. 124, July 20, 1956: 105–112.

One of six reports prepared for the Study of the Biological Effects of Atomic Radiation by the National Academy of Sciences.

Miller, C. E. and Marinelli, L. D. Gamma-ray activity of contemporary man. Science, v. 124, July 20, 1956: 122–123.

Moloney, William C. and Kastenbaum, Marvin A. Leukemogenic effects of ionizing radiation on atomic bomb survivors in Hiroshima City. Science, v. 121, February 25, 1955: 308–309.

Incidence of leukemia is "high" at distances close to the hypocenter, regardless of presence or absence of severe radiation complaints.

Morgan, K. Z. Maximum permissible internal dose of radionuclides: recent changes in values. Nuclear science and engineering, v. 1, December 1956: 477–500.

Muller, Hermann J. After effects of nuclear radiation. National safety news, v. 74, August 1956: 43–48. Bibliography.

———. Genetic damage produced by radiation. Science, v. 121, June 17, 1955: 837–840.

———. The genetic damage produced by radiation. Bulletin of the atomic scientists, v. 11, June 1955: 210 ff.

Article based upon Japanese analyses of fallout from the March 1, 1954, superbomb.

———. How radiation changes the genetic constitution. Bulletin of the atomic scientists, v. 11, November 1955: 329–338.

———. Radiation and human mutation. Scientific American, v. 193, November 1955: 58–68.

———. Radiation damage to genetic material, Parts I and II. American scientists, v. 38, 1950: 33, 399.

Nader, J. S., and others. Radioactive fallout in rain in the Cincinnati area. Journal of the American Water Works Association, v. 46, November 1954: 1096–1100.

Precipitation in the Cincinnati area was measured for suspended and soluble radioactivity from March 1953 to March 1954. "Background" level in precipitation was 0.03 to 0.08 $\mu\mu$c/ml. Maximum concentration, on April 29, 1953, 319 $\mu\mu$c/ml (500 $\mu\mu$c/ml corrected for decay). Maximum fallout from a single rain on May 22, 1953, 1.55 inches containing 85.7 $\mu\mu$c/ml, giving 8.75 curies per square mile. Soluble activity averaged 60–80 percent of total before and after tests, fell to 30 percent during tests. Samples from creeks and tapwater show most of activity removed by natural purification. Accumulated and decayed rain activity on June 10 and December 10, 1953, was 2.4 and 0.15 curies per square mile, respectively.

A Navy medical team studies fallout effects. Bulletin of the atomic scientists, v. 12, February 1956: 58–59.

Reprint of article on radiation research performed by doctors from Naval Medical Research Institute after 1954 nuclear tests in Marshalls. Original article in Research Reviews, November 1955. Also summarized in Science, v. 122, December 16, 1955: 1178–1179.

Neher, H. V. Gamma rays from local radioactive sources. Science, v. 125, May 31, 1957: 1088–1089.

New research facts on how foods weather A-bombing. Food engineering, v. 28, November 1956: 59f.

Oceanography, fisheries and atomic radiation. Science, v. 124, July 6, 1956: 13–16.

Part of a continuing study on the Biological Effects of Atomic Radiation conducted by the National Academy of Sciences.

Ophel, I. L. Fallout and the strontium-90 hazard. Science, v. 125, March 1, 1957: 399.

Plough, H. H. Radiation tolerance and genetic effects. Nucleonics, v. 10, 1952: 16–20.

Radiation and health (editorial). Science, v. 125, April 19, 1957: 719.

Questions necessity for establishing a National Radiation Health Institute in the Public Health Service. Points out that any radiation health agency should deal with radiation from all sources, not just atomic radiation.

Randolph, M. L., and others. Effect of bone-marrow treatment on mortality of mice irradiated with fast neutrons. Science, v. 125, May 31, 1957: 1083–1084.

Rediske, J. H. and Selders, A. A. The absorption and translocation of strontium by plants. Plant physiology, v. 28, 1953: 594–605.

Rosenfeld, A. H., and others. Fallout: some measurements and damage estimates. Bulletin of the atomic scientists, v. 11, June 1955: 213–216.

Rotblat, Joseph. The hydrogen-uranium bomb. Bulletin of the atomic scientists, v. 11, May 1955: 171–172, f.

Speculation on the composition and possible radiological effects of the superbomb tested in the Pacific in 1954.

Russell, W. L. Comparison of X-ray induced mutation rates in drosophila and mice. American naturalist, v. 90, January–February 1956: 69–80.

Until recent years, estimates of genetic hazards of ionizing radiation in man were based primarily on information obtained from drosophila. Investigations on radiation-induced mutation rates in mice showed a higher mean rate in the mouse and led to conclusion that estimates of human hazards based on drosophila mutation rates may be too low.

———. Radiation in mice—the genetic effects and their implications for man. Bulletin of the atomic scientists, v. 12, January 1956: 19–20.

———. Shortening of life in the offspring of male mice exposed to neutron radiation from an atomic bomb. Proceedings of the National Academy of Sciences, v. 43, Apr. 1957: 324–329.

Schubert, Jack. Radioactive poisons. Scientific American, v. 193, August 1955: 35–39.

The biological effects of nuclear radiation are as yet imperfectly understood. Maximum permissible limits of exposure must be determined more precisely if byproducts of nuclear technology are to be safely handled.

Setter, L. R., and Goldin, A. S. Radioactive fallout in surface waters. Industrial and engineering chemistry, v. 48, February 1956: 251–255.

Slatis, Herman M. Current status of information on the induction of mutations by irradiation. Science, v. 121, June 10, 1955: 817–821.

Some biological effects of radiation from nuclear detonations. American naturalist, v. 88, 1954: 209–314.

Stefannizzi, A. Radioactivity of atmospheric precipitates. Journal of geophysical research, v. 55, 1950: 373–378.

The radioactivity of snow and rain was detected in 33 cases. It was found that snow usually has a greater activity than rain; that rain in thundershowers is more active than ordinary rain; and that at least a certain amount of activity is acquired by precipitates during their fall from the clouds to the ground level.

Strauss, Lewis L. Fallout from an H-bomb. Metal progress, v. 67, May 1955; 98–99.

———. Radioactive fallout (statement released February 15, 1955) Armed Forces chemical journal, v. 9. March–April 1955: 43–44.

Sturtevant, A. H. The genetic effects of high energy irradiation of human population. Engineering and science, v. 18, January 1955: 9–12.

———. Social implications of the genetics of man. Science, v. 120, September 10, 1954: 405–407.

Tajima, Eizo and Doke, Tadayoshi. Airborne radioactivity. Science, v. 123, February 10, 1956: 211–214.

Twenty-four hour air filters in Tokyo from March 16 to May 31, 1955, were measured for artificial and natural radioactivity. The samples consisted of 650 cubic meters per day and assuming a 10 percent collection efficiency of the Whatman No. 14 filter papers for small particles, the peak activity from fission products, observed on April 12, was $3 \times 10^{-15} \sim 1.2 \times 10^{-14}$ curies per liter, which is comparable to the concentration of natural activity. No consistent correlation of fission product activity with temperature was found and only a slight tendency for rainfall to clean the air was noted. A better correlation was observed with trajectories of high pressure areas.

Thomas, Harold A., and others. Radioactive fallout in Massachusetts surface waters. Journal of the American Water Works Association, v. 45, June 1953: 562–568.

Following the Nevada nuclear-weapons tests, tests were made in Massachusetts which showed a noticeable increase in radioactivity in rain falling. Results are tabulated and the presence of natural radioactivity is considered. On standing there seems to be a settling out of radioactive products.

Thompson, Raymond. They play tag with atomic clouds. Science digest, v. 38, August 1955: 9–12.

Work of planes and crews from Air Force's Air Research and Development Command in tracking radioactive clouds after test atomic explosions.

Toombs, Alfred. Radioactive "garbage"—newest threat to man and nature. Natural history, v. 65, September 1955: 344–349.

Tsivoglov, E. C., and Towne, W. W. Sources and control of radioactive waste pollutants. Sewage and industrial wastes, v. 29, Feb. 1957: 143–156. Bibliography.

Turekian, K. K. and Kulp, J. Lawrence. Strontium content of human bones. Science, v. 124, August 31, 1956: 405–407.

U. N. appoints committee to study effects of ionizing radiation. Bulletin of the atomic scientists, v. 12, Jan. 1956: 13.

Uses and effects of atomic radiation. Scientific monthly, v. 84, January 1957: 3–25.

Contents: Radiation and the human body. Radiation and genetics. Uses of atomic radiation and energy. What we most need to know.

Van Middlesworth, L. Radioactivity in thyroid glands following nuclear weapons tests. Science, v. 123, June 1, 1956: 982–983.

Radioactivity reported and confirmed in thyroid glands of cattle, presumably from fallout.

———. Impact of atomic energy on the life sciences. Technology review, v. 57, July 1955: 471–472.

Primarily concerned with detrimental effects of radiation on human beings.

Warren, Shields. Antipersonnel effects of nuclear weapons. Confluence, v. 5, July 1956: 131–138.

———. Radiation and the human body. Scientific monthly, v. 84, January 1957: 3–6.

Symposium paper presented at all-day scientific program on the uses and effects of atomic radiation held by the American Association for the Advancement of Science, October 12, 1956 at the Carnegie Institution of Washington.

Webb, J. H. The fogging of photographic film by radioactive contaminants in cardboard packaging materials. Physical review, v. 76, August 1, 1949: 375–380.

After the detonation of the experimental atom bomb at Alamogordo, N. Mex. on July 16, 1945, a radioactive contaminant was encountered in strawboard material used by the Eastman Kodak Co. for packaging photographic sensitive films. This paperboard was manufactured in a mill situated at Vincennes, Ind., on the Wabash River. A run of strawboard, produced on August 6, 1945, showed this new and unusual type of radioactive contaminant.

Weiss, H. V. and Shipman, W. H. Biological concentration by killer clams of cobalt-60 from radioactive fallout. Science, v. 125, April 12, 1957: 695.

Woollam, D. H. M. and Millar, J. W. Why are children born deformed? Science news, v. 41, 1956: 27.

Wright, J. H., and others. High-speed computer for predicting radioactive fallout. Journal of research of the National Bureau of Standards v. 58, February 1957: 101–109.

Yoshii, G., and others. Biological decontamination of fission products. Science, v. 124, August 17, 1956: 320–321.

MEDICAL JOURNAL ARTICLES

Atomic bomb casualties; general effects of exposure to radiation. Cancer bulletin (Texas ed.) v. 7, May–June, 1955: 55–58.

Bergsma, Daniel. Public health aspects of atomic energy in peacetime. Public health reports, v. 71, January 1956: 43–46.

Bond, V. P., and others. Hematological changes in human beings exposed to fallout radiation. Radiation research, v. 3, October 1955.

Presented before the Radiation Research Society, New York, May 16–18, 1955.

All individuals exposed to fallout radiation showed depression of the total white, lymphocyte, neutrophil, and platelet counts, more marked in the higher exposure groups. The total white count was consistently lowest during the sixth and seventh postexposure, followed by an upward trend. Six months after exposure, values for the highest exposure group had not returned to the levels of a control population comparable with respect to age and sex distributions.

Clark, Dwight E. Association of irradiation with cancer of the thyroid in children and adolescents. Journal of the American Medical Association, v. 159, 1955: 1007–1009.

Cowan, F. P., and others. Health physics and medical aspects of a strontium 90 inhalation incident. American journal of roentgenology, radium therapy and nuclear medicine, v. 67, May 1952: 805–809.

Cronkite, Eugene P. Radiation injuries of atomic warfare; pathogenesis and therapy. Journal of the Omaha Midwest Clinical Society, v. 13, January 1952: 6–13.

———. and Brecher, G. Radioactivity; effects of whole body irradiation. Annual review of medicine. v. 3, 1952: 193–214.

———. and others. Response of human beings accidently exposed to significant fallout radiation. Journal of the American Medical Association, v. 159, October 1955: 430–434.

After detonation of a thermonuclear device in the Marshall Islands in the spring of 1954, radioactive fallout occurred over an area of thousands of square miles beyond the range of thermal and blast injury. Marshallese and Americans were accidentally exposed on Islands in the area, receiving whole-body radiation, beta radiation injury to skin, and minimal internal contamination.

DeCoursey, E. Ionizing radiation injury from atomic bombs. Journal of the Kentucky Medical Association, v. 50, September 1952: 391–393.

Dunning, Gordon M. Criteria for evaluating gamma radiation exposures from fallout following nuclear detonations. Radiology, v. 66, April 1956: 585–594.

———. Protecting the public during weapons testing at the Nevada test site. Journal of the American Medical Association, v. 158, July 16, 1955: 900–904.

Folley, J. H., and others. Incidence of leukemia in survivors of the atomic bomb in Hiroshima and Nagasaki, Japan. American journal of medicine, v. 13, September 1952: 311–321.

The genetic effects of the atomic bomb. Cancer bulletin (Texas ed.) v. 7, July–August 1955: 70–72.

Genetics and radiation. Eugenics quarterly, v. 3, September 1956: 131–138; December 1956: 201–208.

Contents: Genetics in the atomic age, by Curt Stern. The estimation of spontaneous and radiation-induced mutation rates in man, by James F. Crow.

Glass, Bentley. The hazards of atomic radiations to man. Journal of heredity, v. 47, 1956: 260.

Greulich, W. W., and others. Physical growth and development of children who survived bombing of Hiroshima or Nagasaki. Journal of pediatrics, v. 43, August 1953: 121–145.

Hamilton, Joseph G. Metabolism of radioactive elements created by nuclear fission. New England journal of medicine, v. 240, 1949: 863–870.

Data are presented on the oral absorption, principal organ of retention, and accumulation in and rate of elimination from the organ of Pu^{23}, Np^{239}, and the long-lived fission products.

Haymond, Herman R. Radiation hazards in medical practice. University of Southern California medical bulletin, v. 9, Winter 1957: 10–15.

Henny, G. C. Radiation protection problems in diagnostic roentgenology. American journal of roentgenology, radium therapy and nuclear medicine, v. 73, 1955: 649.

Henshaw, P. S. and Hawkins, J. W. Incidence of leukemia in physicians. Journal of the National Cancer Institute, v. 4, February 1944: 339–346.

Heustis, Albert E. and Van Farowe, Donald. X-ray radiation study shows hazards can be controlled. Hospitals, Journal of the American Hospital Association, v. 25, August 1951: 72–80.

Kirsh, I. E. Radiation dangers in diagnostic radiology. Journal of the American Medical Association, v. 158, 1955: 1420.

Lange, R. D., and others. Refractory anemia occurring in survivors of the atomic bombing in Nagasaki, Japan. Blood; the journal of hematology, v. 10, April 1955: 312–324.

Laug, E. P. Radioactive fallout and our food supply. Antibiotic medicine, v. 2, February 1956: 89–91.
Leukemia in atomic bomb survivors. I. General observations, by R. D. Lange and others. Blood; the journal of hematology, v. 9, June 1954: 574–585. II. Observations on early phases of leukemia, by W. C. Moloney and R. D. Lange. Ibid., July 1954: 663–685.
Looney, W. B. Late effects (25 to 40 years) of the early medical and industrial use of radioactive materials. Journal of bone and joint surgery, v. 37–A, December 1955: 1169–1187; v. 38–A, January 1956: 175–218; v. 38–A, April 1956: 392–407.
Lough, S. Allan. Radiological health—an over-all view. American journal of public health, v. 45, November 1955: 1447–1453.
Macht, S. J. and Lawrence, P. S. National survey of congenital malformations resulting from exposure to roentgen radiation. American journal of roentgenology, radium therapy and nuclear medicine, v. 73, March 1955: 442–466.
Maher, Thomas F. Nature and significance of radioactive fallout. New England Journal of medicine, v. 252, 1955: 740–741.

Principal hazards of radioactivity produced by detonations of atomic weapons are I (half-life 11.5 days) and Sr (Half-life approximately 30 years). General protective measures that can be taken against radioactive fallout are shelter and decontamination, and the avoidance of ingestion or inhalation of contaminated mediums from the environment. Persons who have been exposed to radioactive fallout may show burns, hematologic and bone-marrow changes, and infection.

Maximum permissible radiation exposures to man; a preliminary statement of the National Committee on Radiation Protection and Measurement. A special report. A. M. A. Archives of industrial health, v. 15, April 1957: 350–355.

Also in Radiology, v. 68, February 1957: 260–261.

Maxwell, C. L. The thermonuclear fallout problem in the State of Illinois. Illinois medical journal, v. 108, November 1955: 269–273.
Mewissen, D. J. and others. A formula for chronic radiation dosage versus shortening of life span: application to a large mammal. Radiation research, v. 6, April 1957: 450–459.
Miller, Robert W. Delayed effects occurring within the first decade after exposure of young individuals to the Hiroshima atomic bomb. Pediatrics, v. 18, July 1956: 1–8.
Moeller, Dade W., and others. Radiation exposures in the United States. Public health reports, v. 68, January 1953: 57–65.
Muller, Hermann J. Comments on the genetic effects of radiation on human population. Journal of heredity, v. 46, 1955: 199–200.
———. Damage to posterity caused by the irradiation of the gonads. American journal of obstetrics and gynecology, v. 67, 1954: 467–483.
Powell, Clinton C. Medical aspects of the control of radiologic health problems. American journal of public health, v. 47, February 1957: 179–183.
Reynolds, Mardelle L. and Lynch, Francis X. Atomic bomb injuries among survivors in Hiroshima. Public health reports, v. 70, March 1955: 261–270.
Rolleston, H. Critical review of the harmful effects of irradiation (X-rays and radium). Quarterly journal of medicine, v. 23, 1930: 101–132.
Russell, L. B. and Russell, W. L. Radiation hazards to the embryo and fetus. Radiology, v. 58, 1952: 369–376.
Salvin, M. Effect of atomic radiation on the incidence of sterility and mutation; Journal of the National Medical Association, v. 48, March 1956: 79–83.
Stoll, B. A. New drugs for irradiation sickness. Journal of radiology, v. 68, March 1957: 380–385.
Stone, R. S. The concept of a maximum permissible exposure. Radiology, v. 58, 1952: 639–661.
Storer, J. B., and others. Relative biological effectiveness of various ionizing radiations in mammalian systems. Radiation research, v. 6, February 1957: 188–288.
Straub, C. P. Limitations of water treatment methods for removing radioactive contaminants. Public health reports, v. 70, September 1955: 897–904.
Sutow, Wataru W., and West, Emory. Studies on Nagasaki (Japan) children exposed in utero to the atomic bomb. A roentgenographic survey of the skeletal system. American journal of roentgenology, radium therapy and nuclear medicine, v. 74, September 1955: 493–499.

Taliaferro, William H. and Taliaferro, Lucy G. Effect of X-rays on immunity: a review. Journal of immunology, v. 66, February 1951: 181–212.

Review documented by 201 references.

Taylor, Lauriston S. Education in radiation protection. American journal of roentgenology, radium therapy and nuclear medicine, v. 73, February 1955: 193–202.

———. Legislative control of radiation. Radiology, v. 66, February 1956: 246–252.

"Two States already have radiation-control regulations, and several others are in the process of developing them * * * The National Committee on Radiation Protection feels that legislation should cover both occupational and non-occupational exposure and all kinds of radiation * * * The Committee believes that the maintenance of the maximum degree of uniformity between the regulations in all States is of prime importance."

———. State control of protection against ionizing radiation. American journal of roentgenology, radium therapy and nuclear medicine, v. 71, April 1954: 691–702.

Thomas, Harold A. The public health implications of radio-active fallout in water supplies. American journal of public health, v. 46, October 1956: 1266–1274.

Warren, Shields. Public health aspects of atomic power development. A. M. A. Archives of industrial hygiene and occupational medicine, v. 9, March 1954: 183–185.

———. Longevity and causes of death from irradiation in physicians. Journal of the American Medical Association, v. 162, September 29, 1956: 464–468.

Williams, E. G., and Ingraham, S. C. Biological-medical considerations in atomic defense. Public health reports, v. 71, February 1956: 173–180.

Wilson, Robert R. Nuclear radiation at Hiroshima and Nagasaki. Radiation research, v. 4, 1956: 349–359.

Use of X-rays and other ionizing radiation; U. N. Scientific Committee on Effects of Atomic Radiation. Armed Forces medical journal, v. 8, March 1957: 358–360.

Vorder Bruegge, C. F. Radiation injury following A-bomb explosion. Annals of internal medicine, v. 36, June 1952; 1444–1458.

Ward, L. J. Lethal radiation from exploded atomic bomb. Mississippi Valley medical journal, v. 74, November 1952: 191–195.

Yamazaki, J. N., and others. Symposium on effects of radiation and other deleterious agents on embryonic development; study of outcome of pregnancy in women exposed to atomic bomb blast in Nagasaki. Journal of cellular and comparative physiology (supp. 1) v. 43, May 1954: 319–328.

Zavon, M. R. Radiation—helpful or harmful? Journal of the American Medical Association, v. 162, 1956: 533.

Technical Periodical Articles, Foreign

BRITISH

Auerbach, Charlotte. Biological hazards of nuclear and other radiations. Nature (London) v. 178, September 1, 1956: 453–454.

A comparison is made of British and United States reports on radiation hazards. Both reports show that the present dangers arise much more from excessive use of X-rays than from fallout or atomic energy establishments.

Biological effects of radiation. Nature (London) v. 179, April 13, 1957: 755–756.

Blifford, Irving H., and others. Relation between air concentration of radioactive fission products and fallout. Nature (London) v. 177, 1956: 990–992.

The daily atmospheric concentration and fallout of radioactivity due to fission products was measured at Washington, D. C., during December 1954–May 1955 by air-filter and gummed-paper techniques, respectively. The apparent rate of decay was used to distinguish between natural Th B and fission products. There was no correlation of individual daily measurements of air concentration and fallout of this material on the ground. Despite many variables the concept of fallout rate may be useful in arriving at some correlation.

Chatterjee, Santimay. Radioactive ashes over Calcutta and a method of dating a nuclear explosion. Atomic scientists journal (London) v. 4, 1955: 273–278.

Cockcroft, John D. Biological effect of nuclear explosions. Pharmaceutical journal (London) v. 174, 1955: 387–388 f.

———. Radiological hazards from nuclear explosions and nuclear power. Nature (London) v. 175, May 21, 1955: 873–875.

Reprint of address to the Parliamentary and Scientific Committee at the House of Commons, London, April 20, 1955.

Average concentration of radioactivity in the air at ground level over the last 3 years due to bomb explosions is about 1 percent of the natural radioactive dust content. Accumulated dose to completely unprotected people from fallout in England is about 0.01 R. Further fallout from airborne debris should bring it to 0.03 R. In the United States, average dose is about 0.1 R. Average dose in England to average person over a generation (30 years) will be about 0.003 R, natural radioactivity gives 3 R. Discusses other radiological hazards and concludes that probably at least 1,000 times the present level of contamination would be needed to give rise to serious harmful effects.

Howard, Alma. The hazards from the increasing use of ionizing radiations: A symposium. III. An attempt to assess the genetic changes resulting from the irradiation of human populations. British journal of radiology, (London) v. 29, June 1956: 270–273.

Leukaemia and natural background radiation. British medical journal, issue 5021, March 30, 1957: 760.

Nishiwaki, Yasushi. Bikini ash. (Letter to the editor.) Atomic scientists journal (London) v. 4, November 1954: 97–109.

Account of the results of the shower of radioactive ash that followed the explosion of the H-bomb at Bikini. Effects of the fallout on the Japanese fishermen, on the fish in the sea, etc., are considered.

———. Effects of H-bomb tests in 1954. Atomic scientists journal (London) v. 4, May 1955: 279–288.

Preston, R. L. and Hogg, B. G. Radioactive fallout in Kingston, Canada. Nature (London), v. 176, 1955: 459.

The monitoring of the radioactive fallout in Kingston, Ontario, during the period February 15 to May 28, 1955, from the nuclear tests conducted by the AEC is reported. Meteorological factors seem to outweigh the magnitude of the bombs at such a great distance from the explosion.

Poyce, M. H. L. Global thermonuclear explosions are impossible. Discovery (Norwich, England) v. 16, December 1955: 495–497.

Some people are afraid that a thermonuclear explosion might set off a reaction in the earth's crust and in the seas, resulting in the earth's destruction. The author of this article attempts to show why this is not possible.

Radiation hazards. Lancet (London), v. 270, June 23, 1956: 999–1000.

Radiation hazards of experimental nuclear explosions. British medical journal (London) No. 4916, March 26, 1955: 775–776.

Read, John. The approach of the physicist to radiation biology. Physics in medicine and biology (London) v. 1, January 1957: 209–224.

Rotblat, Joseph. The hydrogen-uranium bomb. Atomic scientists journal (London) v. 4, March 1955: 224–228.

This British scientist feels that the various bombs that have been detonated throughout the world have resulted in more dangers from radioactivity than officials admit. He believes that genetic aberrations may well result from present radioactivity in the atmosphere.

Sevitt, S. The bombs. Lancet, v. 269, July 23, 1955: 199–201.

———. The case of Mr. Kuboyama; a clinical and pathological study of the first victim of the atomic bomb. Medical world (London), v. 84, May 1956: 385–390.

Spiers, F. W. Radioactivity in man and his environment. British journal of radiology (London), v. 29, August 1956: 409–417.

———. and Haldane, J. B. Genetical effects of radiation from products of nuclear explosions. Nature (London) v. 177, February 4, 1956: 226–227.

Relative radiation dose-rates to man and to Drosophila are discussed. Data previously presented by Prof. J. B. S. Haldane on the genetical effects of radiation resulting from nuclear explosions are reviewed. A reply from Professor Haldane presents revised calculations of radiation dose rates.

Stanford, R. W., and Vance, J. The quantity of radiation received by the reproductive organs of patients during routine diagnostic X-ray examinations. British journal of radiology, v. 28, May 1955: 266–273.

Strontium-90 in man. (Letters to the editor.) British medical journal, issue 5024, April 20, 1957: 943–944.

Strontium-90 in man. British medical journal issue 5021, March 30, 1957: 752–753.

Sutton, G. Thermonuclear explosions and the weather. Nature (London) v. 175, February 19, 1955: 319–321.

Available evidence points to conclusion that atomic tests cannot be held responsible for any worldwide extremes of weather encountered in 1954.

FRENCH

Abribat, Marcel and Pouradier, Jacques. Evolution of the amount of artificial radioactive elements in the atmosphere of Paris. Comptes rendus, v. 237, 1953: 1233–1255 (in French).

The β-activities of airborne dust particles, rain waters, and natural waters were assayed daily between January and September 1953. The dust-particle activity showed 2 maximum March 24–June 25 and August 30–September 17, which probably resulted from atomic explosions in the United States and Russia, respectively. Rainwater activity gave only the earlier maximum while no changes in the activity of natural waters were found.

———, and others. Artificial radioactivity in rainwater of the Paris area. Comptes rendus v. 234, March 10, 1952: 1161–1163 (in French).

Considerable radioactivity was observed 8–15 days after the explosion of atomic bombs in Nevada.

——— ———. Evolution of atmospheric radioactivity in Paris region. Comptes rendus v. 240, 1953: 2310–2312 (in French).

Daily measurements of radioactivity have shown the passage of many atomic clouds, and particularly the series of explosions in the United States of America and Russia, while those in the Pacific and Australia have been identified in Milan. For the Australian explosion in October 1953 there was no radioactive increase in the air in the Paris region, while for the Pacific explosion there were measurable fluctuations but very feeble. For the Russian explosions in August 1954 the fluctuations were much greater than for the Pacific ones.

——— and others. On the artificial radioactive products present in the atmosphere in the region of Paris. Comptes rendus hebdomadaires des séances de l'Académie des Sciences (Paris) v. 235, July 16, 1952: 157–159(in French).

The radioactivity of solid matter in rain water and air collected near Paris in November 1951, and in April and May 1952, follows the same decay law as that observed for fission products after a nuclear detonation in Nevada in November 1951.

Besson, A. and Pelletier, J. Radioactivity and air pollutions. Bulletin de L'Académie Nationale de Médecine (Paris), par. 140, January 24–31, 1956: 43–45.

Bouquiaux, J. Ionizing radiations and public health problems. Archives Belges de médecine sociale, hygiéne, médecine du travail et médecine légale (Brussels). v. 14, May 1956: 230–268 (in French).

Garrigue, Hubert. The abnormal radioactivity of the atmosphere. Comptes rendus, v. 235, 1952: 1489–1499 (in French).

Further measurements on the radioactivity of the atmosphere near Puy de Dome are tabulated. The activity in April 1952 with a mean life of 25 hours was ascribed to the complex particles A. In June 1952 an activity with a mean life of 100–400 hours was attributed to the previously observed A″ particles. The activity associated with the A″ particles was also observed in the soil and air.

———. Atmospheric radioactivity of atomic origin. Comptes rendus, v. 237, November 16, 1953: 1232.

Aircraft measurements in October 1953, show at most, traces of radioactivity. Snow sample (7 kg) on November 3, 1953, yielded 0.1 x 10^{-18} curies/cm ^{3} (radon equivalent) in the residue, about 20 percent above background. Only β—activity was found.

———. Atmospheric radioactivity of atomic origin. Comptes rendus, v. 240, January 10, 1955: 178–180 (in French).

An improved collector has been installed in the aerial laboratory, but in recent months little activity caused by atomic explosions has been detected.

Garrigue, Hubert. Establishment of a flying laboratory and the improvement of apparatus for the study of weak radioactivity in the atmosphere. Comptes rendus, v. 230, June 26, 1950: 2279–2280 (in French).

Describes in general terms the equipment of a plane to measure the radioactivity of the air. The first flight, on May 13, 1950, showed a high concentration of natural activity and a measurable amount of activity of unknown origin with a long half-life.

———. Fresh outbreak of activity of atomic origin in the atmosphere. Comptes rendus, v. 240, 1955: 1453–1455 (in French).

During the period December 1954 to February 1955 values of the order of 1 β-emitting atom per cm^3 of air have been obtained for observations on the ground and in the air in Puy de Dôme region.

———. The invasion of radioactive air of atomic origin and its influence on atmospheric precipitation. Comptes rendus, v. 232, March 5, 1951: 1003–1004 (in French).

Reports on aircraft observations in June 1950 and in January and February 1951. On February 3, 1951, at 4,100 meters, a maximum concentration of 0.15×10^{-18} curies/cm^3 radon equivalent of material of 30–50 hour apparent half-life. Attempts to show that precipitation cleanses the tropospheric air.

———. Observations on the impurities in free air. Comptes rendus, v. 236, 1953: 2309–2311 (in French).

Results of analysis of atmospheric radioactivity and pollution from measurements on free air and on atomic precipitation (snow) at heights from ground level to the summit of the Puy de Dôme (1450 m) during January-February 1953. The radioactivity was probably of distant origin (from nuclear fission explosions); the dust and soot of local origin.

———. On the radioactivity of the atmosphere. Comptes rendus, v. 228, May 16, 1949: 1583–1584 (in French).

A radioactive substance with a half-life of 25 hours was detected in aircraft flights at about 6,000 meters in the summer of 1946 and again, in much weaker concentration, in the summer of 1948. Highest value reported, August 1, 1946, at 6,000 meters, 2.0×10^{-18} curies/cm^3. Observations at Puy-de-Dôme, 1,500 meters, gave no definite positive results. Speculation that material is from Bikini tests or possibly of meteoric or cosmic ray origin.

———. Prospecting the radioactivity of the air. Comptes rendus, v. 237, October 12, 1953: 802–803 (in French).

Measurements aboard an airplane (3,000 meters altitude) indicated a sudden influx of radioactive particles "A" with a mean life of 25 hours on August 8 (traces) and, in appreciable amounts, on August 15, 28, and September 5, 1953. The maximum of intensity of about 0.1×10^{-18} equivalent curie of Rn activity/cc. probably prevailed around August 15. No activity could be detected prior to these dates in air or in snow on the summit of Puy-de-Dôme (1,460 meters), nor did the evaporation of hail (collected on the ground), precipitated during a "microcyclone," which occurred on August 8, disclose any abnormal activity.

———. Radioactivity of the atmosphere of atomic origin. Comptes rendus, v. 237, 1953: 1232–1233.

Recent measurements at the summit of Puy-de-Dôme indicate the radioactivity of the snow may be attributed to atomic explosions.

———. Research on atmospheric radioactivity. Comptes rendus, v. 238, 1954: 2074–2075 (in French).

In flight at 2,800 meters, traces of radioactive particles were gathered by means of a corona effect similar to that of Sella, and of long period, on April 24, 1954. At Puy-de-Dôme (1,465 meters) after a fall of rain and snow on May 3–4, 1954, samples of the fallen residue showed very feeble radioactivity of period 10 days or more. This radioactivity of the airborne particles was independent of their probable natural electrical charge and mobility and was indicative of an atomic cloud.

———. Researches in radioactivity at the top of Puy-de-Dôme. Comptes rendus, v. 233, December 3, 1951: 1447–1448 (in French).

Snow collected at Puy-de-Dôme which fell on November 19–20, 1951, showed β-activity about 43 percent above background with an apparent half-life of 10 days. Speculation on role of particles as condensation nuclei.

———. Studies on the radioactivity of the atmosphere. Comptes rendus, v. 233 Oct. 15, 1951: 860–862 (in French).

The existence in the atmosphere of a radioactive substance, labeled "A," of several hours half life has been confirmed in flights at 3,300 meters, and at a ground station at 1,460 meters. Concentrations of Rn, Tn, "A," and "A'" observed from March 15 to August 14, 1951, are tabulated. The concentration of substance "A," of 20- to 30-hour half life, is related to atomic explosions and precipitation.

Lacassagne, A. Medical consequences of atomic bomb explosions. Bruxelles-Médical, (Brussels) v. 35, September 11, 1955: 1821–1833 (in French).

Martin, Charles Noel. Accumulative effects on the global surface caused by thermonuclear explosions. Comptes rendus, v. 239, 1954: 1287–1289.

HNO_3 is formed which locally lowers the pH of rain water. This can affect plant metabolism. C^{14} production and its absorption by living things is discussed.

Nahmias, M. E. Detection at a distance of atomic bomb tests. Mem. Artillerie Franc. 28, 1954: 393–402.

Nahmias describes the radioassay of atmospheric air, rain, and snowfall as the basis for detecting atomic bomb tests. Tables and graphs give time versus Ra equivalents, distance versus Roentgens/hours of radiation, and distribution of radio-elements which can be expected.

Ravina, A. The first known effects of hydrogen bomb on man. Presse médicale (Paris), v. 62, June 5, 1954: 881 (in French).

Tanaevsky, Olga and Vassy, Etienne. Variations of the natural and artificial radioactivity of the atmosphere. Comptes rendus, v. 241, 1955: 38–40.

The natural activity of the atmosphere, presumably Rn, Tn and their decay products, was most evident during periods of weak-winds at the Val Joyeux Scientific Station. In 30 of the 66 cases of high activity, the winds were from the southwest or west southwest, indicating a Rn source in that direction. The artificial radioactivity, detected in rains and snows, was strongest at the beginning of the precipitation. The highest activity measured was 0.724 microcuries/1.

GERMAN AND AUSTRIAN

Gerlach, Walther. The hazards of radiation and its danger to life. Universitas (Stuttgart, Germany), v. 2, 1957: 125–131.

Haxel, O., and Schumann, G. On the radioactive contamination of the atmosphere. Naturwissenshaften (Berlin), v. 40, 1953: 458 (in German).

Beginning on March 19, 1953, radioactivity in the air near Heidelberg was measured continuously by means of air filtering, using, in general, 48-hour exposures. The long-lived activity, presumably from atomic explosions, showed several peaks in the period from mid-March to mid-June, reaching a maximum of 2.5 curies/m^3 in mid-April. An examination of the decay rates allows the determination of the time of explosion. It was found that fission products reached Heidelberg in as little as 7 days from the Nevada test site.

Herbst, W., and others. Considerations of the suitability of radioactive atomic aerosols as tracers in meteorological flow investigations. Naturwissenschaften (Berlin) v. 41, 1954: 156–160 (in German).

A large increase in radiation from the ground at Wittental (Brunswick), October 18–20, 1951, led to an attempt to trace the increased radioactivity to atom bomb explosions in the United States and the corresponding measurements by Holter and Glasscock at Helena, Mont. For the explosions on October 6, 7, and 14 the probable track of air at the 500 mb level from Helena to Wittental was determined in each case and shown on a chart.

——— and Philipp, K. The path of an atomic explosion aerosol. Naturwissenschaften (Berlin), v. 40, 1953: 54 (in German).

Experiments at Wittental show that during October 16–24, 1951, a high value of the radioactive background was discovered. A similar high value had been reported at Helena, Mont., between October 6–16, 1951. It is suggested that the same air mass was at these two places at the different times and that radioactivity measurements permit the path of the air mass in which the explosion occurred to be plotted.

Sittkus, A. Observations on radioactive vapour from atomic experiments in the years 1953/54. Naturwissenschaften, (Berlin) v. 42, 1955: 478–482 (in German).
Records data from rainfall at Freiburg, Germany, on the radioactivity of the atmosphere and describes a method of deducing the time of the explosion from the observations.

———. The path of an atomic explosion aerosol. Remarks of Mr. Sittkus to W. Herbst. Naturwissenschaften, (Berlin) v. 40, 1953: 198.
No comparable effect was detected on other geiger counters in the same neighbourhood at the time when Herbst and Philipp recorded the effect they believe due to an atomic bomb. In reply these authors pointed out that they used thin-walled counters to detect β-radiation, whereas Sittkus used tubes suitable for detecting the penetrating component of cosmic rays.

Steinhauser, F. Atomic bomb explosions and weather events. Universitas (Stuttgart) 1954: 1189–1196 (in German).

———. Atomic energy and world weather. Universum natur and technick (Vienna) v. 16, 1954: 481.

———. Atomic energy experiments and weather. Osterreichische Hochschulzeitung (Vienna) 6 jg., Wien 1954 (in German).

Tsuzuki, M. Radioactive damage of Japanese fishermen caused by Bikini ashes. Münchener medizinische wochenschrift (Munich), v. 97, August 5, 1955: 988–994 (in German).
Short description of clinical experiences with radioactive injuries of the 23 fishermen during 1 year. All of the 23 fishermen in the boat were afflicted with acute radioactive-sickness as a result of contact with radioactive rain and ashes. They were injured through the combination of external as well as internal radiation.

INDIAN

Bandopadhyay, K. G., and others. Radioactive nuclei in rains over Calcutta. Science and culture (India), v. 21, 1955: 273–275.
The radioactivity in dusts carried down by rains between March and September 1955 were determined by β-ray assay. Histograms of relative activities as a function of specific rainfalls are given.

Chatterjee, Santimay, and others. Dating a nuclear explosion. Science and culture, v. 20, 1955: 403–404.
A method is proposed for dating nuclear explosions from the composite beta-decay curves of the radioactive dusts.

——— ———. Measurements on radioactive dusts over Calcutta. Science and culture, v. 20, 1955: 399–401.
A brief report of the measurements of the radioactivity of rain water samples collected in Calcutta from April 29 to the middle of July 1954, is presented. Measurements of energy and half lives indicated that the dusts originated from nuclear explosions.

——— ———. Presence of radioactive dusts over Calcutta. Science and culture, v. 19, May 1954: 570–571.
Beginning April 4, 1954, settled dust in Calcutta was analyzed for radioactivity; none was found until the first rain occurred on April 29.

ITALIAN

Neuwirth, R. Meteorological utilization of measurements of the artificial radioactivity of the air and precipitation. Geofisica pura e aplicata (Milan, Italy) v. 32, 1955: 147–158. (In German.)
German, French, and American measurements of the rainfall and air activity are being evaluated. For that purpose, trajectories from the experimental grounds for bomb tests in Nevada to Western Germany are drawn. By means of intermediate values, the test possibilities of air paths—first only scheduled—are given. The so-called deposit spaces and meridional circulations, which are significant particularly in divergence regions, prove to be of especial importance. The mechanism of activation of precipitation is discussed. A connection between the activity of precipitation and air masses could only be found in individual cases. But it seems that semitropical air air masses dispose of a higher specific activity in comparison with the polar air masses.

Radioactive precipitations caused by the experimental explosion of atomic weapons. Minerva medica (Turin) v. 46, March 31, 1955: 640–642 (in Italian).

Santomauro, L. and Cigna, A. First measurements of the radioactivity in atmospheric precipitations. Annali di geofisca (Rome), v. 6, 1953: 381–387.

Measurements conducted between February 1951 and November 1952 showed that nuclear-weapon tests at Las Vegas, Eniwetok, and Montebello were followed, 1, 2, and 3 weeks later, respectively, by an increase in the radioactive content of rain and snow falling in Italy.

Spena, A. The genetic problem in its relation to the use of atomic energy. Annali di medicina navale e tropicale (Rome), v. 61, July–August 1956: 569–582.

JAPANESE

Arakawa, Akio, and others. Climatic abnormalities as related to the explosions of volcano and hydrogen-bomb. Geophysical magazine (Tokyo) v. 26, 1955: 231–255.

The effects of volcanic explosion on climatic abnormalities are investigated statistically and synoptically. The abnormal weather during the summer season of 1954 is found to have features similar to climatic abnormalities caused by volcanic dust. The distribution of temperature anomalies and its annual variation are discussed in relation to the tropospheric circulation.

Arakawa, H. Abnormal weather caused by the H-bomb. Astronomy and meteorology, (Tokyo) v. 20, 1954.

———. Possible atmospheric disturbances and damages to the rice-crops in northern Japan that may be caused by experimentation with nuclear weapons. Geophysical magazine (Tokyo) v. 26, 1955: 125–134.

———, and Tsutsumi, K. A decrease in the normal incidence radiation values for 1953 and 1954 and its possible cause. Geophysical magazine (Tokyo) v. 27, 1956: 205–208.

Arizumi. On the distribution of ash-fall (meteorological investigation on the H-bomb experiment at Bikini Island—II) Journal of the Meteorological Society of Japan (Tokyo), Nos. 9–10, 1954.

Egawa, Tomoji, and others. Investigations on the contamination of field crops by artificial radioactivities as a result of the H-bomb tests at Bikini Atoll. Soil and plant food, v. 1, 1955: 19–20.

Crop samples taken between June and October 1954 were analyzed for radioactivity. Rare earth elements contributed the greater part of the activity. Polished rice showed no activity.

Horie, Kuniko. Damping of radioactivity of the Bikini ashes. Kagaku (Science) (Tokyo), v. 25, 1955: 636–637.

The radioactivity (β- and γ-radiation) of the H-bomb ashes was measured over a period of 600 days by means of an electroscope and a Geiger-Muller counter. Absorption by Al foils shows that the half-life is shorter for radiation of lower energy.

Ito, Gakuro and Moriuchi, Yasuyuki. Some problems on the radiological protection: especially concerning with the maximum permissible dose. Ôyô Butsuri (Tokyo?), v. 24, January 1955: 3–17 (in Japanese).

Kakehi, H. Ash of Bikini and its effect on human body. Journal of Japan Physicians' Society, v. 31, May 1, 1954 (in Japanese).

Discusses physical and chemical composition of radioactive ashes which fell on the fishermen of the Fukuryu Maru and gives a clinical study of its effects. Estimated radiation received by fishermen in 2-week stay on ship as 200 r. Discusses hazard from contaminated tuna.

Kaneshige, Kankuro. Japan-United States radiobiological conference. Contemporary Japan (Tokyo), v. 23, Nos. 4–6, 1955: 296–310.

Kawabata, Toshihuri. Studies on the radiological contamination of fish. Japanese journal of medical science and biology (Tokyo), v. 8, October 1955: 337–372.

The decay rate of the radioactivity of the organs of fish caught by the crew of the "Shunkotsu Maru" was measured. After 3 months, the spleens, kidneys, and gonads retained considerable radioactivity, suggesting the presence of long-lived radioactive elements. At the end of this time, the liver and other organs retained only about 10 percent of the initial radioactivity and the bile had lost most of its activity. The retention by the pyloric caeca and the intestinal contents varied considerably and was probably dependent on the food consumed. Absorption of the ash of the organs on Dowex 50 and elution located most of the activity in the fractions eluted with 0.5 percent oxalic acid and 5 percent citrate buffers of pH 4.1 and 4.6. Qualitative separation with carriers identified the main radioactive element of the citrate buffer, pH 4.1, eluate as Zn^{65}.

Kimura, Kenjiro. Radioactive ashes on the fifth Fukuryu-Maru, the fishing boat that suffered from the hydrogen bomb test on March 1, 1954. Kagaku, (Tokyo) v. 24, 1954: 300–302.

By ordinary procedures with carriers and by separation with cation-exchange resins, the ashes were analyzed and the following radioactive nuclides were detected, Zr^{95} (65 days), Nb^{95} (35 days), I^{32} (2.4 hours), Te^{132} (77.7 hours), Nb^{95m} (90 hours), I^{131} (8.141 days), Ba^{140} (12.8 days), La^{140} (40.0 hours), Sr^{89} (53 days), Sb^{127} (93 hours), Ru^{103} (39.8 days), and Ru^{106} (1.0 year) etc.

———, and others. Detection of rhodium–103m in the "Bikini ashes." Bulletin of the Chemical Society of Japan, v. 29, 1956 (in English) 395–398.

The radiochemical analysis of the so-called Bikini ashes which fell on a Japanese fishing boat, the No. 5 Fukuryu Maru on March 1, 1954, are described as of some 25 days after detonation of the bomb. The collected sample (10^{-7} counts/minimum) was ignited and dissolved in 6N HC1, insolubles were filtered off, and the activity of small aliquots of the filtrate was measured. Total activity was estimated about 10^{-6} counts/minimum. Ru (10 mg.) was added to the filtrate as a carrier, the acidity of solution was adjusted to 2N, H_2S was passed through to precipitate R_u as sulfide, and the precipitate was dissolved with HNO_3; H_20, KM_n0_4, and concentrated H_2O_2. The appropriate aliquot portion of the distillate was taken up in a counting dish and evaporated to dryness; the activity was measured and found to be 1.5 x 10^{-5} counts/minimum.

Kosaka, Takao, and others. Radioactive rain and contaminated atmosphere observed in Niigata City (Japan). First report: the effect on environment and human body. Niigata Medical Association journal, v. 69, 1955: 1–6.

Koyama, Y. and others. Clinical course of the radiation sickness caused by Bikini ashes: intermediate Report. Iryo (Tokyo) v. 9, January 1955: 5–45 (in Japanese).

Clinical observations are summarized covering a 5-month period on 16 patients exposed to fallout from the Bikini explosion on March 1, 1954.

——— ———. Conference of the radioactive disease caused by the atomic bomb explosion in the central Pacific. Iryo (Tokyo) v. 9, January 1955: 56–68 (in Japanese).

Mitsui, Shingo, and others. Investigations on the radioactive contamination of crop plants as a result of hydrogen-bomb detonation. Soil and plant food, v. 1, 1956: 15–18.

I. Radioactive contamination of crop plants and soil. II. Root and foliage uptake of Bikini ash.

Miyake, Y. Artificial radioactivity in rain water observed in Japan, from autumn 1954 to spring 1955. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 6, May 1955: 26–32.

At about midnight of September 18, 1954, a typhoon (No. 14, 1954) ran away toward the sea after attacking Japan. In place of the typhoon a colder and less moist air flowed in from the north. Just at the time, a new activity of artificial origin was detected in rain water at Niigata and Hirosaki, both situated along the Japan sea coast of the northern part of the main island. From 22d to 24th of September, the activity increased rapidly, spreading over a wide area in Japan and finally an activity as strong as 0.3 x 10^6 curie/liter was counted in rain water at Yamagata.

———. The artificial radioactivity in rain water observed in Japan from May to August 1954. Papers in meteorology and geophysics, Meteorological Institute, Tokyo. v. 5, September 1954: 173–177 (in English).

Radioactivity in rainfall was measured at several places in Japan after the spring, 1954, Pacific tests. The maximum activity, 0.5 x 10^{-6} c/1, was observed at Kyoto on May 16, 1954. Meteorological trajectories indicate air that was over Bikini on May 8 reached Japan on the 16th and it is speculated that an explosion on May 5 is responsible for the activity. On August 3, airplane measurements with a dust impinger indicate 0.8 $\sim$ 2.0 x curie/cc on the average from 1000–3000 meters over Tokyo.

Miyake, Y. Rain from south and snow from north. Kayaku Asahi, (Tokyo) December 1954 (in Japanese).

Discusses detection of nuclear explosions by various methods including observations of fission product activity in the atmosphere. Deposition of 750 cpm on a vaseline coated paper (30×30 cm) on May 13–16, 1954. Eighty-six thousand cpm/1 observed in rain at Kyoto on May 14, apparently from May 5 test at Bikini. Thereafter, strong contamination of rain observed at many places on Pacific Coast of Japan. Since May 1954, activity of rain on Pacific side about an order of magnitude greater than on Japan Sea side of Japan. On September 22, 1954, a record-breaking 124,000 cpm/1 from a Russian test was observed in rain at Yamagata, associated with a cold front advancing from Siberia, almost no activity in warm front rain on the Pacific coast. Discusses possible hazard from contaminated snow.

——— and Sugiura, Y. Radiochemical analysis of radio-nuclides in sea water collected near Bikini Atoll. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 6, 1955: 33–37.

A radiochemical analysis of sea water containing fission materials collected near Bikini Atoll in June 1954, was performed. The sea water was boiled with hydrochloric acid; iron and lanthanum salts each 5 mg as Fe and La were added to it. They were precipitated as hydroxide, which was dissolved in hydrochloric acid and ferric chloride was extracted with ethyl ether. The remaining solution was evaporated to dryness and the residue was dissolved in hydrochloric acid. Using the latter solution the group separation was done with cation exchanger resins.

———, and others. Artificial radioactivity in the sea near Japan. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 6, May 1955: 90–92.

Sea water collected around the Bikini Atoll from July to September 1954, was analyzed for total radioactivity by adding 2 g. solid NH_4Cl, 1 ml. of an aqueous solution of Ferric alum (86.3 g./l.), and 1 ml. of $BaCl_2$ solution (17.8 g./l.) to 1 l. of H_2O heated to 60–70 while being stirred. NH_4OH was added until the solution was faintly pink to phenolphthalein. After 2-minutes boiling the precipitate settled on standing for several hours at room temperature before being filtered on a filter disk lain above a glass filter. Counting rates of 2.1 ± 1.6 to 140.8 ± 6.8 counts/minute/l. were obtained.

——— ———. On the distribution of radioactivity in the sea around Bikini Atoll in June 1954. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 5, January 1955: 253–263.

Report of an oceanographic survey in the late spring of 1954 in the Marshall Islands area to investigate the radioactivity of the waters following the Castle tests. Maximum value was 7025 cpm/1, 450 km west of Bikini at a depth of 75 m (1,000 cpm ≈ 5.9 mμc). Almost all radioactivity was in solution, a filter of pore size 0.5μ passed 99 percent of the activity. Distribution of the radioactivity and its relation to ocean currents is shown. Vertical cross sections show marked decrease in activity below thermocline, about 150 m. Estimate flow of radioactivity through cross-section 150 km west of Bikini was 10^5 curie/hour. Coefficient in mixed fission product decay law ranged from -1.3 to -1.6, mean -1.5.

——— ———. Radiochemical analysis of fission products contained in the soil collected at Tokyo, May 1954. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 6, 1955: 93–94.

Soil (300 g.) was leached with 50 ml. 6N HCl on a steam bath and the filtered solution evaporated to dryness. The residue was dissolved in distilled water and an aliquot of the solution was subjected to chemical analyses, in which the sample was dried on a stainless steel planchet and its β-rays were counted. Group separation of the extract was made after addition of carriers of Ce, Ba, and Sr. Precipitation with H_2S showed very weak activity which was only a few percent of the total. The hydroxide group contained an appreciable amount of radionuclides, but most of them were insoluble when changed into fluoride forms. The filtrate of fluoride solution also showed a weak activity. The radionuclides obtained in the carbonate fraction were separated into Ca, Sr, and Ba and the Ca fraction was separated by concentrated HNO_3 and the Ba fraction obtained by precipitation as chromate. Results show radionuclides of rare earths $= 9 \times 10^{-13}$ curie/g. $Sr^{89} = 3 \times 10^{-13}$ curie/g., and $Ba^{140}- = 7 \times 10^{-13}$ curie/g.

Ohashi, S., and others. Pathological findings in the fatal case (the late Mr. Kuboyama) of the radiation sickness caused by Bikini ashes. An intermediate report. Iryo (Tokyo), v. 9, January 1955: 46–55 (in Japanese).

Autopsy findings and the case history are summarized from a case diagnosed as radiation sickness caused by exposure to fallout from a thermonuclear explosion. The patient died 207 days following exposure while on a fishing boat said to be located about 100 miles east of Bikini at the time of the explosion. Evidence was also found of a secondary virus hepatitis and aspergillus fumigatus pneumonia.

Otsuka, R. and Shimada, K. On the upper air current in lower latitude of the north western Pacific ocean at the beginning of March 1954. Meteorological investigation on the H-bomb experiments at Bikini Island. Journal of the Meteorological Society of Japan, v. 32, No. 7–8, 1954.

Ota, Michio, and others. Contamination of grapes by radioactive substances. Soil and plant food (Tokyo) v. 1, 1955: 43–44.

The K content of grapes was determined by measuring K^{40} content from 1951 to 1954. After the radioactive fallout in 1954, the grapes were shown to be contaminated by radioactivity.

Obo, Fujio. Radioactive rains and fishes in the Kagoshima area. Medicine and biology (Tokyo) v. 33, 1954: 19–23 (in Japanese).

Results are given of radioactivity determinations of rains, well and city water, vegetables, domestic animals, milk, and fishes. Radioactivity was determined in a radiation counter (Scientific Research Lab. model 32), at a distance of 1 cm. for 10 minutes. Samples were obtained during 18–27 May, 1954. The highest and lowest values obtained were: 4000–80 counts/min/1/ (c. p. m.) for rains, 20–0 c. p. m./cc. for well water, and 71–0 c. p. m./100 cc. for city water.

Radioactivity in the pelagic fish. Bulletin of the Japanese Society of Scientific Fisheries (Tokyo) v. 20, 1955: 907–926.

I. Distribution of radioactivity in various tissues of fish. Bulletin of the Japanese Society of Scientific Fisheries, v. 20, 1955: 907–915.

Pelagic fishes caught after atomic explosion experiment at Bikini Atoll in the Pacific were examined by radiochemical techniques. Generally the radioactivity was large in liver, kidney, gall bladder, and heart, and then in pyloric caeca, stomach, intestine, and gonad: there was little activity in skin, bone, and muscle. This order varied with species. Large radioactivity of the stomach contents did not necessarily mean large activity in the tissues, indicating considerable participation of diffusion of sea water into the fish body. Muscles from various sites showed slight difference in the activity. The dark muscle, however, showed several times as large activity as ordinary muscle.

II. Group separation of radioactive elements in fish tissues: p. 916–920.

Analytical group separation was performed with various ashed tissues of some fishes exposed to radioactive ash. The radioactivity was particularly large with element belonging to the third group, both A and B subgroups The second group showed considerable activity in pyloric caeca and kidney of skipjacks. The radioactivity of the first and fourth groups was detected in some tissues; the fifth group showed slight activity.

III. Separation and identification of zinc 65 in the muscle of skipjack.

Muscles of skipjack caught in the vicinity of the Bikini Atoll after the explosion were ashed, treated with Dowex 50, and eluted with various solvents. A fraction obtained with 0.5 percent oxalic acid and ammonium citrate (pH 4.18) contained Zn^{65}.

Saiki, Masamichi, and others. The radioactive material in the radiologically contaminated fishes caught in the Pacific Ocean in 1954. Bulletin of the Japanese Society of Scientific Fisheries (Tokyo) v. 20, 1955: 902–906.

The radioactivity of several samples of Coryphaena Hippurus caught in the southern Pacific in May 1954, after the atomic explosion at Bikini, was found, in decreasing order, in spleen, kidney, liver, pyloric caeca, heart, gill, intestine, gastric wall, ovary, testis, gastric content, red muscle, skin, vertebrae, and muscle. The red muscle of Neothunnus Macropterus showed 54.8 counts/min./0.20 g. activity on dry basis; the activity was decreased to 27.6 by soaking 25 g. muscle in 25 cc. water, and to 14.1 by soaking in 0.5 percent Na ethylenediaminetetraacetate solution. The radioactive substances in these fish tissues were found, upon analysis, to belong to the III group, particularly to III–B group. Examination of synchroscope patterns by scintillation counter indicated the presence of Zn^{65} among the radioactive substances. Sr^{90} was suggested to be present in very small amount.

Shimizu, Kentaro. Hiroshima and Bikini. Oriental economist, (Tokyo) v. 22, July 1954: 344–346.

A report by the doctor who treated the Japanese fisherman injured by the Bikini test.

Shinjiro, T. Death ash, experience of 23 Japanese fishermen. Japan quarterly, v. 2, 1955: 37.

Tajima, Eizo. Why fishing boats were contaminated by radiation. Shizen, December 1954 (in Japanese).

Many Japanese fishing boats were examined with a G–M counter following the Bikini tests of 1954. Decks and other washable parts were weakly irradiated, lamps and other unwashable parts were strongly irradiated. Directional relationships of contaminants on individual ships coincided with those of the prevailing winds. Ships to the west of Bikini averaged 123 cpm; those to the east, 1,800 cpm. Activity to the east shows sharp rises on test dates, sharp drops on other dates. To the west of Bikini, a strip of water from 15° N. to about the equator is contaminated, and the contamination of the boats may have been due to this.

Takase, Akira. Distribution of radioactivity in various tissues of fish and group separation of radioactive elements in them. Bulletin of the Institute of Public Health (Tokyo), v. 4, 1955: 27.

Radiological studies were made of several kinds of fish which had been caught in the fishing ground including the area from longitudes 128° East to 162° East and from latitudes 3° North to 33° North during the period from April 25 to July 7, 1954, and which had been rejected as highly contaminated radiologically at the time of landing.

———. Separation of the radioactive elements in the muscle of skipjack by ion-exchange resin, and confirmation of the presence of radioactive zinc. Bulletin of the Institute of Public Health (Tokyo), v. 4, 1955: 22–26.

An ashed sample of skipjack muscle caught in June 1954, near Bikini Atoll was analyzed for elements separated by an anion-exchange method (Dowex 50) with the use of 0.2N HCl, 0.5 percent oxalic acid, and 2 percent NH_4 citrate as eluents at each pH value of 3.53, 2.18, 4.60, 5.02, 5.64, and 6.42.

Yamada, Yoshio, and others. Measurement of radioactivity in contaminated crops. Soil and plant food (Tokyo), v. 1, 1956: 25–26.

A method called the direct method was developed to correct for natural K^{40} radiation in plant samples. The K_{20} content of the ashed sample is determined by flame-photometry. The radioactivity in a 100-mg. sample is measured and the natural radioactivity from K^{40} determined by calculation subtracted. Tea samples tested gave evidence of contamination by radioactive fallout.

Yamamoto, Ryozaburo. Atmospheric oscillation caused by the H-bomb. Astronomy and meteorology (Tokyo), v. 20 No. 8, 1954.

Yamasaki, F., and Koneko, H. On the artificial radioactivity in rainwater. Journal of the Scientific Research Institute (Tokyo), v. 49, June 1955: 137–143.

Rainwater in Tokyo was examined for artificial radioactivity from April to December 1954. The most active rain occurred on May 17, as reported by Miyake. Wide variability in activity was noted from sample to sample, even in the same rainfall. In a single rainfall, the specific activity appeared to be negatively correlated with rainfall intensity. Rainwater collected in the early stage of a single rainfall did not always show the strongest radioactivity. Exponent in the decay law for mixed fission products ranged from −0.9 to −1.4 in five rain samples investigated.

Yatazawa, Michihiko, and Ishihara, Takashi. Radioactive contamination of plants in Japan covered with fallout from H-bomb detonations in March–May 1954 at Bikini Atoll, Marshall Islands. I. Distribution of deposited radioactivity. Soil and plant food (Tokyo), v. 1, 1955: 21–22.

In May 1954 rains contained radioactivity up to 0.2 muc. per liter. The provisional permissible level of unknown radioisotopes in H_2O is given as 10^{-7} muc./ml. for $\beta-$ or $\gamma-$ emitters. The safety factor for these values is at least 100. From these values the permissible level for foods was calculated as 0.22 muc./day. Food plants tested ranged 0–1.25 muc./10 g. dry matter. It is concluded that serious radioactive contamination of plants was probable.

RUSSIAN

Marei, A. N. Radioactive wastes and public health problems. Meditsinskaia radiologiia (Moscow) v. 1, July–August 1956: 3–7 (in Russian).

Yakobson, I. I. Initial radioactive investigations in Russia. Akademiya Nauk Uzbek S. S. S. R., v. 5, 1953: 118–135.

SWEDISH

Muller, Hermann J. The manner of dependence of the permissible dose of radiation on the amount of genetic damage. Acta radiologica (Stockholm), v. 41, January 1954: 5–20.

Morgan, K. Z. Maximum permissible concentration of radioisotopes in food, water and air and maximum permissible equilibrium amounts in the body. Acta radiologica v. 41, January 1954: 30–46.

Warners, C. J. Note on radioactive compounds in the atmosphere. Tellus (Stockholm) v. 7, August 1955: 403–404.

Since January 1, 1955, daily measurements of the radioactivity of the air have been made by the Royal Netherlands Meteorological Institute. The highest values noted were in the period April 28–30, in tropical air transported from the south, and reached 27.4 x 10^{-10} μc per liter.

OTHER NATIONS

Akpinar, S. and Akpinar, R. Radioactive precipitations in Istanbul and Uludag (Turkey) Istanbul, v. 20C, 1955: 287–302 (in English).

From the decay curves of fission products in rain and snow, it was possible to determine the date of announced United States atomic tests and unannounced Russian tests with a probable maximum uncertainty of ± 8 days.

Gabites, J. F. Drift of radioactive dust from the British nuclear bomb test in October 1953. New Zealand Journal of science and technology (Wellington, N. Z.) v. 36B, September 1954: 160–165.

To account for the second wave of radioactivity which reached Wellington 38–54 hours after the blast in Woomera, Australia, it is apparently necessary to assume the material started from a height of 22,000–25,000 feet. The dust was dispersed downward by eddy diffusion and carried by lower level atmospheric circulations to New Zealand. The first wave which occurred 30 hours after the blast remains unexplained.

Holubec, K. Lesson of Hiroshima and Bikini. Časopis lékařů českých (Prague) v. 95, May 11, 1956: 518–525 (in Czech).

Levi, H. Natural background radiation and radioactive fallout. Ugeskrift for laeger (Copenhagen) v. 117, December 15, 1955: 309–311 (in Danish).

Piédrola, Gil G. and Amaro, Lasheras J. Precipitation of radioactive dust (fall-out); necessity for a national detection and defense organization. Medicina colonial (Madrid) v. 28, October 1, 1956: 229–231 (in Spanish).

Rose, D. C. and Katzman, J. Radioactive deposits found at Ottawa after the atomic explosions of January and February 1951. Canadian journal of physics, v. 30, March 1952: 111–116.

Gamma-ray measurements indicated that considerable radioactive matter, apparently consisting of fission products, fell on January 29, and on February 7, 1951. The earlier fall came from an explosion in Nevada on January 27, and the February 7 material appeared to be 3 to 5 days old. The quantity was estimated to be equivalent to 1 γ Ra per square mile.

Ryder, N. V., and Watson-Munro, C. N. The detection of radioactive dust from the British nuclear bombs of October 1953. New Zealand journal of science and technology (Wellington, N. Z.), v. 36B, September 1954: 155–159.

Thirty hours after the nuclear explosion near Woomera, Australia, in October 1953, a peak in β-activity of 50 cpm was observed in Wellington, New Zealand, on an air filter. A second peak of 6 cpm was observed a few hours later. Decay curves indicated the activity was fission product activity.

Szalay, A. Unusual radioactivity observed in the atmospherical precipitation in Debrecen (Hungary) between April 22–December 31, 1952. Acta physica (Budapest, Hungary) v. 5, 1955: 1–14.

The authors investigated, by means of an end-window β-counter tube equipment the activity of precipitation fallen in Debrecen between April 22 and December 31, 1952. At times the precipitations showed radioactivity which proved to be due to fission products deriving from atomic explosions. These anomalous activities, with a lag of a few days, were in correlation to time with the atomic explosions published during the same period.

Popular Periodical Articles

A-bomb survivors due to lose quarter of life. Science news letter, v. 69, March 31, 1956: 201.

All about A-bombs, fallout, dangers in the future. U. S. news and world report, v. 38, April 29, 1955: 96ff.

Extracts from transcript of hearings held by the Joint Committee on Atomic Energy, April 15, 1955.

Amrine, Michael. Atomic clouds over America. Science digest, v. 33, June 1953: 23–30.

———. Fallout, can man survive? Progressive, v. 21, February 1957: 6–10.

The author is deeply concerned about the dangers of radioactive fallout from bomb tests, and questions the administration's apparent lack of concern.

Armagnac, A. P. How the H-bombs spread radioactivity. Popular science, v. 166, April 1955: 144–145.

———. Will bomb dust endanger your health? Popular science, v. 170, February 1957: 163–167.

Arnold, James R. Effects of the recent bomb tests on human beings. Bulletin of the atomic scientists, v. 10, November 1954: 347–348.

"The death of a Japanese fisherman on September 23 [1954] not only shocked the world but made the radioactive fallout seem the most fearful consequence of the H-bomb."

As the bomb goes off and the cloud heads east * * * science tackles radiation peril. Life, v. 38, March 21, 1955: 32–39.

Ascoli, Max. There must be an end to it (editorial). The Reporter, v. 16, May 16, 1957: 8–9.

Urges end of bomb testing.

Reprinted in extension of remarks of Charles O. Porter, Congressional record [daily edition] v. 103, May 13, 1957: A3637–A3638.

Atom outgrows its proving ground. U. S. news and world report, v. 36, March 26, 1954: 45–47.

"Thousands of square miles now can be damaged by radioactive particles, from one H-bomb, carried by the wind."

The atom: "Unpleasant debate." Newsweek, v. 48, November 26, 1956: 64–66.

Various experts in the atomic energy field comment on dangers of strontium 90.

Atomic radiation: the r's are coming. Time, v. 58, June 25, 1956: 64f.

Ban the "dirty bomb," (editorial). New Republic, v. 136, April 29, 1957: 3–4.

Proposal that the United States should seek an international agreement to limit the radioactive debris from bomb tests.

Bengelsdorf, Irving. Can the atom change the weather? Saturday review, v. 39, July 7, 1956: 31–37.

Berninger, Karl. The bomb and the weather. Contemporary issues (London) v. 6, March-April 1955: 114–116.

Maintains that atomic tests do influence weather conditions; points out meteorological phenomena to bolster this thesis.

Berrill, Norman J. A Canadian scientist asks have we gone too far with the atom tests? Maclean's magazine (Toronto) v. 68, July 9, 1955: 7–9 ff.

"Every time an atomic explosion occurs anywhere in the world more highly dangerous radioactive particles are set free. Professor Berrill says it's time our leaders told us the truth—that even if we survive we may breed a future race of morons or monsters."

———. The menace of radiation. Atlantic monthly, v. 196, October 1955: 49–54.

"Radioactive fallout from test explosions of atomic bombs has made clear to Americans that nuclear warfare would mean annihilation of large areas. Less well understood is the fact that leakage of radioactive materials resulting from careless operation of atomic powerplants and other peacetime uses of nuclear energy can be just as deadly."

Bishop, R. Can fish survive the atom? Field and stream, v. 61, June 1956: 70–71.

Blifford, Irving H. Total radioactive fallout. Science news letter, v. 69, April 28, 1956: 267.

Bomb watchers; radioactive dust in Japan. Time, v. 67, April 16, 1956: 56 f.

Bombs and the species. Economist, (London) v. 175, May 14, 1955: 557–558.

Cattle caught in fallout cancerless. Science news letter, v. 71, April 20, 1957: 248.

Danger, strontium 90. Newsweek, v. 48, Nov. 12, 1956: 88 f.

Data on atomic radiation transmitted to U. N. committee. United States Department of State bulletin, v. 35, October 29, 1956: 687.
Davis, Helen M. Hazards of smog. Science news letter, v. 67, May 7, 1955: 298–299f.
"The problem of preventing harmful radioactive fallout is like that of smoke control. Filters and precipitators can reclaim valuable wastes from industrial chimneys."
De Roos, Robert. What are we doing about our deadly atomic garbage? Collier's, v. 134, August 20, 1954: 28–34.
Espinasse, Paul G. Biology and the bomb. Nation, v. 180, June 25, 1955: 579–581.
Despite the comforting statements of AEC on the background radiation increase caused by atomic tests, this author points out that a very little upset in the "balance of nature" might have very serious ramifications.
Facts about A-bomb fall-out. U. S. news and world report, v. 38, March 25, 1955: 21–26.
"Not a word of truth in scare stories over tests."
Abridged in Reader's digest, v. 66, June 1955: 22–24.
Fallout detector developed by Navy, Army-Navy-Air Force register, v. 76, December 10, 1955: 6.
Fallout hazard to grow; strontium–90. Science news letter, v. 71, February 23, 1957: 115.
Ferreus. Courage or perdition? The 14 fundamental facts of the nuclear age. Review of politics, v. 16, October 1954: 395–411.
Each fact is discussed in the light of its relationship to future policy.
Foulks, J. G. Do H-bomb tests threaten human survival? Canadian chemical processing, v. 39, April 1955: 29–30 f.
Glass, Bentley. The hazards of atomic radiations to man, British and American reports. Bulletin of the atomic scientists, v. 12, October 1956: 312–317.
Discussion of reports issued by National Academy of Sciences and British Medical Council on effects of radiation.
H-bomb contamination. Science news letter, v. 67, February 26, 1955: 134.
H-bomb such as that exploded in Marshall Islands would contaminate 7,000 square miles with radioactivity.
Turbulence in the atmosphere could make the area threatened twice as wide as the cigar shape (up to 40 miles) described in official AEC report.
H-bombs without fallout (editorial). Bulletin of the atomic scientists, v. 12, September 1956: 234ff.
Claims that statements on minimum fallout from H-bombs confuses public thinking.
Hayasaka, I. Appeal to the western evolutionists against the hydrogen bomb by the Japanese Society for the Study of Organic Evolution. Science, v. 120, October 8, 1954: 9A.
Same: Bulletin of the atomic scientists, v. 11, January 1955: 34.
Hicks, C. B. How dangerous is fallout? Popular mechanics, v. 106, November 1956: 198–202ff.
Holmes, Robert H. Latest about after-effects of A-bomb. U. S. news and world report, v. 38, May 13, 1955: 60–68.
The director of the Atomic Bomb Casualty Commission describes the present situation of the Hiroshima survivors, the effects of the bombing upon their offspring, and the present condition of Hiroshima itself.
Holzman, B. Have atom bomb tests fouled up the weather. Look, v. 17, August 11, 1953: 32–33.
Hot stuff: big hurdle for atom power. Business week, July 23, 1955: 72–82.
Problem of radioactive wastes discussed.
How dangerous are the bomb tests? Time, v. 69, June 3, 1957: 62, f.
How radiation can affect our children. Challenge, v. 5, October 1956: 54–59.
Interview with Warren Weaver, chairman, Committee on Genetic Effects of Atomic Radiation, National Academy of Sciences.
How safe humans? Newsweek, v. 49, February 18, 1957: 71.
Humanitarian bombs; minimum widespread fallout. New republic, v. 135, July 30, 1956: 3–4.
Inglis, David R. Prospects for stopping nuclear tests. Bulletin of the atomic scientists, v. 13, January 1957: 19–23.
A fervent plea for the ending of thermonuclear tests. The author believes the chief reason for ending them is not the radioactive fallout problem as much as it is the vital need to get on with some sort of disarmament.

Jacobs, Paul. Clouds from Nevada; a special report on the AEC's weapons-testing program. The Reporter, v. 16, May 16, 1957: 10–29.
Lengthy article describing the hazards to people living near the atomic testing grounds in Nevada.

Katz, Sidney. How serious is the threat of radiation? Maclean's magazine (Toronto), v. 69, December 8, 1956: 18–19, 115–118.

Knapp, H. A. South Woodley looks at the H-bomb. Bulletin of the atomic scientists, v. 10, October 1954: 306–311.

Lang, Daniel. Fallout. New Yorker, v. 31, July 16, 1955: 31–41.

Lapp, Ralph E. Civil defense faces new peril. Bulletin of the atomic scientists, v. 10, November 1954: 349–351.
Assessment of fallout and its impact upon civil defense. Urges that the Federal Government release classified data on fallout to provide guidance to civil defense organizations.

———. Confused alert. Life, v. 38, June 27, 1955: 48–49.

———. Fallout and candor. Bulletin of the atomic scientists, v. 11, May 1955: 170f.

———. Global fallout. Bulletin of the atomic scientists. v. 11, November 1955: 339–343.
Following a review of the fallout literature, the author concludes that, although many in the Pentagon maintain that blast and heat are the real military effects of nuclear weapons, it appears that local fallout from the uranium superbomb is a potent weapon. Remote fallout from even a large scale nuclear bombing, however, does not appear to be a global hazard to humanity.

———. Humanitarian H-bomb. Bulletin of the atomic scientists, v. 12, September 1956: 261–264.
"The superbomb can be designed to be relatively clean [by giving most of the energy to fusion, which produces nonradioactive helium] or very dirty [by maximizing fission]. The former would be desirable in the test series whereas the latter would seem to fulfill the requirements of a strategic weapon.

———. Radioactive fallout. Bulletin of the atomic scientists, v. 11, February 1955: 45–51; June 1955: 206–209.

———. Radioactive fallout. New Republic, v. 132, February 14, 1955: 8–12. Discussion. *Ibid.*, v. 132, April 4, 1955: 23.

———. Strontium limits in peace and war. Bulletin of the atomic scientists, v. 12, October 1956: 287–289, ff.
Questions whether MPC figures released by AEC are in fact safe.
Reply: New republic, v. 135, October 15, 1956: 5–6.

Laurence, William L. Promise of tomorrow. Colliers, v. 136, December 23, 1955: 46, ff.

Libby, Willard F. The radioactive fallout. Civil defender, v. 1, December 1955: 16.
Speech given at Annual Meeting of National Association of State Civil Defense Directors, November 3, 1955.

———. Radioactive fallout. National safety news, November 1955: 60, ff.
Remarks prepared for delivery at the fourth annual conference of the United States Civil Defense Council, Hotel Statler, Boston, Mass., September 29, 1955.

———. Radioactive fallout, with editorial comment. Bulletin of the atomic scientists, v. 11, September 1955: 256–260.
Speech given before Alumni Reunion at the University of Chicago, June 3, 1955.

———. Radioactive fallout and radioactive strontium. Science, v. 123, April 20, 1956: 657–660.
Based on a speech given at Northwestern University, Evanston, Ill., January 19, 1956.
The radioactivity produced in magaton weapons is placed largely and immediately in the stratosphere while the smaller kiloton weapons produce clouds which do not reach into the stratosphere, and the bulk of their radioactivity is left in the troposphere. In the troposphere where rain occurs, particulate matter will be washed down in an period of days or weeks. It is easy to show that one-tenth inch of ordinary rainfall will probably remove almost completely all particulate matter except that which is so small as to be nearly of molecular dimensions.
Also in Ordnance, v. 41, July–August, 1956: 40–43, with title: Fallout from the A-bomb.
Reprinted in Congressional Record [daily edition] February 20, 1956: A1577–A1579.

Libby, Willard F. What the atom can do to you and for you. U. S. news and world report, v. 42, May 17, 1957: 64–75.

A member of the Atomic Energy Commission gives "authoritative" answers to such questions as: Should we worry over fallout from atomic tests? When will we get usable atomic power? Are new atomic weapons in sight?

———, and others. Forum on the Schweitzer declaration. Saturday review, v. 40, May 25, 1957: 8–13, 35–36.

There are four articles in this forum concerning atomic tests and the dangers of the resulting radiation. One is Dr. Libby's answer to the protest of Dr. Albert Schweitzer.

Lodge, Henry C. Data on atomic radiation. United States Department of State bulletin, v. 33, July 11, 1955: 54.

Lorentz, Pare. Fight for survival. McCalls, v. 84, January 1957: 28–29.

Dangers of radioactive poisons from bomb tests and radioactive waste.

Reprinted in Congressional Record [daily edition] January 28, 1957: A498–A500; February 7, 1957: A860–A862.

MacLeod, Iain. Nuclear explosions. Vital speeches, v. 21, April 15, 1955: 1165–1168.

House of Commons discussion on the effects of increased atmospheric radioactivity on mankind's hereditary constitution.

Mansfield, Michael J. From A-bomb to U-bomb. New leader, v. 38, May 23, 1955: 16–20.

Senator Mansfield, concerned about the dangers to life and health caused by atomic and hydrogen bomb tests, suggests the possibility of ceasing such experiments.

McWilliams, Carey. Perils unknown. Nation, v. 180, April 9, 1955: 302–306.

Severe indictment of the administration for its failure to report to the people on the dangers of atomic war, and for its failure to become concerned by the increased atmospheric radioactivity caused by repeated atomic tests.

Measured fallout; control of fallout. Time, v. 68, July 30, 1956: 61.

Miller, Harold. Nuclear weapons and genetics. Fellowship, v. 21, September 1955: 5–9.

Reprint of article originally written for British magazine Reconciliation by a medical physicist of Sheffield, England.

Miller, Robert W. Safeguarding children from radiation risks. Children, v. 3, November–December 1956: 203–207.

Muller, Hermann J. Race poisoning by radiation. Saturday review, v. 34, June 9, 1956, 9, ff.

Article on "local" and remote fallout and possible effects. Discussion of mutations produced by radiation.

Reprinted in Congressional Record [daily edition], v. 103, May 15, 1957: 6222–6225.

Next to last words. New republic, v. 136, May 6, 1957: 4.

New dangers of H-bomb. Science news letter, v. 67, March 5, 1955: 147.

Discussion of AEC's report on the Effects of High-Yield Nuclear Explosions.

Dreaded fission products come from A-bomb material which is present in only small amounts in H-bombs, if at all. Therefore, fallout problem probably less serious with H-bombs, except for their greater size.

Nishiwaki, Yasushi. Death in the rain. Nation, v. 180, August 6, 1955: 111–114.

Nordheim, L. W. Tests of nuclear weapons. Bulletin of the atomic scientists, v. 11, September 1955: 253–255, f.

Testing of atomic weapons is essential to their development. On the other hand such testing may prove dangerous because of radioactivity. The author examines the evidence on both sides and concludes that the dangers of the tests are much less than the dangers of being caught with inadequate weapons.

Now there's a warning about too much X-ray; scientists say it's far more dangerous than "Fallout." U. S. news and world report, v. 40, June 22, 1956: 60–70.

The National Academy of Sciences' report, June 13, 1956, contains estimate that the average person receives 30 times as much radiation from X-rays as from fallout.

Nuclear weapons tests; statements by scientists. Science, v. 124, November 9, 1956: 925–926.

The peril of strontium-90. Time, v. 69, May 6, 1957: 24.
Willard Libby's answer to Dr. Schweitzer on dangers of fallout.

Passin, Herbert. Japan and the H-bomb. Bulletin of the atomic scientists, v. 11, October 1955: 289–292.
This article tells how the accident in which several Japanese fishermen became victims of fallout has poisoned relations of Japan with the United States.

Poling, J. Men who really know about bomb-dust radiation! Better homes and gardens, v. 35, May 1957: 71f.

Radiation dangers. New republic, v. 136, May 20, 1957: 8–9.
"What is known about the hazards of bomb tests? What risks is it reasonable to run?"

Radiation hazards accumulate. F. A. S. newsletter, No. 57–4, April 15, 1957: 1 f.
Summary of recent news articles on nuclear testing and fallout.

Radioactive man. Economist (London) v. 180, July 21, 1956: 206.

Real power of the super-bomb; radioactive fallout. New republic, v. 131, November 8, 1954. 3–4.

Report on Hiroshima: Thousands of babies, no A-bomb effects. U. S. news and world report, v. 38, April 8, 1955: 46–48.
"From medical records of the only people who have lived through an atomic blast. Their children are normal. Radiation burns have healed. There's no radiation blindness."

Robinson, Donald. If H-bombs fall. Saturday evening post, v. 229, May 25, 1957: 25, 105–113.
"Exactly how would Americans behave under a thermonuclear attack? A special disaster-research team has come up with these surprising answers to an urgent question."

Rotblat, Joseph. The atomic challenge; radiological hazards. The new statesman and nation (London) v. 50, August 13, 1955: 177–178.
Discussion of (1) radioactivity, and its distribution resulting from fission; (2) radiation damage in a fallout area; (3) radiological hazards involved in peacetime uses of atomic energy.

Round the world tracer; tracing radioactive air masses. Time, v. 67, March 12, 1956: 73.

Rovere, R. H. Letter from Washington, perils of thermonuclear warfare. New Yorker, v. 31, February 26, 1955: 98–100.

Rs from the sky; fallout from test, March 1, 1954. Time, v. 65, June 20, 1955: 66.

Russell, Bertrand, Man's peril from the hydrogen bomb. The Listener (London) v. 52, 1954: 1135.

Safer H-bomb. U. S. news and world report, v. 41, July 27, 1956: 8.

Schweitzer, Albert. A declaration of conscience. Saturday review, v. 40, May 18, 1957: 17–20.
Complete text of Dr. Schweitzer's plea for control over the testing and military uses of atomic weapons. Stresses the radiation hazards involved.
Reprinted in Congressional Record [daily edition], v. 103, May 15, 1957: 6217–6221.

Sherrod, Robert. The grim facts of the H-bomb accident. Saturday evening post, v. 227, July 17, 1954: 20–21 ff.
"What really happened to those Japanese fishermen who wandered too close to the hydrogen blast at Bikini."

Stapleton, Bill. Navy vs. the H-bomb. Collier's, v. 134, July 23, 1954: 19–25.
"The heretofore secret story of 10 warships that were unexpectedly trapped in radioactive dust, but saved themselves with a shower of sea water."

Stevenson, Adlai E. Why I raised the H-bomb question. Look, v. 21, February 5, 1957: 23–25.
"The defeated Democratic candidate warns that H-bomb tests are still exposing all of us to a substance so lethal that just one spoonful would poison everyone on earth."

Straight, Michael. The ten-month silence. New republic, v. 132, March 7, 1955: 8–11.
Why did the administration wait 10 months after the H-bomb tests to tell the people of the lethal effects of fallout. Author criticizes AEC for its emphasis on secrecy.

Strauss, Lewis L. Truth about radioactive fallout. U. S. news and world report, v. 38, February 25, 1955: p. 35–38.

Strontium 90. (Editorial) Commonweal, v. 65, March 1, 1957: 556.

Teller, Edward. The nature of nuclear warfare. Air force, v. 40, January 1957: 43–47.

Main thesis: "If we so prepare ourselves that a terrible attack could hurt us but could not destroy us, then such an attack, I believe, will never come."

This dangerous planet. Newsweek, v. 45, January 17, 1955: 52–54.

This article is a consideration of the following question: "Is the lingering radiation spread around the earth by past or future H-bombs capable of generating [a] biological catastrophe, an insidious weakening of the human race, perhaps its extinction?"

Tunnel to nowhere. Atlantic, v. 199, April 1957: 6, f.

Article on radioactive waste disposal.

Unpleasant debate. Newsweek, v. 48, November 26, 1956: 64-66.

Describes debate among scientists on amounts of strontium 90 that can safely be absorbed by humans.

Same article, abbreviated with title, Will strontium 90 poison the world? Science Digest, v. 41, February 1957: 29–33.

Unseen cloud; Sir Anthony's Bikini. Economist (London), v. 179, June 16, 1956: 1077–1078.

Waddington, C. H. Atoms and genes. Nation, v. 183, August 18, 1956: 137–140.

Discussion of reports issued by National Academy of Sciences and British Medical Council on effects of radiation. Emphasizes dangers of fallout.

———. Peril from A-dust; with editorial comment. Nation, v. 180, February 19, 1955: 155–157.

War on the unborn. Economist (London), March 26, 1955: 1072.

Westergaard, M. Man's responsibility to his genetic heritage. Bulletin of the atomic scientists, v. 11, November 1955 : 318, f.

What the H-bomb fuss is all about. U. S. News and World Report, v. 41, October 26, 1956: 126–134.

Statements by Stevenson, Dewey, Dulles, Nixon, and Kefauver.

What will radioactivity do to our children? U. S. News and World Report, v. 38, May 13, 1955: 72–78.

Interview with Dr. H. J. Muller. Biggest danger is in the careless use of X-rays.

What you should know about danger from X-rays. U. S. News and World Report, v. 40, June 29, 1956: 44–48.

What's back of the "fallout" scare. U. S. News and World Report, v. 42, June 7, 1957: 25–28.

"Official position is that United States cannot afford to give Russia a monopoly on nuclear experiments.

Why is all the agitation directed against British and American bomb tests—and not against the tests conducted by Soviet Russia?"

White, E. B. Letter from the East. New Yorker, v. 32, November 3, 1956: 198–202 ff.

Wilson, E. Raymond. Japan's atomic fears. Christian century, v. 74, May 1, 1957: 553–554.

This article tells of Japan's efforts to be the "conscience of the world" regarding atomic and hydrogen bomb tests. The author feels that Japan's efforts toward leadership in abandoning such tests and in disarmament are not as effective as they might be.

Newspaper Articles

Atomic peril cut in fallout data. New York Times, October 13, 1956: 3.

Report of Dr. Libby's speech on radiation at dedication of new building of American Association for Advancement of Science.

Baldwin, Hanson W. A military atom problem. New York Times, June 14, 1956: 14.

An analysis of the National Academy of Sciences report and the possible effect of its findings on defense policies.

Cahan, William G. Effects of radioactivity. New York Times, October 31, 1956: 32.

A physician writes a letter to New York Times on possible role of radioactivity in inducing cancer.

Cowen, Robert C. Atomic radiation: new challenge for humanity? Christian Science Monitor, August 3, 1956: 9.

Review of reports issued by National Academy of Sciences and British Medical Council.

Cutler, Robert. Reply to Finletter on the bomb. New York Herald-Tribune, October 12, 1956: 1, f.

Danger level of strontium. New York Times, October 28, 1956: 10E.
Letter to the editor from eleven Professors of Chemistry, Physics and Biology.
"The question of what is to be considered as the maximum permissible concentration (M. P. C.) of radioactive strontium in human bone is one which probably will not receive a definite answer for many years. It is the answer to this question that is the basis of most of the disagreements that are not of political origin. * * *"

Engle, William. Radiation—your friend or foe? The American Weekly, January 6, 1957: 4–6.

Fallout of a Soviet bomb. The Times (London) April 22, 1957: 6.
Recent Soviet nuclear explosion was of a kind calculated to release the maximum quantity of radioactive fission products into the atmosphere, observes Professor Shiokawa, of Shizuoka University, Japan.

Fallout tests at Harwell. The Times (London) May 30, 1956: 7.
Report on measurements of radioactivity caused by thermonuclear explosions.

Fallout: the peril point? Washington Sunday Star, June 2, 1957: A23.

Gallup, George. Public favors H-tests' halt, if—Washington Post, May 19, 1957: E5.

Germans cite gain in hydrogen fusion. New York Times, March 3, 1957: 22.
Team of German nuclear physicists working in Hamburg report solution of problem of hydrogen fusion without an atomic bomb trigger, a method which does not produce radioactive fallout.

Harsch, Joseph C. Dangers of fallout. Christian Science Monitor, April 25, 1957: 1.

Henry, Thomas R. Does radiation exposure hasten aging? Washington Star, September 3, 1956: A8. (Vistas in Science).

Henry, Thomas R. Scientists warn world of genetic atom peril. Washington Star, April 23, 1957: A5.
Account of symposium on genetics at 94th annual meeting of the National Academy of Sciences.

Increased radioactive fallout on Britain. Manchester Guardian Weekly, May 31, 1956: 3.
Report of Manchester Guardian's scientific correspondent of a tour of Britain's Atomic Energy Authority's Research Establishment at Harwell. Mentions methods used to measure fallout.

Is fallout good? (editorial). Washington Post, April 27, 1957: A12.
Questions Dr. Libby's statements on relative harmlessness of fallout.

Kaempffert, Waldemar. How fallout from atomic explosions could be controlled is an unanswered question. New York Times, July 29, 1956: E9. (Science in review.)

Krock, Arthur. H-bomb issue raises problem for voters. New York Times, October 21, 1956: E3.

Lapp, Ralph E. Will radiopoison accompany "Age of Megaton?" Washington Post, July 1, 1956: E1, f.
Discusses effects of remote fallout of invisible radioactive particles carried high into stratosphere by force of a bomb explosion. Urges limitation of nuclear tests.

Laurence, William L. Facts on the effects of atomic fallout are being presented for public decision. New York times, June 2, 1957: E11. (Science in review.)

———. Schweitzer versus atomic authorities on the dangers of weapons testing, New York times, April 28, 1957: E11 (Science in review.)

Navy expanding fallout studies. New York times, December 14, 1956: 19.
Network of 10 to 12 monitoring stations to study distribution of radioactivity in the air to be set up from Greenland to the South Pole by Naval Research Laboratory, during International Geophysical Year.

The nuclear test issue (symposium). Washington Sunday Star, May 5, 1957: A35.
Springarn, Jerome. The world gropes for a control plan.
Amrine, Michael. Strontium is center of debate on tests.
Libby, Willard F. Actual risk is small.
Fowler, John M. and Goldberg, Norman. The cautious side.
Fryklund, Richard. European report: concern but no position.

Plumb, Robert K. Geneticists ponder effects of atomic bomb explosions on future generations. New York Times, January 16, 1955: E11. (Science in Review.)

"Dr. Alfred H. Sturtevant of the California Institute of Technology said his calculations indicate that 1,800 of the 90 million children born in the world in 1954 were adversely affected by radiation from bomb tests."

Selove, Walter. No last word yet on nuclear fallout hazard. Washington Post, May 5, 1957: E1, f.

The specter of strontium-90-fallout. The American Weekly, January 6, 1957: 8.

Attempts to reassure public that strontium-90 released from bombs exploded to date is not harmful.

Texts of Japanese and U. S. notes on tests. New York Times, May 14, 1957: 18.

Texts of a note April 29 from Japan on atomic tests and the United States' note of reply.

Thompson, Dorothy. Contradictions in H-bomb policy; fallout peril minimized to calm public and maximized in tests to alert it. Washington Star, July 25, 1956: A19.

Tiltman, Hessell. Japan feels intensely on H-tests. Washington Post, February 24, 1957: E3.

"Popular fears of radioactive contamination have passed the bounds of scientific evidence and become an emotional issue."

Two scientists back tests of H-bombs, New York Times, November 6, 1956: 45.

Dr. Edward Teller and Dr. Ernest Lawrence declared "The radioactivity produced by the testing program is insignificant."

U. N. gets three plans to curb atom tests. Washington Post, January 22, 1957: A11.

Three proposals laid before U. N. General Assembly's 80-nation Political Committee a few hours after AEC reported that the Russians had set off at least their fifth nuclear explosion since August.

1. Japan, Canada and Norway: Called for advance registration of all nuclear tests and for observation of the radiation results by U. N. experts.

2. Sweden: Proposed that all nuclear explosions be banned until the U. N. Scientific Committee on the Effects of Radiation had completed studies now in progress. This would amount to a 2-year moratorium.

3. Philippines: Proposed that Western powers and Russia agree on a common testing ground as the only place where nuclear explosions could take place. Tests would be registered and each country limited as to number and size of explosions permitted in any 1 year.

Ubell, Earl. Fallout danger seen only if H-war comes. New York Herald-Tribune, October 17, 1956: 1f.

———. Small X-ray dose kills human cell. New York Herald-Tribune, January 10, 1957: sec. II, p. 1.

Dr. Theodre Puck, biophysics professor of the University of Colorado, reported to a conference on cell biology that human cells could be killed with an X-ray dose of 96 roentgens. Previously scientists had believed human cells might withstand 300 to 200,000 roentgens. This new finding indicated that radiation from X-ray machines and atomic bomb fallout may be more dangerous than supposed.

Unna, Warren. Libby says test fallout won't be peril in any foreseeable circumstances. Washington Post, April 27, 1957: 1, f.

Young, Wayland. Ourselves and the bomb. Manchester Guardian, v. 76, March 28, 1957: 3.

Arguments against Britain's testing of an H-bomb at Christmas Island.

PAMPHLETS

Birkett, Henry S. Humanity versus the hydrogen bomb. London, Independent Labour Party, [195–], 31 pp.

Eisenbud, Merril. AEC fallout monitoring network. New York, American Institute of Chemical Engineers, 1955 (preprint 192). 14 pp.

News Chronicle, London. In the shadow of the H-bomb; a reprint of the News Chronicle articles of March 1–11 [1955], with additional material [by] B. H. Liddell Hart [and others]. London, News Chronicle Book Department, 1955. 23 pp.

Nuclear tests, a universal threat. New York, Contemporary Press, 1955, 4 pp.

Pace, F. C. Radioactive fallout from atomic weapons. Toronto, Ontario, Canadian Institute of International Affairs, November 1956. 12 pp. (Behind the Headlines, v. 16, No. 5.)

Summary of some of the more important reports and articles of the past 2 years.

Radiation and mankind's future. The American forum, v. 19, June 17, 1956, 11 pp.

Participants in radio discussion included Dr. Bentley Glass, Congressman Chet Holifield and Dr. Willard F. Libby. Nuclear and thermonuclear tests, and fallout: pp. 6–10.

Radiation hazards: atoms for peace and weapons tests. New York, International review service, 1956. 21 pp.

Strauss, Lewis L. The effects of high-yield nuclear explosions. Washington, United States Government Printing Office, February 1955, 19 pp.

———. Facts about radiation as they relate specifically to the atomic tests conducted in Nevada. Washington, United States Government Printing Office, April 15, 1955. 12 pp.

Statement before the Joint Committee on Atomic Energy.

Teichmann, Horst. Weather influence of atomic explosions as a disturbance to the periodic flow in the stratosphere. Naturwissenschaften (Berlin) 1954. 1 p.

Weaver, Warren. Radiations and the genetic threat. [Philadelphia] Franklin Institute, 1957: 283–294.

Reprinted from Journal of the Franklin Institute, v. 263, April 1957.

1956 Philip C. Staples Memorial Lecture, delivered at the stated meeting of the Franklin Institute, Wednesday, November 21, 1956.

Wexler, Harry. Nuclear explosions and weather. Washington, D. C., United States Weather Bureau, 1955, 9 pp.

Statement for the Joint Congressional Committee on Atomic Energy.

Speeches and Other Unpublished Material

AEC issues effective regulation on standards for protection against radiation, Washington, United States Atomic Energy Commission, January 25, 1957. 4 pp.

Code of Federal Regulations, Part 20 "Standards for Protection against Radiation" (10 C. F. R.) attached.

Atomic Scientists Association, Great Britain. Statement on strontium hazards. [London, England, 1957], 7 pp. Mimeographed.

"Calculations * * * show that an H-bomb of the type tested at Bikini in 1954, if exploded high in the atmosphere, may eventually produce bone cancers in 1,000 people for every million tons of TNT of equivalent explosive power * * *. These thousand casualties would be spread all over the world and occur in the course of several decades."

Bugher, John C. Radiation and human health. [New York?, 1956], 13 pp. Mimeographed.

Talk presented before the Forum Session on "World Health Goals" at the Fourth Annual meeting of the National Citizens Committee for the World Health Organization, November 14, 1956 at the Ambassador Hotel, Atlantic City, N. J.

Bugher, John C. Tomorrow's atom and you. Washington, United States Atomic Energy Commission, 1955, 8 pp. Mimeographed.

Speech for delivery to National Health Council, New York City, March 23, 1955.

Dunning, Gordon. Immediate radiations from a nuclear detonation. Washington, United States Atomic Energy Commission, November 15, 1956, 15 pp. Mimeographed.

Remarks prepared for delivery before the Washington Academy of Sciences, Washington, D. C., November 15, 1956.

Eisenbud, Merril. Global distribution of radioactivity from nuclear detonations, with special reference to strontium–90. Washington, United States Atomic Energy Commission, November 15, 1956, 19 pp. Mimeographed.

Remarks prepared for delivery before the Washington Academy of Sciences, Washington, D. C., November 15, 1956.

Eisenhower, Dwight D. Statement [on nuclear weapons]. Washington, D. C., October 24, 1956, 9 pp. Mimeographed.

Reprinted in New York Times, October 24, 1956: 18, f.; also U. S. News and World Report, v. 41, November 2, 1956: 143–151; Department of State bulletin, v. 35, November 5, 1956: 704–709.

Health and safety procedures established for 1957 series of nuclear tests in Nevada. Washington, United States Atomic Energy Commission, May 13, 1957, 11 pp. Mimeographed.

Libby, Willard F. The Atomic Energy Commission and national security. Washington, United States Atomic Energy Commission, 1954. Mimeographed.

Speech given before the Washington Conference of Mayors, December 2, 1954.

———. Isotopes in meteorology. Washington, United States Atomic Energy Commission, March 20, 1957, 30 pp.

Describes Project Sunshine for measurement of mixing of particulate matter in stratosphere and troposphere by use of bomb fallout products, particularly Sr^{90} and Cs137. Mimeographed.

Remarks prepared for delivery before the American Meteorological Society, Chicago, Ill., March 20, 1957.

———. Letter dated April 25, 1957, to Dr. Albert Schweitzer, Lambarene Hospital, Lamberon, Gabon, French Equatorial Africa.

———. The radioactive fallout. Washington, United States Atomic Energy Commission, 1955, 8 pp.

Speech given at Annual Meeting of National Association of State Civil Defense Directors, November 3, 1955.

———. Radioactive fallout. Washington, United States Atomic Energy Commission, 1957, 24 pp., charts. Mimeographed.

Speech given before the spring meeting of the American Physical Society, Washington, D. C., April 26, 1957. To be published in the proceedings of the National Academy of Sciences within a few months.

———. Radioactive fallout from nuclear tests. Washington, United States Atomic Energy Commission, 1957, 8 pp. (Press release.) Mimeographed.

Remarks prepared for delivery before the University of New Hampshire Distinguished Lecture Series, Durham, N. H., April 11, 1957.

Lieberman, Joseph A. Engineering aspects of the disposal of radioactive wastes from the peacetime applications of nuclear technology. Washington, United States Atomic Energy Commission, November 16, 1956, 14 pp.

Remarks prepared for delivery before the Engineering and Sanitation, Laboratory, and Occupational Health Sections, American Public Health Association, Friday, November 16, 1956, Atlantic City, N. J.

Manly, Charles G. Radiation hazards in the atomic energy program. Washington, United States Atomic Energy Commission, May 14, 1956, 9 pp. Mimeographed.

Remarks prepared for presentation before Eastern New York Chapter, American Society of Safety Engineers, Latham, N. Y., Monday, May 14, 1956.

Murray, Thomas E. Morality and security—the forgotten equation. Washington, United States Atomic Energy Commission, November 10, 1956, 11 pp.

"No tests should be held of weapons whose magnitude would exceed the upper limit which we must set to the size of our nuclear weapons. Second, we should accelerate the testing of a wide range of weapons in the lower order of nuclear force." Mimeographed.

Remarks prepared for delivery before the Catholic Association for International Peace, Trinity College, Washington, D. C., November 10, 1956.

Murray, Thomas E. Some things the world should understand about H bombs. Washington, United States Atomic Energy Commission, November 17, 1955, 11 pp. Mimeographed.

Remarks prepared for delivery at the Golden Jubilee dinner of Fordham Law School, Waldorf-Astoria Hotel, New York, November 17, 1955.

Newell, John F. Handling and disposal of radioactive wastes. Washington, United States Atomic Energy Commission, April 25, 1957, 12 pp. Mimeographed.

Remarks prepared for presentation before the Annual Conference of the American Industrial Hygiene Association, St. Louis, Mo., April 25, 1957.

Satterfield, W. J. Some aspects of radiation hazards. Washington, United States Atomic Energy Commission, October 25, 1956, 12 pp. Mimeographed.

Remarks prepared for presentation before New York Chapter, American Society of Insurance Management, October 25, 1956.

Shilling, C. W. Everybody's business. Remarks prepared by C. W. Shilling, M. D., Deputy Director, Division of Biology and Medicine, United States Atomic Energy Commission, for presentation at Hunter College, February 20, 1957, at the Symposium on Radiation Hazards to Mankind, sponsored by Hunter College, Graduate Division, and Sloan-Kettering Institute for Cancer Research. Washington, United States Atomic Energy Commission, 1957, 21 pp. Mimeographed.

States that we must continue to live with radioactive fallout, for we must continue testing of nuclear weapons. Must gamble with the probability of radiation damage to a few individuals in order to secure survival of the total society.

Strauss, Lewis L. Statement [on fallout, after Marshall Islands tests] Washington, United States Atomic Energy Commission, July 19, 1956, 1 p. Mimeographed.

"We are convinced that mass hazard from fallout is not a necessary complement to the use of large nuclear weapons."

Sturtevant, A. H. The genetic effects of high-energy irradiation of human populations. Pasadena, Calif., California Institute of Technology, 1955, 12 pp.

Address given January 11, 1955, California Institute of Technology.

United States Library of Congress. Legislative Reference Service. Eisenhower statements on atomic weapons, atomic war and fallout in 1956. Washington, May 7, 1957, 12 pp. Typewritten.

——— ———. Hydrogen bomb tests: the arguments for and against; by Margaret E. Urist. Washington, D. C., May 31, 1956, 18 pp. Typewritten.

Warren, Shields. [Telegram to Lewis L. Strauss to correct Adlai Stevenson's statement of October 15, 1956, regarding dangers of strontium 90 from bomb testing] Washington, United States Atomic Energy Commission, October 17, 1956, 2 pp.

"Dr. Warren is scientific director of the Cancer Research Institute of New England Deaconess Hospital * * * one of the Nation's outstanding authorities on medical radiology, biology and pathology."

"From genetic standpoint, radioactive fallout including strontium 90 has given only small dose to date as compared with dose from naturally occurring and hence unavoidable background radiation. If weapons testing continues at present rate for 30 years, genetic dose still insignificant and only fraction of background."

Technical Reports

Agricultural and biological investigations pertaining to contamination by fission products (miscellaneous papers). Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, 1954, 22 pp. (Foreign Weapons Effects Report 10).

Material received from the United Kingdom in connection with the Tripartite Conference in Washington, February 15 to 19, 1954, on the effects of Atomic Explosions on Human Beings and Their Environment, under area 2 of the Technical Cooperation Program. Complete transcripts of the proceedings of the 5-day conference are on file in the Classified Technical Library, AEC, and in the Armed Forces Special Weapons Project Headquarters, Washington, D. C.

Bell, Carlos G. Sanitary engineering aspects of long-range fallout from nuclear detonations. (Thesis) Oak Ridge, Tenn., United States Atomic Energy Commission, 1955, 248 pp. (NYO-4654).

Available from the Office of Technical Services, Department of Commerce.

Bradshaw, R. L. and Cottrell, W. D. Atomic weapons test fallout at ORNL on March 19, 1953. Oak Ridge, Tenn., Oak Ridge National Laboratory, 1953, 5 pp., photos.

Brookhaven National Laboratory, Upton, N. Y. A study of the airborne radioactivity at Brookhaven National Laboratory from the Nevada Tests, March through June 1953. Upton, N. Y., November 1953 (BNL 252 (T39)).

Routine and special observations were made at Brookhaven on the radioactivity in settled dust and rainwater during the Nevada tests in the spring of 1953, together with some meteorological interpretation. It was estimated that 30.2 curies of activity were deposited on the 25.9×10^6 square mile Brookhaven site during the 4-month period. Results of various monitoring devices are given. Rain samples were investigated for solubility of fission products. Using Whatman-41 filter (efficient down to 0.7μ) 27.5 percent of

activity in residue, 72.5 percent in filtrate. Using Whatman-41 and HA molecular filter (efficient down to 0.2μ) 83.4 percent of activity in residue, 16.6 percent in filtrate, indicating significant portion of activity on particles between 0.2 and 0.7 μ. Other studies on solubility of fallout and on variation in rainout during course of storm, also gamma energy spectrum of rainout.

——— ———. Summary and observations on radioactive fallout detected in the precipitation and dust during November and December 1952. January 16, 1953 (unpublished).

An increase in radioactivity was noted in routine Brookhaven surveys in the second week of November 1952. Radioautographs showed little evidence of specific centers of activity but showed slight clouding of the film after 4 days' exposure, indicating that most of the particulate activity was from very small particles. Total activity estimated to have fallen on the 25.9×10^{10} cm^2 laboratory site during the 2-month period, November–December 1952, was:

	Curies
Settled dust, solid residue	.04
Settled dust, filtrate	.14
Precipitation, solid residue	.22
Precipitation, filtrate	.05
Total	.97

——— ———. Twelve-month postexposure survey on Marshallese exposed to fallout radiation, by E. P. Cronkite and others. Upton, N. Y., August 1955, 15 pp. (BNL-384).

Available from Office of Technical Services, United States Department of Commerce.

——— ———. United States Naval Medical Research Institute, Bethesda, Md. Medical survey of Marshallese 2 years after exposure to fallout radiation, by R. A. Conard and others. Upton, N. Y., March 1956, 18 pp. (BNL-412).

Available from Office of Technical Services, United States Department of Commerce.

The medical survey of the Rongelap people 2 years after exposure to fallout radiation shows that the people appear to have been in a generally good state of health and nutrition.

Bugher, John C. The medical effects of atomic blasts. Washington, United States Government Printing Office, 1954, 14 pp.

Speech delivered at the 7th Annual Industrial Health Conference in Houston, Texas, September 23, 1954.

California. University. Radiation Laboratory. California cattle thyroid activity associated with fallout, 1955, by Margaret R. White and Ernest L. Dobson. Berkeley, Calif., 1956, 14 pp.

Evidence is presented which appears to indicate that the radioactivity was taken into the body of cattle through food.

——— ———. Confirmation of radioactivity in thyroids of various animals, July 15 to September 10, 1954, by Ralph L. Gunther and Hardin B. Jones. Berkeley, Calif., September 1954, 12 pp.

Addendum, September 1954, 5 pp.

——— ———. A summary and evaluation of the problem with reference to humans on radioactive fallout from nuclear detonations, by Hardin B. Jones. Berkeley, Calif., January 14, 1957, 38 pp. (UCRL-3644).

California. University. University at Los Angeles. Atomic Energy Project. Recommendations for civil defense relative to radiological safety, by Andrew H. Dowdy. Los Angeles, Calif., February 15, 1951, 57 pp. (UCLA-113).

A factual, simple explanation of the atomic bomb phenonomena, specifically relating to radiation hazards under various conditions of detonation.

Available from Office of Technical Services, United States Department of Commerce.

——— ———. Soil-plant interrelationships with respect to the uptake of fission products. I. The uptake of Sr 90, Cs 137, Ru 106, Ce 144, and Y 91, by James W. Neel and others. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, March 9, 1953, 44 pp. (UCLA–247).

Available from Office of Technical Services, United States Department of Commerce.

Chicago University. Enrico Fermi Institute for Nuclear Studies. The Chicago sunshine method; absolute assay of strontium-90 in biological materials, soils, waters, and air filters, by E. A. Martell, Chicago, May 1956, 65 pp. (AECU–3262).

Available from Office of Technical Services, United States Department of Commerce.

——— ———. Strontium-90 concentration data for biological materials, soils, waters, and air filters. Project sunshine bulletin No. 12, by E. A. Martell. Oak Ridge, Tenn., Technical Information Service Extension, August 1, 1956, 67 pp. (AECU–3297 Rev.).

Results obtained since December 1, 1955 are presented for strontium-90 analyses of samples of biological materials. Data are tabulated on fresh milk and cheese, soil samples, rain, snow and surface waters, and air filters collected from various parts of the world.

Cronkite, Eugene P., and others eds. Some effects of ionizing radiation on human beings. Washington, United States Atomic Energy Commission, 1956, 106 pp. (TID–5358).

A report on the Marshallese and Americans accidentally exposed to radiation from fallout and a discussion of radiation injury in the human being.

From the Naval Medical Research Institute; U. S. Naval Radiological Defense Laboratory, San Francisco; and Medical Department, Brookhaven National Laboratory, Upton, N. Y.

Dale, G. C. Safety levels for contamination from fallout from atomic weapons trials. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, 1957, 21 pp. (Foreign Weapons Effects Report 107).

Originally issued by United Kingdom Atomic Energy Authority Atomic Weapons Research Establishment as AWRE Report No. O–41/55.

Flocker, William J. The absorption of radioactive strontium by certain crop plants as influenced by the chemical properties of some Arizona soils. Ann Arbor, University Microfilms, 1955. Bibliography: p. [94]–98. (Microfilm AC–1 No. 12,288.)

Hanford Atomic Productions Operation, Richland, Wash. The absorption by plants of beta-emitting fission products from the Bravo soil, by A. A. Selders [and others]. Richland, Wash., December 20, 1955, 10 pp.

Barley and bean plants were grown to maturity in soil from a Pacific island which contained fallout material from a thermonuclear explosion.

———, ———. A comparative study of Hanford and Utah range sheep, by L. K. Bustad [and others] Richland, Wash., November 30, 1953, 44 pp. (Contract W–31–109–Eng–52).

Observations made on sheep representing flocks in Utah adjacent to Nevada Proving Ground compared with findings made on experimental sheep exposed to various amounts of radioiodine. The Utah sheep showed no evidence of the radiation damage observed in experimentally treated sheep. Estimations on amounts of radioiodine on vegetation required to cause serious damage following a contamination event are included.

Harley, John H., ed. Operation Troll; joint preliminary report, United States Atomic Energy Commission [and] Office of Naval Research. New York, United States Atomic Energy Commission, New York Operations Office, 1956, 41 pp. (NYO-4656).

"Operation Troll was a joint undertaking of the AEC and ONR to evaluate residual radioactivity in the Pacific Ocean a year after the Pacific nuclear tests held in the spring of 1954 * * *. Widespread low-level activity was found in sea water, plankton and fish samples, but none of the levels was high enough to cause concern as a possible hazard."

———. and others. Summary of analytical results from the HASL strontium program to June 1956. New York, United States Atomic Energy Commission, New York Operations Office, 1956, 38 pp. (NYO–4751).

Hawaii. University. Hawaii Marine Laboratory, Honolulu, Hawaii. Radioisotope uptake in marine organisms with special reference to the passage of such isotopes as are liberated from atomic weapons through food chains leading to organisms utilized as food by man. Annual report, 1954–55. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, September 1, 1955, 47 pp. (AECU–3079).

Available from Office of Technical Services, United States Department of Commerce.

Hicks, E. P. and Penney, W. G. The base surge: the mechanism of fallout. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, 1954, 24 pp. (Foreign Weapons Effects Report 19).

Originally issued by United Kingdom Department of Atomic Energy. Atomic Weapons Research Establishment as Paper No. 14 of Symposium, The Physical Effects of Atomic Weapons.

Johns Hopkins University. Operations Research Office, Chevy Chase, Md. Some civil defense problems in the Nation's capital following widespread nuclear attack, Chevy Chase, Md., November 1956, 43 pp. (ORO–SP–1).

Knolls Atomic Power Laboratory, Schenectady, N. Y. Determination of radioactive fallout, by J. J. Fitzgerald. Schenectady, N. Y., 1956, 25 pp. (KAPL–1439).

Available from Office of Technical Services, United States Department of Commerce.

——— ———. Evaluation of the effects of atomic bomb detonations in the State of Nevada on airborne contamination at Knolls Atomic Power Laboratory, Knolls site and environs, by L. J. Cherubin. Schenectady, N. Y., May 7, 1951, 15 pp. (KAPL–559).

——— ———. Method for evaluating radiation hazards from a nuclear incident; by J. J. Fitzgerald, and others. Schenectady, N. Y., 1954, 53 pp. (KAPL–1045).

Available from Office of Technical Services, United States Department of Commerce.

——— ———. Quarterly report of KAPL environmental monitoring radiological services sub-unit, April, May, June, 1953. Schenectady, N. Y., 1953. (KAPL–1008).

High fission-product activity was found following heavy rain on April 26, 1953. Measurements at ground level, 3–50 mrep/hr. Fission product contamination on vegetation ranged to $2.2\times10^{-2}\mu c/g$, Radioiodine $4\times10^{-5}\mu c/g$. Soil contamination reached $9\times10^{-4}\mu c/g$. Fission product activity in the rain ranged to $4.3\times10^{-4}\mu c/cc$. On June 7, rainfall contamination of $1.9\times10_{-}{}^{5}\mu c/cc$. was observed.

Martin, Dudley C. The absorption and translocation of radiostrontium by the leaves, fruits and roots of certain vegetable plants. East Lansing, Mich., Michigan State College of Agriculture and Applied Science, 1954, 133 pp.

Thesis for the degree of Ph. D.

Maryland University. Bureau of Business and Economic Research. Baltimore and the H-bomb. College Park, 1955, 16 pp.

Massachusetts Institute of Technology. Engineering Practice School, Oak Ridge, Tenn. Permanent methods of radioactive waste disposal: an economic evaluation, by A. C. Herrington [and others]. Oak Ridge, Tenn., March 11, 1953, 50 pp. (K–1005).

Nine proposed methods for permanent disposal of radioactive waste materials are discussed and evaluated. Bibliography of 82 references.

———. Sedgwick Laboratories of Sanitary Science. The removal of radioactive fallout from water by municipal and industrial water treatment plants, by Robert A. Lauderdale and Rolf Eliassen. Cambridge, Mass., March 1, 1956, 79 pp. (contract AT (30–1)–621).

Menzel, R. G. and Brown, I. C. Removal of radioactivity from soil by leaching Washington, D. C., United States Department of Agriculture, 1953, 4 pp.

Michigan. State College of Agriculture and Applied Science, East Lansing, Mich. The absorption and distribution of radiostrontium (Sr 89) and radioruthenium (Ru 103) in certain vegetable crops (thesis), by Charles Glenn Johns. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, September 1955, 33 pp. (AECU–3101).

Available from Office of Technical Services, United States Department of Commerce.

——— ———. Growth and accumulation of radioactivity in plants grown on "fission fallout" contaminated soil, by William G. Long [and others]. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, June 9, 1955, 7 pp. (AECU–3039).

Available from Office of Technical Services, United States Department of Commerce.

National Academy of Sciences. The biological effects of atomic radiation; gonadal dose from the medical use of X-rays, by J. S. Laughlin and I. Pullman, Washington, D. C., March 1957, 105 pp.

National Research Council. The biological effects of atomic radiation. A report to the public. Washington, D. C., 1956, 40 pp.

———. The biological effects of atomic radiation. Summary reports. Washington, D. C., 1956, 108 pp.

———. Pathological effects of atomic radiation. Washington, D. C., 1956, various paging. (Publication No. 452.)

Neel, J. V. and Schull, W. J. The effect of exposure to the atomic bombs on pregnancy termination in Hiroshima and Nagasaki. Washington, National Academy of Sciences, 1956, 241 pp. (National Academy of Sciences Publication No. 461.)

New York Committee on Atomic Information, Inc. Biological, medical, and health effects and implications of atomic energy in New York City, [April 26, 1949]; eight exploratory meetings held at the Academy of Medicine, New York City, by M. S. Levine and L. C. Longarzo. New York, June 23, 1950, 34 pp. (AEC File No. NP–1427).

Review and appraisal of the use of radioactive isotopes in biological, medical and health areas. Bibliography of open literature and AEC reports included.

Oak Ridge National Laboratory, Oak Ridge, Tenn. External and internal exposure to ionizing radiation and maximum permissible concentration (MPC) of radioactive contamination in air and water following an atomic explosion, by K. Z. Morgan and C. P. Straub. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, April 4, 1952, 17 pp. (AECU–2332).

If an atomic bomb is exploded at sufficient elevation to take full advantage of blast and thermal effects, there ordinarily will not be an appreciable amount of surface radioactive contamination.

——— ———. Symposium on effects of radiation and other deleterious agents on embryonic development, given at research conference for biology and medicine of the Atomic Energy Commission, sponsored by the Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee, April 20, 21, 1953. [Philadelphia, Wistar Institute of Anatomy and Biology, 1954], 337 pp.

Ohio State University, Columbus, Ohio. Office of Radiation Safety. Health physics conference; June 13, 14 and 15, 1955. Columbus, Ohio, 1955, 224 pp.

Princeton University, Princeton, N. J. The nature of atmospheric dust: radioactive and electron measurements on fallout on Princeton, New Jersey, October 21, 1954, to August 10, 1955, by Clarence Heininger and John Turkevich. Princeton, N. J., 1955, 24 pp.

Rainey, C. T., and others. Study of the dispersal of radioactive aerosols over California. Sacramento, Calif., Office of Civil Defense, 1951.

Air samplers were set up in 19 localities throughout the state during the period of the Nevada tests. Cooperation in operating the samplers was secured from local Health Departments, and stations of the United States Weather Bureau, the Civil Aeronautics Administration, and the Plant Quarantine Service. The samplers were operated continuously from October 6 to December 5, 1951, except for a short period between the two test series in November.

The Rand Corporation. A catalog of fallout patterns, by S. M. Greenfield [and others] Santa Monica, Calif., 1956, charts.

Consists of 26 carefully computed fallout patterns with instructions for matching them approximately to a given yield and wind condition.

———. Effects of environment in reducing dose rates produced by radioactive fallout from nuclear explosions, by J. E. Hill. Santa Monica, Calif., 1954, 10 pp. (Research Memorandum 1285–1).

"From the standpoint of civilian defense against very heavy fallout contamination it would be important to select such safe locations [underground areas] prior to an attack and educate the public to their utilization, in case of a fallout emergency situation."

———. A mathematical model of the phenomenon of radioactive fallout; by R. R. Rapp, Santa Monica, Calif., June 15, 1956, 14 pp. (P–882–AEC).

———. Worldwide effects of atomic weapons. Project Sunshine. Santa Monica, Calif., c 1956, 96 pp. (R–251–AEC).

"This preliminary report discusses the various aspects of long-range contamination due to the detonation of large numbers of nuclear devices. An improved methodology for assessing the human hazard is developed, and an extensive experimental program is proposed.

. . . Report represents the 1953 estimate of the fallout problem."

Ratner, Benjamin. Winds and fallout: a climatological appraisal. Washington, United States Department of Commerce, Weather Bureau, June 1955, 19 pp.

Rensselaer Polytechnic Institute, Troy, N. Y. The fate of fission products deposited in the reservoirs of the Troy, New York, area following nuclear detonations during the spring tests (at Yucca Flats, Nevada) of 1953, by E. J. Kilcawley [and others], Troy, N. Y., December 16, 1953, 152 pp. (Contract AT (30–1) 1556).

Rochester, N. Y. University. Atomic Energy Project. Hemolytic effect of radiation, by R. Davis [and others], Rochester, N. Y., November 15, 1949, 19 pp. (UR–99).

Observations on the effect of total body radiation of dogs in single doses of 150 and 250 r on the red blood corpuscles; observations on the effect of x-radiation in doses ranging from 10 to 20,000 r on heparinized blood from a normal human donor exposed in-vitro. 37 references included.

——— ———. Medical aspects of civil defense against atomic weapons, by William F. Bale and others. Rochester, N. Y., September 1, 1950, 265 pp (UR–112).

Available from Office of Technical Services, United States Department of Commerce.

——— ———. A unit for exposing animals to radioactive dusts. Rochester, N. Y., January 17, 1957, 47 pp. (Report No. UR–309).

Setter, Lloyd R. and Straub, Conrad. The distribution of radioactivity from rain. Cincinnati, Ohio, United States Public Health Service, Robert A. Taft Sanitary Engineering Center, 1957, 18 p. Mimeographed.

For presentation at the American Geophysical Union Meeting, Washington, D. C., April 29 to May 1, 1957.

Setter, Lloyd R. and Goldin, A. S. Radioactive fallout studies in surface waters. Cincinnati, Ohio, United States Public Health Service, Robert A. Taft Sanitary Engineering Center, April 5, 1955.

Between March 10 and November 15, 1954, 79 rain and 6 dust samples were collected in Cincinnati, and measured for beta activity. A total of 0.53 μc per square meter was deposited during the period. The maximum accumulated activity at any one time was 0.125 $\mu c/M^2$ and by November 16 0.07 $\mu c/M$ remained from 1954 deposition, due to decay. An additional 0.0127 $\mu c/M^2$ theoretically remained from 1953 deposition. Vegetation, roofing, dust, etc., was 2 to 1,000 times more active than the rain. Nonfilterable activity in rain averaged from 46 to 69 percent of total, individual values ranged from 18 to 91 percent. Maximum rainfall activity 10.6 $\mu\mu c$/ml on March 13. Studies of activity in cisterns and pond. Monthly average pond activity ranged from 28 to 230 $\mu\mu c$/ml, 57 to 92 percent nonfilterable.

Sparrow, Arnold H. and Rubin, B. A. Effects of radiation on biological systems. Oak Ridge, Tenn., Technical Information Service, 1955, 53 p.

U. S. Air Force. Radioactivity fallout plots. Washington, Air Weather Service, Military Air Transport Service, June 1956, 39 p.

U. S. Armed Forces Special Weapons Project. Dosage and dose-rate curves of residual radiation based on multiple decay of gross fission products. Washington, D. C., 1951, 20 p.

——— ———. Radiological defense. Washington, D. C., United States Government Printing Office, 1948, 3 v.

U. S. Army. Disposal of radioactive material. Washington, United States Government Printing Office, 1956. 3 p. (Army Regulation 755–380.)

———. Chemical Center. Chemical and Radiological Laboratories. Application of reciprocity to gamma-ray shielding studies. Special report covering May 1955 to October 1955, by Ralph R. Fullwood and Donald R. Roberts, 19 p.

Deals with shielding from fallout.

U. S. Army Institute of Pathology. Statistical analysis of the medical effects of the atomic bombs. From the Report of the Joint Commission for the Investigation of the Effects of the Atomic Bomb in Japan, by Ashley W. Oughterson [and others], April 19, 1951. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, February 28, 1955, 288 p. (TID–5252.)

Formerly issued as NP–3040, Medical effects of atomic bombs; the report of the Joint Commission for Investigation of the Effects of the Atomic Bomb in Japan; volume V.

A detailed statistical analysis of the medical histories of 6882 individuals in Hiroshima and 6621 in Nagasaki who were alive 20 days after the atomic bombings.

Available from Office of Technical Services, United States Department of Commerce.

U. S. Atomic Energy Commission. Assuring public safety in continental weapons tests. Washington, United States Government Printing Office, 1953, 210 p.

——— ———. Biological effects of external x and gamma radiation, by R. E. Zirkel. Oak Ridge, Tenn., Technical Information Service, August 1956, 477 p. (TID–5220).

———. Discussion of radiological safety criteria and procedures for public protection at the Nevada test site, by Gordon M. Dunning. Washington, D. C., February 1955, 75 p. (WASH–290).

———. The effects of atomic weapons. Washington, United States Government Printing Office, June 1950, 456 p.

———. The effects of high-yield nuclear explosions; statement by Lewis L. Strauss, Chairman, and A report by the United States Atomic Energy Commission. Washington, United States Government Printing Office, 1955, 19 pp.

———. Report of off-site radiological safety activities, Operation TEAPOT, Nevada test site, Spring 1955. Prepared for the Test Division, Santa Fe Operations Office, by J. B. Sanders [and others]. [Las Vegas, Nev.?] 1955, 153 pp.

———. Safety levels for contamination from fallout from atomic weapons trials. Oak Ridge, Tenn., Technical Information Service Extension, April 12, 1957, 21 pp. (Foreign Weapon Effects Reports, 107).

———. Sanitary engineering aspects of the atomic energy industry. A seminar sponsored by the AEC and the Public Health Service, held at the Robert A. Taft Engineering Center, Cincinnati, Ohio, December 6–9, 1955. Oak Ridge, Tenn., Atomic Energy Division, Technical Information Service Extension, 1956, 2 v.

———. Meteorology and atomic energy. Washington, United States Government Printing Office, July 1955. 169 pp.

———. Semiannual reports. Washington, United States Government Printing Office, 1953:

13th semiannual report, January 1953. Describes the mobile and fixed monitoring networks in operation for the Nevada tests of spring 1952.

14th semiannual report July 1953. Radioactivity from Nevada tests: 48–52. Discusses fallout from spring 1953 tests. Highest off-site radiation level in populated areas was a 13-week exposure of 9 r, lifetime theoretical maximum dose of 13 r, near Bunkerville, Nev.

15th semiannual report, January 1954. Effects of tests on animals: 50–51.

16th semiannual report. July 1954. Radiation exposures in recent weapons tests: 51–54. Effects of fallout on Mashallese and on Japanese fishermen.

17th semiannual report, January 1955. Residents of Marshall Islands: 43–44. Radiobiology conference in Japan: 44.

18th semiannual report, July 1955. Fallout during Operation Teapot: 77–81.

19th semiannual report, January 1956. Long term effects of fallout from nuclear weapons: 69–72.

20th semiannual report, July 1956. Radiation effects and treatment: 104–115.

21st semiannual report, January 1957. Fallout after Eniwetok tests and its effects on natives and servicemen: 112. Radioactive wastes: 151–161.

———. United States sea disposal operations; a summary to December 1956, by Arnold B. Joseph, Washington, D. C., 1957, 9 pp. (WASH–734).

———. Los Alamos Scientific Laboratory. A hand method for the computation of fallout patterns. Los Alamos, N. Mex., March 1956, 44 pp. (LAMS–2019).

——— ———. Removal of radioactive fallout from water by municipal and industrial water treatment plants, by R. A. Lauder and Rolf Eliassen. New York, March 1, 1956, 79 pp. (NYO–4441).

——— ———. Health and Safety Division. Dust and precipitation sampling program, April through June 1951. New York, 1951.

A network of trays for collecting and filtering settled dust and precipitation was established at 10 stations, principally in the Northeastern United States, during the 1951 Pacific test series. Air filter sampling was also attempted, but at the sampling rate of 1 cfm no significant radioactivity was found. The maximum tray activity was slightly over 1000 d/m/ft^2/day of beta activity at Rochester, N. Y., on April 12, 1951.

——— ——— ———. Survey of fallout of radioactive material following the Las Vegas, Nevada test explosion. New York, February 27, 1951.

Radioactivity was reported in snow in the northeastern United States during the Nevada tests of January and February 1951. Snow and rain samples were collected on February 2 and 3 and again on February 6–9. Activity in filtrate and residue was measured, the highest total activity found was 25,300 d/m/l, corrected to February 2, 1951, at Hannibal, N. Y., in a sample collected on February 3.

——— ———. Health and Safety Laboratory. Description of fallout monitoring system used at Health and Safety Laboratory. [New York, N. Y., n. d.], 6 pp.

——— ——— ———. Fallout countermeasures for AEC facilities, by Alfred J. Breslin and Leonard R. Solon. Oak Ridge, Tenn., Technical Information Extension, 1955, 42 pp.

Available from Office of Technical Services, United States Department of Commerce.

——— ——— ———. Radioactive debris in North America from Operation TEAPOT, by Daniel Lynch. New York, August 1955, 6 pp. (NYO–4659).

Total fallout from the spring, 1955, Nevada tests as observed at over 100 stations in North America for the period of February 18 through May 20, 1955, is given. Fallout in the United States ranged from 3.1 millicuries per square mile at Ely, Nev., extrapolated to January 1, 1956. (Data in the immediate vicinity of the tests are not given.)

——— ——— ———. Radioactive fallout in North America from Operation TEAPOT, by R. J. List. Oak Ridge, Tenn., Technical Information Service, February 1956, 128 pp. (NYO–4696).

Meteorological trajectories of debris from the Nevada atomic tests in the spring of 1955 are given, together with the daily fallout at over 100 gummed-film stations in North America for the period February 18–May 20, 1955. Experiments indicate that on dry days, inverted and vertical gummed films collect about 40 percent as much as normal horizontal films; on days with precipitation, about 1 to 4 percent as much. Radio autographs of individual raindrops indicate that during a period of fallout at Chicago, about three drops in a hundred were radioactive.

———. Test Information Office, Las Vegas, Nev. Background information on continental nuclear tests. The spring 1953 series. Las Vegas, 1953, 35 pp.

U. S. Department of Defense and U. S. Atomic Energy Commission. Medical aspects of atomic weapons. Washington, United States Government Printing Office, 1950, 24 pp.

U. S. Federal Civil Defense Administration. Annual report for fiscal year 1956. Washington, United States Government Printing Office, 1957, 119 pp.

Radioactive fallout forecast program: 20.

———. Construction of fallout plots from coded messages provided by the U. S. Weather Bureau. Battle Creek, Mich., May 25, 1955, 10 pp. (Advisory Bulletin No. 188).
Supplement No. 1, August 16, 1955.
Supplement No. 2, September 27, 1955.
Supplement No. 3, January 26, 1956.
Supplement No. 4, October 4, 1956.

———. Cue for survival; Operation Cue. AEC Nevada test site. Washington, United States Government Printing Office, 1955, 162 pp.
Nuclear radiation-residual: 97–99.

———. Facts about fallout. Washington, United States Government Printing Office, 1955, 16 pp.

———. Fallout and winds. Rev. February 1956. Washington, United States Government Printing Office, 1956, 8 pp. (Technical Bulletin, 11–21).

———. Introduction to radioactive fallout; instructor's guide. Washington, United States Government Printing Office, 1955, 10 pp. (Instructor's Guide, 19–1).

———. Medical aspects of nuclear radiation. Washington, United States Government Printing Office, July 1956, 3 pp. (TB–11–24, Radiological Radiation Series).
References.

———. Protection against fallout radiation. Washington, United States Government Printing Office, September 1955, 2 pp. (TB–11–19, Radiation Defense Series).

———. Questions and answers on fallout. Battle Creek, Mich., May 27, 1955, 6 pp. (FYI–ES–194).

———. Radiation physics and bomb phenomenology. Washington, United States Government Printing Office, June 1956, 8 pp. (TB–11–22, Radiological Defense Series).
Gives method of calculating multiple radioactive decay for fission products by use of Kaufman's equation for multiple decay. References.

———. Radiological instruments for civil defense. Washington, D. C., United States Government Printing Office, March 5, 1956, 9 pp.

———. Residual radiation in relation to civil defense. Battle Creek, Mich., February 9, 1955, 10 pp. (Advisory Bulletin No. 179).

———. What you should know about radioactive fallout. Washington, United States Government Printing Office, 1956, 24 pp. (Public Affairs Booklet 7).
Revised June 1956.

United States Food and Drug Administration. Civil defense information for food and drug officials. Compiled and edited by Lowrie M. Beacham [and others]. Washington, 1955, 188 pp.

United States Naval Medical Research Institute, Bethesda, Md. Emergency laboratory organization for the care of large numbers of human beings accidentally exposed to ionizing radiation, by C. R. Sipe [and others]. Bethesda, Md., November 18, 1955, 13 pp. (NM006012.04.91).
The accidental exposure of a group of Marshallese and Americans to radioactive fallout in the spring of 1954 necessitated organizing and equipping an emergency medical team to conduct essential laboratory and clinical examinations on the exposed individuals.

———. Radiology and radioactivity effects of whole body radiation, by E. P. Cronkite and W. George Brecher. Bethesda, Md., November 15, 1951, 25 pp. Documented by list of 165 references.

United States National Bureau of Standards. Handbooks. Washington, United States Government Printing Office, 1949:
42. Safe handling of radioactive isotopes. 1949.
48. Control and removal of radioactive contamination in laboratories. 1951.
49. Recommendations for waste disposal of phosphorus–32 and iodine–131 for medical users. 1951.
50. X-ray protection design. 1952.
51. Radiological monitoring methods and instruments. 1952.
52. Maximum permissible amounts of radioisotopes in the human body and maximum permissible concentrations in air and water. 1953.
53. Recommendations for the disposal of carbon–14 wastes. 1953.
54. Protection against radiations from radium, cobalt–60, and cesium–137. 1954.

55. Protection against betatron-synchrotron radiations up to 100 million electron volts. 1954.
56. Safe handling of cadavers containing radioactive isotopes. 1953.
57. Photographic dosimetry of X- and gamma rays. 1954.
58. Radioactive-waste disposal in the ocean. 1954.
59. Permissible dose from external sources of ionizing radiation. 1954.
60. X-ray protection. 1955.
61. Regulation of radiation exposure by legislative means. 1955.
62. Report of the International Commission on Radiological Units and Measurements. 1956.

United States National Committee on Radiation Protection. Maximum permissible radiation exposures to man. Washington, United States Government Printing Office, 1957, 32 pp. (National Bureau of Standards Technical News Bulletin, v. 41, No. 2).

United States Naval Medical Research Institute, Bethesda, Md. Skin lesions, epilation and nail pigmentation in Marshallese and Americans accidentally contaminated with radioactive fallout, by R. A. Conard [and others]. Bethesda, Md., 1955, 28 pp. (Research Report Project NM-006-012.04.82). Also in Science v. 122, 1955: 1178-1179.

United States Naval Radiological Defense Laboratory, San Francisco, Calif. The effects of combined administration of strontium-90 and external radiation, by S. H. Cohn and W. L. Milne. San Francisco, Calif., 1956, 20 pp. (USNRDL-TR-89).

Effects of strontium-90 and external body radiation administered separately and in combination were determined in terms of platelet level, body weight, mortality, bone alkaline phosphatase level, and thymic and spleen weight.

———. An estimate of the predominant chemical species resulting from a shallow underwater burst of an atomic bomb, by L. R. Bunney and N. E. Ballou. San Francisco, Calif., 1954, 26 pp. (USNRDL-435).

———. A fallout plotting device, by E. A. Schuert. 19 pp. (USNRDL-TR-127).

"A fallout plotting device was developed. The method requires no drafting equipment and is ideally suited for field use. At Operation Redwing it was found that untrained personnel could quickly become proficient in its employment."

———. Calculated activities and abundances of U-235 fission products, by R. C. Bolles and N. E. Ballou. San Francisco, Calif., 1956, 257 pp. (USNRDL-456).

———. Performance specifications for a sound national shelter system, by W. E. Strope. San Francisco, Calif., 1957, 27 pp., illus., tabs. (USNRDL-TR-132).

"The fundamental requirement of a sound shelter system is protection against nuclear radiations from fallout." Estimated shelter construction costs per person range from $30 to about $300, resulting in an estimated total construction cost for the national shelter program of about $15 to $20 billion.

———. Physical state of fission product elements following their vaporization in distilled water and seawater, by A. E. Greendale and N. E. Ballou. San Francisco, Calif., 1954, 28 pp. (USNRDL-436).

———. Radiotoxicity resulting from exposure to fallout simulant. II. The metabolism of an inhaled and ingested simulant of fallout produced by a land-based nuclear detonation, by S. H. Cohn (and others). San Francisco, Calif., 1957, 24 pp., table, diagrams. (USNRDL-TR-118).

Study reproduced in the laboratory an acute exposure of mice to early fallout (2 days old) such as might result from a land-based nuclear detonation.

———. Study of airborne activity resulting from decontamination of ships contaminated with radioactive fallout, by P. Farma. San Francisco, Calif., May 22, 1956, 27 pp. (Project NS 083-001).

———. Uptake, distribution, and retention of fission products in tissues of mice exposed to a simulant of fallout from a nuclear detonation, by S. H. Cohn [and others]. San Francisco, Calif., December 5, 1955, 25 pp. (Project NM-006-015.04).

———, and U. S. Naval Medical Research Institute, Bethesda, Md. Internal radioactive contamination of human beings accidentally exposed to radioactive fallout material; by S. H. Cohn [and others], San Francisco, Calif. May 9, 1956, 50 pp. (USNRDL-TR-86).

Evaluation of nature and extent of the internal radioactive contamination which occurred in human beings as a result of a nuclear detonation in spring 1953 (Operation CASTLE). In view of the short half-life of the most abundant fission products in this situation, the possibility that chronic irradiation effects will occur is quite small.

U. S. Naval Research Laboratory, Washington, D. C. Fallout dosages at Washington, D. C., by I. H. Blifford and H. B. Rosenstock. Washington, D. C., November 3, 1955, 9 pp. (NRL–4654).

Total infinity dosage at Washington, D. C. due to fallout between January 1951 and May 1955 estimated. Nevada tests contributed about 60 percent of the total amount and the Russian tests about 33 percent.

———. Fallout protection offered by standard enlisted men's barracks, by C. W. Malich and L. A. Beach. Washington, D. C., March 28, 1957, 21 pp. (NLR–4886).

———. Radioactivity of air and fallout samples collected on the 80th meridian, by I. H. Blifford and L. B. Lockhart. Washington, D. C., Aug. 1956, 3 pp., tables (NRL Memorandum Report 626).

———. Relationship between the air concentration of radioactive fission products and fallout; by I. H. Blifford [and others] Washington, D. C., November 4, 1955, 10 pp. (NRL–4607).

U. S. Office of Education. Protection against radioactive fallout. [Washington] February 7, 1956, 12 pp. (Civil Defense education project, information sheet 35).

U. S. Public Health Service. Public health problems in civil defense: Outline guide covering sanitation aspects of mass evacuation, radioactive fallout [etc.]. Washington, D. C., United States Government Printing Office, 1956, 28 pp. (publication 498).

———. Water quality studies on the Columbia River, by Gordon G. Robeck [and others]. Cincinnati, Ohio, United States Public Health Service, Robert A. Taft Sanitary Engineering Center, 1954, 99 pp., tables.

Contains radioactivity analyses.

U. S. Weather Bureau. Meteorology and atomic energy. Prepared for United States Atomic Energy Commission. Washington, D. C., 1955, 169 p. Bibliography.

Washington. University, Seattle, Wash. Applied Fisheries Laboratory. Radiobiological survey of Bikini, Eniwetok, and Likiep atolls; July–August 1949. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, 1952, 146 pp. (AECD–3446).

Available from Office of Technical Services, United States Department of Commerce.

A description of tumors on *Ipomoea tuba* from the A-bomb test sites on Eniwetok Atoll; Appendix to radiobiological survey of Bikini, Eniwetok, and Likiep Atolls, July–August 1949, by Susann F. Biddulph and Orlin Biddulph. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, [1952] 24 pp. (AECD–3446 App.).

Tumors on plants of *Ipomoea tuba*, a vine with large heart-shaped leaves, found in an area 400 to 600 yards from the bomb crater on an Eniwetok atoll, 17 months after an atomic explosion, are described in detail. After careful consideration of all possible casual agents, it was concluded that the tumors were radioinduced.

Weisbecker, L. W. and Lane, W. B. Hot laboratory for producing synthetic radioactive fallout. In [Reports of] 1957 Nuclear Congress, Fifth Hot Laboratories and Equipment Conference, March 14–15, 1957, Philadelphia, Pa. [New York, American Society of Mechanical Engineers] 1957, pp. 364–369.

White, M. R. Human and cattle thyroid radioactivity associated with fallout: October 1955 to October 1956. (UCRL 3703).

Technical Reports, Foreign

French

Atomic energy in its repercussions on life and health. Paris, L'Expansion editeur, 1956, 254 pp. (in French).

Papers given at a scientific conference in Paris, July 1955, on dangers of atomic energy and radiation.

Garrigue, Hubert. Investigation of the atomic cloud. Publications scientifiques et techniques du Ministére de l'air. No. 228. [Paris] 1949, 24 pp.

In 3 out of 23 flights at 6,000 meters over France in July 1946, an unknown radioactive material with a half-life of 25 ± 5 hours was detected. Average concentration 0.013×10^{-16} curies/cm^3 radio equivalent. The first detection was on July 20, 1946, 20 days after the first explosion at Bikini. Similar material was detected in 6 out of 15 flights in July, 1948, the first on July 2, 75 days after the Eniwetok test.

JAPANESE

Analysis of the "Bikini ash." (Special collection of papers). Tr. from Japan Analyst (Tokyo) v. 3, 1954: 333–368, 75 pp. (AEC–tr–2104).

Kimura, Kenjino. Introduction to special collection of papers:1–6.

Incident of the Bikini ashes and the fishing boat is reported. Experiences on the boat are recorded, and fallout analyses are compared with those of Nagasaki and Hiroshima.

———. Radiochemical analysis of "Bikini ashes" fallen on board the No. 5 Fukurya Maru on March 1, 1954: pp. 7–27.

Comprehensive analysis was done in order to find the proper method of medical treatment for the victim fishermen on board. Analysis was started on March 18, and ash was found which consisted mostly of $Ca(OH)^2$, activity of which was 0.37 mc./g. on April 23. Cations of the third group (especially rare-earth metals) and fifth group were found to have strong activity by chemical separation. Fractions of each group, anions, Zr and Nb fraction, and U fraction were separated by an ion-exchange method.

Shiokawa, Takanobu, and others. Radiochemical studies on "Bikini ashes" (March 1, 1954): pp. 28–42.

Decay characteristics of the ashes which were brought back by the crew of the Fukuryu Maru No. 5 were; untreated ash $I = ct^{-1.81}$, water solution part $t^{-2.71}$ insol. part $t^{-1.68}$. Radioactive species separated by chemical method with carrier or collector were; nuclide, activity of nuclide (counts/min.) /activity of original sample (counts–min.), and the date of separation, Sr^{89} $6,000/80 \times 10^4$, April 24; Zr^{95}, $280/80 \times 10^4$, –; Ag^{111}, $200/200 \times 10^4$, April 14; Ru^{103}, $2.300/25 \times 10^4$ etc.

Yamatera, Hideo, and others. Radioactive dust from No. 5 Fukuryu Maru: pp. 43–54.

Analyses of radioactive dust collected on board No. 5 Fukuryu Maru were done by chemical separation and measurement of γ-ray energy and half-life of each species. Results are summarized as follows, radioactive nuclide and approximate percent of radioactivity given: Ru^{103}, 4.3–57; Ru^{106}, 1.4; Te^{129}, 1.3; I^{131}, 4.5; I^{132}, 1.0; Te^{132}, 1.0; etc.

Kiba, Toshiyasu and others. Radioactive substances found on the contaminated fish: pp. 55–60.

Radiochemical investigation was done on the substance collected from the surface of tuna fish which were brought back by the No. 5 Fukuryu Maru. Most of radioactivity was found on the scales, which could not be decontaminated by treating with H_2O; 80 percent of activity was removed by washing dried scales with 3N HCl. Paper chromatographic separation of the HCl fraction showed the presence of Ba^{140}, Sr^{89}, Te^{132}, and probably Zr^{95}, La^{140}, and rare earths.

Honda, M. A proposed method of analysis of radioactive substances in rain-water: pp. 73–75.

Kyoto, Japan, University. Institute for Chemical Research. The radioactive dust from nuclear explosion. November 1954, 133 pp. (Bulletin of the Institute for Chemical Research, Supplementary issue, November 1954) (in English).

Shimizu, S., and others. Radioactive dust from nuclear detonation. Survey of the radioactive contamination of the No. 5 Fukuryu Maru: pp. 1–3.

A collection of reports on investigations on No. 5 Fukuryu Maru, a fishing ship which was in the vicinity of the Bikini atoll when nuclear detonation occurred on March 1, 1954. The radiation dosage rate of contamination observed for combined β- and γ-radiation at every part of the ship on March 19, April 21, and May 16 is recorded. The av. value of total γ-dosage for the crew was supposed to lie between 200 and 500 r.

Kikuchi, Takehiko, and others. Properties and size of the radioactive ashes obtained from the No. 5 Fukuryu Maru: pp. 4–11.

Size and radioactivity of the ashes collected from the ship have been measured. Ashes consist of particles which appeared dark when observed through an occular microscope. When observed by side illumination the particles appeared white and several black spots were seen on the surfaces.

———. Radioautographic studies of the radioactive ashes obtained from the No. 5 Fukuryu Maru: pp. 12–17.

Radioautographic studies have been made of the radioactive ashes obtained from the ship by the use of X-ray film, radioautographic stripping plates, and plates of α-emitters. The radioactivity was found not proportional to the size of the particle, and the distribution of radioactivity in each particle was not uniform.

———. Radioautographic studies of the materials obtained from the No. 5 Fukuryu Maru contaminated by radioactive ashes: pp. 29–34.

The contamination was associated with the presence of small radioactive particles. Although these particles were easily scattered, it was difficult to remove them completely. The particles did not penetrate into the interior of clothes of fine meshes. Decontamination by washing with sea water was not perfect.

———. The contamination of the fishes caught by the No. 5 Fukuryu Maru and the foods manufactured from these fishes: p. 35–38.

The radio-contaminated tunas and other fish caught by the ship in the vicnity of Bikini Atoll were studied. The contamination was caused directly by radioactive ashes and was limited to the surface of the fish. No radioactivity was detected in muscles and bones. The contamination of tuna expressed as Co^{60} was 10^{-2}—10^{-3} microcurie per square centimeter of skin and 10^{-1} microcurie per g. scales.

Ishibashi, Masayoshi, and others. Radiochemical analysis of the Bikini ashes: pp. 35–39.

The following nuclides were detected in the Bikini ashes by radiochem. procedures: Ca^{45}, Sr^{89}, Y^{91}, Zr^{95}, Ru^{103}, Nb^{95}, Rh^{103m}, Ru^{106}, Te^{129}, I^{131}, Ba^{140}, La^{140}, Ce^{144}, Pr^{144}, and U^{237}. The ion-exchange method was used for analysis of contaminated rain water which fell on the Kyoto area on May 16, 1954 from which the presence of Sr^{89}, Zr^{95}, and Ba^{140}, was detected. Rare earths seemed also to be present.

———. Analysis of carrier-free radioisotopes by paper chromatography: pp. 60–74.

Rf values of Ru-Rh, Zr, Nb, Y, Ce-Pr, I, Ca, and Sr, are listed. Zr and Nb were separable only when they were developed with mandelic acid of pH 5.2 and 7.9 Elements of the Ce group seemed to be separated when developed with acetylacetone-BuOH.

Kikuchi, Takehiko, and others. The metabolism of fission products. I. The metabolism of the radioactive ashes obtained from the No. 5 Fukuryu Maru: pp. 75–83.

When the radioactive ashes were administered by mouth, the radioisotopes which were chiefly absorbed were alkaline earths, and were deposited mainly in the bones. When, after the removal of the alkaline earths, the radioisotopes contained in the radioactive ashes were administered by mouth in the form of chloride or citrate, the radioisotopes chiefly absorbed were heavy metals such as Ru and Rh.

———. I. Metabolism of the radioisotopes contained in the radioactive ashes obtained from the No. 5 Fukuryu Maru: pp. 84–90.

Among the radioisotopes obtained by separation from ashes on the ship, i. e., Y^{91}, $C^{141, 144}$, Pr^{144}, Ca^{45}, $Sr^{89, 90}$, $Ru^{103, 106}$, Rh^{106}, Zr^{95}, Nb^{95}, and I^{131}, Sr, Ca, and Y were accumulated chiefly in the bones of adult mice, and the elimination of radio-Sr from there was very slow. When administered by mouth, radio-Sr and Radio-Ca were readily absorbed from the digestive tract, while the absorption of radio-Y from the tract was poor.

———. Paper XI, Studies on the metabolism of fission products III. Radioautographic studies on the localization of radiostrontium and radiocalcium in the bones: pp. 99–105.

———. The effects of EDTA–Na (Na Ethylenediaminetetraacetate) upon the metabolism of radiostrontium and radioyttrium in mice: pp. 106–111.

The toxicity of EDTA–Na, inert Sr $(NO_3)_2$ and Ba $(NO_3)_2$ has been examined. Simultaneous injection of EDTA–Na showed no significant effect upon the distribution of radio-Sr in the bones of mice. The distribution of Radio-Y in the bones of mice tended to decrease following the simultaneous subcutaneous injection of Y^{91} and EDTA-Na.

Japanese Committee for Compilation of Report on Research in the Effects of Radioactivity. Research in the effects and influences of the nuclear bomb test explosions. Tokyo, Japan Society for the Promotion of Science, 1956: 1824 pp. in 2 v.

Tokyo, Japan. University. Fisheries Institute and Faculty of Agriculture. Studies on influence of radio-activity from the point of fisheries science. Tokyo, November 10, 1954, 215 pp.

Data for the Committee on the Effects of Radioactivity, Science Council of Japan. Preliminary report iv, by cooperation of staff under Profs. Mori, Matsue, Suehiro, and Hiyama.

BRITISH AND CANADIAN

Canada. Defense Research Board. Fallout from large nuclear explosions with application to civil defense, by G. H. Gilbert. Ottawa, Canada, 1955, 10 pp. (Operational Research Group Memorandum No. 5516).

Appendix A: A report by the United States Atomic Energy Commission on the effects of high-yield nuclear explosions, 8 pp.

"It is important to note that information relating to high level winds is very sparse, and that no information is available on the distribution of particle size and radio-activity within the nuclear cloud resulting from high yield explosions. Any conclusions on results presented in this paper must therefore be considered tentative."

Great Britain. Atomic Energy Research Establishment. Aspects of the travel and deposition of aerosol and vapour clouds, by A. C. Chamberlain. Harwell, Berkshire, England, 1953, 35 pp. (A. E. R. E. HP/R. 1261.)

———. Determination of strontium in sea water using both radioactive and stable isotopes, by R. W. Hummel and A. A. Smales. Harwell, Berkshire, England, 1955, 5 pp. (A. E. R. E. C/R 1755.)

———. Estimation of maximum permissible levels of radiation, by A. C. Chamberlain. Harwell, Berkshire, England, August 1950, 52 pp. (A. E. R. E. HP/R–551.)

———. The effects of Operation Hurricane on plants and soils, by R. Scott Russell [and others]. Harwell, Berkshire, England, 1955, 106 pp. (Soil-Plant-Animal Relationships Report 3).

Fallout samples collected in air filters by aircraft and on gage sheets 24 hours following the Monte Bello burst were analyzed.

———. The metabolism of strontium in man, by G. E. Harrison [and others]. Harwell, Berkshire, England, 1955, 18 pp. (Soil-Plant-Animal Relationships Report 2).

———. The Monte Bello rat, November 1953, by D. W. H. Barnes [and others]. Harwell, Berkshire, England, 1955, 22 pp. (Soil-Plant-Animal Relationships Report 1).

Rats inhabiting Monte Bello at the time of the atomic explosion were examined for both external signs of radiation damage and damage from the ingestion of contaminated food.

———. The radiological dose to persons in the United Kingdom due to debris from nuclear test explosions, by N. G. Stewart [and others]. Harwell, Berkshire, England, 1955, 20 pp. (A. E. R. E. HP/R 1701).

———. The radiological dose to persons in the United Kingdom due to debris from nuclear test explosions prior to January 1956, by N. G. Stewart [and others]. Harwell, Berkshire, England, 1956, 22 pp. (A. E. R. E. HP/R 2017).

———. Radiostrontium and radiocaesium measurement in biological material, to December 1956, 11 pp. (A. E. R. E. HP/R–2182).

———. Radiostrontium fallout in biological materials in Britain, by R. J. Bryant [and others]. Harwell, Berkshire, England, September 5, 1956, 44 pp. (A. E. R. E. HP/R 2056).

Methods are described for the determination of strontium 90 due to fallout from nuclear explosions in soils, vegetation, bones, and milk. Current levels in Britain are shown to be very similar to those reported for United States.

———. Some factors influencing the gastro-intestinal absorption of strontium in rats, by H. G. Jones. Harwell, Berkshire, England, 1955, 3 pp. (Soil-Plant-Animal Relationships Report 6).

———. Summary of leading points [of Tripartite Conference on Soil-Plant-Animal Relationships of Fission Products]. Harwell, Berkshire, England, 1955, 2 pp. (Soil-Plant-Animal Relationships Report 15.)

"Order of importance of hazards from fallout in initial period is (*a*) external radiation, including beta radiation from activity on skin, (*b*) contamination of herbage and crops, (*c*) inhalation of activity and contamination of water supplies. With passage of time (*b*) becomes dominant."

Great Britain Atomic Weapons Research Establishment. Agricultural and biological investigations pertaining to contamination by fission products. Aldermaston, Berkshire, England, July 16, 1954, 26 pp.

Great Britain Medical Research Council. The hazards to man of nuclear and allied radiations. London, Her Majesty's Stationery Office, 1956, 128 pp.

Concludes that risk of radiation is controllable within limits that man can accept.

INDIAN

India (Republic) Defense Science Organisation. Nuclear explosions and their effects. Foreword by Jawaharlal Nehru. [Delhi] Publications Division, Ministry of Information and Broadcasting, Government of India, August 1956, 184 pp.

Bibliographical footnotes.

Scholarly analysis of the strontium hazard fills about 10 percent of the text. A three-page chapter contains a warning for the future and shows the great concern of India about the continued nuclear tests.

RUSSIAN

U. S. S. R. Academy of Sciences. Conference on the peaceful uses of atomic energy. July 1–5, 1955. Session of the Division of Biological Science. Washington, United States Atomic Energy Commission, 1956, 198 pp., in English translation by Consultants Bureau.

CONGRESSIONAL HEARINGS AND CONGRESSIONAL RECORD EXCERPTS

Morse, Wayne. Address . . . Radiation hazards. Remarks in the Senate. Congressional Record [daily edition] v. 103, May 23, 1957: 6747–6750.

Senator Morse discusses the unfortunate effects of radiation on the human system, particularly on the reproductive processes, and berates the administration for its failure to control or stop the testing of atomic weapons.

National Radiation Health Institute to protect mankind from atomic fallout. Congressional Record [daily edition], v. 103, February 14, 1957: 1763–1769

Bill introduced by Senator Neuberger to create a National Radiation Health Institute to perform research in effects of radiation on health. Contains reprints of Unna, Warren. Fallout held sure to hurt 6,000 babies. Washington Post.

Payne, Frederick G. United States participation in scientific commission within the United Nations to study certain effects of nuclear explosions. Congressional Record, v. 101, April 13, 1955: 4339–4340.

United States Atomic Energy Commission. Division of Biology and Medicine. Report on questions raised by Mrs. Jan Howard, East Palo Alto, Calif., regarding radioactive fallout from weapons tests. Extension of remarks of Hon. Charles S. Gubser. Congressional Record, v. 101, May 25, 1955: 7075–7076.

United States Congress. House. Committee on Appropriations. Fallout from nuclear explosions. Testimony of witnesses from Armed Forces Special Weapons Project, Army and Navy. In Department of Defense appropriations for 1958; hearings before the subcommittee of the Committee on Appropriations, 85th Congress, 1st session. Washington, United States Government Printing Office, 1957: pt. 2, p. 1532.

Contains reprint of Kulp, Laurence J. and others. Strontium–90 in man.

——— ———. Committee on Government Operations. Civil defense for national survival. Hearings before a subcommittee, 84th Congress, 2d session. Washington, United States Government Printing Office, 1956, 3,145 pp. (in 7 pts).

Statements by Willard F. Libby: pp.4–66; Willard Bascom: pp. 127–190; Merle A. Tuve: pp. 191–211; Lester Machta: pp. 601–634; Lauriston S. Taylor: pp. 725–756; Ralph E. Lapp: pp. 763–797; Charles L. Dunham: pp. 899–925; Eugene P. Cronkhite: pp. 925–938; H. Bentley Glass: pp. 2709–2742.

——— ———. Civil defense for national survival. Twenty-fourth intermediate report of the Committee on Government Operations. Washington, United States Government Printing Office, 1956, 103 pp. (House Report No. 2946).

Nuclear explosion effects: pp. 8–9.

Radioactive fallout: pp. 9–12.

——— ———. Joint Committee on Atomic Energy. Health and safety problems and weather effects associated with atomic explosions. Hearing before the Joint Committee on Atomic Energy, 84th Congress, 1st session, April 15, 1955. Washington, United States Government Printing Office, 1955, 60 pp. (committee print).

Testimony by various authorities, including AEC and Weather Bureau experts, on various phases of possible hazards from atomic tests. Eisenbud states the cumulative fallout in the United States from early 1951 to January 1, 1955, varies from 21 millicuries per square mile in Arizona to 120 millicuries per square mile in New Mexico. The normal radioactive background in the United States varies from about 0.01 to 0.05 mr/hr. On six occasions, the background radiation was elevated to about 1 mr/hr beyond a few hundred miles from the test site, Troy, Chicago, Rochester, Salt Lake City (twice) and one other.

——— ———. Hearings on atomic-hydrogen weapon radiation, Congressional Record [daily edition], v. 103, May 22, 1957: 6659–6664.

List of witnesses and a detailed outline describing the scope and content of the hearings.

——— ———. Senate. Committee on Armed Services. Testimony [on fallout] of Gen. James M. Gavin, Chief of Research and Development, Department of the Army, in study of airpower; hearings before the Subcommittee on the Air Force (Symington Committee) of the Committee on Armed Services, 84th Congress, 2d session, Washington, United States Government Printing Office, 1956: pt. X, pp. 860–861.

——— ———. Senate. Committee on Armed Services. Civil defense program; hearings before the Subcommittee on Civil Defense * * * 84th Congress, 1st session, February 22, 26, March 3, 4, and 8, 1955. Washington, United States Government Printing Office, 1955, 920 pp. (in 2 pts.) (committee print).

Statement of Willard F. Libby: pp. 4–22.

Exhibit 1. Statement [on fallout] by Lewis L. Strauss: pp. 231–233.

A report by the United States Atomic Energy Commission on the effects of high-yield nuclear explosions: pp. 234–240.

Exhibit 2. Remarks by Dr. Willard F. Libby for delivery at the Washington Conference of Mayors, Washington, D. C., Thursday, December 2, 1954: pp. 240–244.

Exhibit 3. Atomic test effects in the Nevada Test Site Region: pp. 244–253.

Statement of Ralph E. Lapp [on fallout]: pp. 688–708.

——— ———. Senate. Committee on Foreign Relations. Control and reduction of armaments. Hearing before a subcommittee * * * 84th Congress, 2d session * * * Washington, United States Government Printing Office, 1957, 1333 pp. (in 13 pts.)

Statements by Mrs. Adelaide N. Baker: pp. 478–492; Arthur H. Compton: p. 819; Mrs. George Gellhorn: pp. 900–903; T. Alexander Pond: pp. 907–911; Mrs. Frederick Faust: pp. 951–953; Warren Weaver: pp. 1135–1154.

UNITED NATIONS PUBLICATIONS

Effects of atomic radiation; unanimity on establishing scientific group [in United Nations]. United Nations review, v. 2, January 1956: 7–18.

Resolution adopted by the General Assembly on December 3, 1955, ibid, p. 63.

International Conference on the Peaceful Uses of Atomic Energy. Proceedings of the International Conference . . . held in Geneva, August 8–August 20, 1955. New York, United Nations, 1956, 16 v.

Vol. 7: Nuclear chemistry and effects of irradiation:

Paper 1058: Radiochemical analysis of radioactive dusts, by Kenjiro Kimura (Japan): pp. 196–209.

Paper 1059: Radiochemical interpretation of the radioactive fallout, by Kenjiro Kimura and others (Japan): pp. 210–213.

Vol. 11: Biological effects of radiation:

Paper 392: The experimental animal for study of the biological effects of radiation, by J. F. Loutit (UK): pp. 3–6.

Paper 616: The influence of ionizing radiations on animal organisms, by A. V. Lebedinsky (U. S. S. R.): pp. 7–24.

Paper 803: Biological effects of radiation, by John C. Bugher (USA): pp. 45–48.

Paper 852: Studies on the radium content of humans arising from the natural radium of their environment, by A. F. Stehney and H. F. Lucas (USA): pp. 49–54.

Paper 256: Effects of whole body exposure to nuclear or X-ray energy on life span and life efficiency, by J. W. Gowen (USA): pp. 105–109.

Paper 244: A formulation of the relation between radiation dose and shortening of life span, by H. A. Blair (USA): pp. 118–120.

Paper 1045: Radiation injury due to radioactive fallout, by Masao Tsuzuki (Japan): pp. 132–133.

Paper 88: The deposition of radioactive substances in bone, by Frank E. Hoecker (USA): pp. 138–145.

Paper 899: Biological damage resulting from exposure to ionizing radiation, by L. H. Gray (UK): pp. 209–212.

Paper 449: The genetic problem of irradiated human populations, by T. C. Carter (UK): pp. 384–386.

Paper 234: How radiation changes the genetic constitution, by H. J. Muller (USA): pp. 387–399.

Vol. 13: Legal, administrative, health and safety aspects of large-scale use of nuclear energy.

Paper 778: The general problems of protection against radiations from the public health point of view, by World Health Organization: pp. 10–14.

Paper 778: The achievement of radiation protection by legislative and other means, by Lauriston Taylor (USA): pp. 14–21.

Paper 394: Radiological hazards from an escape of fission products and the implications in power reactor location, by W. G. Marley and T. M. Fry (UK): pp. 102–105.

Paper 572: Radiation from clouds of reactor debris, by J. Z. Holland: pp. 110–118.

Paper 451: Radiation injury and protection—maximum permissible exposure standards, by W. Binks (UK): pp. 129–131.

Paper 89: Maximum permissible exposure standards, by Robert S. Stone (USA): pp. 132–138.

Paper 79: Maximum permissible concentration of radioisotopes in air and water for short exposure periods, by Karl Z. Morgan and others: (USA): pp. 139–158.

Paper 247: Permissible exposure to ionization radiation, by Lauriston S. Taylor (USA): pp. 196–197.

Paper 1041: Maximum permissible exposure standards, by Masanori Nakaidzumi (Japan): p. 198.

Paper 944: On the maximum permissible dose of X- and gamma-radiation, by W. Jasiński and I. Zlotowsky (Poland): pp. 199–200.

Paper 245: Validity of maximum permissible standards for internal exposure, by R. C. Thompson and others (USA): pp. 201–204.

Paper 250: Is the concept of "critical organ" valid in determining the maximum permissible level for exposure to radioactive materials? By J. N. Stannard (USA): pp. 205–209.

Paper 276: Atomic energy and meteorology, by H. Wexler and others (USA): pp. 333–344.

Paper 1055: Radioactivity in rainwater and the air observed in Japan, 1954–1955, by Yasuo Miyake (Japan): pp. 345–349.

Paper 278: The absorption of fission products by plants, by J. H. Rediske and F. P. Hungate (USA): pp. 354–356.

Paper 1066: Biological cycles of fission products in agriculture in Japan, by R. Sasaki (Japan): pp. 357–359.

Paper 393: The behavior of I^{131}, Sr^{89} and Sr^{90} in certain agricultural food chains, by A. C. Chamberlain and others (UK): pp. 360–363.

Paper 280: The accumulation of radioactive substances in aquatic forms, by R. F. Foster and J. J. Davis (USA): pp. 364–367.

Paper 1052: Biological cycle of fission products considered from viewpoint of contamination of marine organisms, by Yoshio Hiyama (Japan): pp. 368–370.

Paper 277: Nuclear science and oceanography, by R. Revelle and others (USA): pp. 371–380.

Paper 1057: On the distribution of radioactivity in the North Pacific Ocean in 1954–1955, by Yasuo Miyake (Japan): pp. 381–384.

Paper 281: Radioactivity in terrestrial animals near an atomic energy site, by W. C. Hanson and H. A. Kornberg (USA): pp. 385–388.

Kenny, A. W. The safe disposal of radioactive wastes. Bulletin of the World Health Organization (Geneva) v. 14, 1956: 1007–1060.

Stone, R. S. Maximum permissible exposure standards. United Nations reports, v. 13, p. 132.

Trusteeship Council considers new petition from Marshall Islanders. United Nations review, v. 2, May 1956: 61–63.

United Nations. Scientific Committee on the Effects of Atomic Radiation, First yearly progress report of the [United Nations] Scientific Committee on the Effects of Atomic Radiation to the General Assembly. United States Department of State bulletin, v. 35, December 10, 1956: 931–935.

———. World Health Organization. The effect of radiation on human heredity; report of a study group, Copenhagen, August 7–11, 1956. [Geneva]. January 24, 1957, various paging.

REPORTS OF INTERNATIONAL AGREEMENTS

Correspondence between President Eisenhower and Soviet Premier Bulganin concerning nuclear tests. Department of State bulletin, v. 35, 1956: 662–664.

U. S. Treaties, etc., 1953 (Eisenhower). Settlement of Japanese claims for personal and property damages resulting from nuclear tests in Marshall Islands in 1954. Agreement between the United States of America and Japan, effected by exchange of notes signed at Tokyo, January 4, 1955. Washington, United States Government Printing Office, 1955. (U. S. Dept. of State. Publication 5842. Treaties and other international acts series, 3160).

U. S. offers aid in measuring radioactive fallout. Department of State bulletin, v. 35, July 2, 1956: 41.

United States and United Kingdom exchange views at Bermuda meeting. Department of State bulletin, v. 36, April 8, 1957: 561–562.

Annex II deals with nuclear tests and methods of limitation.

Wadsworth, James J. Coordination and dissemination of information on effects of atomic radiation. Department of State bulletin, v. 33, November 21, 1955: 851–858.

Statements of United States Representative to the General Assembly pointing out the need for a fact gathering service to coordinate and disseminate information about the dangers of radioactivity.

Warren, Shields. Launching the U. N. study of effects of atomic radiation. Department of State bulletin, v. 34, May 21, 1956: 860–861.

First meeting of Scientific Committee on Effects of Atomic Radiation, March 14–23.

Appendix 10

Oak Ridge National Laboratory,
Oak Ridge, Tenn., August 21, 1957.

Mr. James T. Ramey,
Executive Director, Joint Committee on Atomic Energy,
Washington, D. C.

Dear Mr. Ramey: Enclosed please find a copy of the material concerning topic VIII D of the outline, fallout and water decontamination, requested by Congressman Holifield for the Joint Committee on Atomic Energy Report.

Enclosed is the biographical sketch also requested in your letter of June 19, 1957.

If I can be of any further assistance to you and the committee, please feel free to write.

Thank you.

Very truly yours,

William J. Lacy,
ERDL Representative at ORNL.

Enclosures: 1. Report on Fallout. 2. Biographical sketch.

Cc: Commanding Officer, Engineer Research and Development Labs, Fort Belvoir, Virginia; Harry N. Lowe, Jr., Chief Sanitary Engineering Branch, Fort Belvoir, Virginia; Dr. Karl Z. Morgan, Director, Health Physics Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Biographical Sketch

William J. Lacy was born in 1928 in Wallingford, Connecticut, attended Lyman Hall High School where he won the prizes in science and chemistry, then he obtained a B. S. degree in 1950 from the University of Connecticut where he majored in Chemistry. He entered graduate school at New York University in September of 1950 and worked as a research associate on an AEC research contract. In May of 1951 he joined the staff at the Engineer Research and Development Labs of Fort Belvoir, Virginia, and immediately was transferred to the Oak Ridge National Laboratory to work on the water decontamination research project.

He has had seven (7) articles published, presented numerous papers and is a member of the American Chemical Society, American Association for the Advancement of Science and the Scientific Research Society of America.

Mr. Lacy is married and has two (2) sons, 2½ and six months, he resides in Oak Ridge, Tennessee.

[Material for Joint Committee on Atomic Energy Topic VIII D]

Removal of Radioactive Fallout From Contaminated Water Supplies

William J. Lacy, Chemist,* Sanitary Engineering Branch, Engineer Research and Development Labs, Fort Belvoir, Va.

There are two possible sources of radioactive contamination of public water supplies, (1) the result of direct discharge into the environment from reactor processing plants, research center using radioisotopes and others and (2) deposition of radioactive material by fallout or wash-in due to weapon's test activities.

Most of the radioactive materials in item one are in solution, fallout, however, may be in the form of insoluble oxides, and its removal may differ from the removal of ionic material.

Studies have been reported on the subject of fallout in particular areas (1), (2), (3), (4). It was reported that 35 percent of the fallout activity was removed by the Albany, New York, water treatment plant, an alum coagulation, settling and filtration plant. Thomas and his coworkers at Harvard (2) (3) working at the Lawrence, Massachusetts, water plant obtained 80 percent removal by coagulation, settling, and filtration. Bell (4) compared the fallout removal results from Cambridge and Lawrence, Massachusetts, and Rochester, New York, with pilot plant results obtained by Straub (5) (6) who used a simulated bomb blast mixture with an age about one month after detonation.

*On loan to Health Physics Division, Oak Ridge National Lab., Oak Ridge, Tennessee.

The comparison indicated the three treatment plants show much lower removals of fallout than Straub obtained on chemical processed radioactive material even though the same procedure was used in both cases. The U. S. P. H. S. reported the analysis of rain water samples containing fallout showed 50 to 100 percent of the "old" radioactive material to be soluble. However, the soluble fraction dropped to about 30 percent during the weapon's testing period.

For reactor made fission products, or a mixture of commercially available radioisotopes, the efficiency of removal would be a function of the various radioelements comprising the mixture. Results in laboratory studies and pilot plant scale investigations by the author indicates removals of about 70 to 85 percent using either alum and soda ash or ferric chloride and limestone coagulants. A series of studies (7) reported that removals of 99 percent could be obtained using a serial coagualtion procedure including an excess lime-soda ash softening or phosphate coagulation step, provided some clay material was added to remove radiocesium.

Conventional wastes treatment processes include coagulation, settling, and filtration, plus disinfection. Often additional treatment, such as fluoridation, aeration, softening, ion exchange, iron and manganese removal are employed.

During coagulation certain of the dissolved constituents are precipitated as insoluble hydroxides or carried along, scavenged, with the heavy metal hydroxides of alum or iron. Coagulation can have its radioactivity removal increased from about 75 percent to almost 90 percent by the addition of clay for cesium and copper sulfate for radioiodine.

It should be pointed out that different radioisotopes respond differently to removal by coagulation. Other factors to be considered include: (1) Chemical and physical form of the radionuclide, (2) concentration or the radioactive material, and (3) optimum pH of flocculation for the coagulant available and the water under treatment. Investigation by the author (8) indicates increase dosages of chemical generally yielded only slightly higher removals while higher pH usually resulted in proportionately higher removals.

Softening using lime-soda ash is one of the more effective chemical methods for the removal of radiostrontium and barium. However, it is necessary to use excesses quantities, over the stoichiometric dosage, for satisfactory results. Studies at MIT (9) (10) have indicated that the radiostrontium is removed by coprecipitation with the hardness or calcium carbonate in a mixed crystal formation.

Ion exchange is another method used by some municipal water treatment plants. Removal of ionic radionuclides by this process is not only technically possible (11), but very satisfactory. The most effective method employes either a mixed bed principal or separate cation-anion exchange columns. Ion exchange units such as home-type water softeners are very effective for removal of 99^+ percent of the radioactive fallout or reactor originated radionuclides from contaminated water. Also ion exchange resins (mixed) can be used with, good results, as slurries for the removal of a vaniety of radioactive contaminants from water solutions (12)

Other methods, such as, the use of clays, powdered metal, charcoal, flotation and various adsorbents all have some merit for the removal of specific radioisotopes or under a given set of condition result in good removals. (13) However, clay seems to have the most practical and over advantage of being (1) available, (2) cheap, (3) effective, (4) simple to use, (5) easy to remove both absorbent and absorber and the radioactive material will not be easily leached once it is attached to the clay particle. Distillation although not a usual municipal water treatment method is used extensively by the military on island bases and where a high quality of water is required. Distillation results in the best single treatment of a contaminated water removing 99.9^+ percent. (14) The major objection to distillation as a water treatment procedure is cost.

As indicated by the literature cited most of the above studies have been made on chemically processed, radiochemically pure radioisotopes and not true fallout from a nuclear detonation. Therefore, it was expected that the actual fallout material not being entirely in the same physical and chemical form could not be as readily removed from contaminated water. However, recent tests by the Corps of Engineers at the AEC Nevada Proving Grounds on some very low level fallout indicated (1) close agreement with laboratory results on removal by coagulation and softening using lime-soda ash and precipitation with trisodium phosphate at a high pH, (2) the ion exchange procedures resulted in 99 to 100 percent removal of the bomb fallout material, (3) the material that was not

a true solution could be removed physically and the material in solution treated chemically and (4) radionuclide once adsorbed on clays were not appreciably leached by tap water.

Many other experiments have been made by myself and others, some are still in progress, which have not been cited here. It is felt that this brief general review plus the six tables showing detailed data, will give the committee a review of the field on water decontamination.

REFERENCE CITED

1. Kilcawley, E. J., H. M. Clark, H. L. Ehrlich, W. J. Kelleher, H. E. Schultze, and N. L. Krascella. Measurement of Radioactive Fallout in Reservoirs. Jour., A. W. W. A., *46*, 1101 (November 1954).

2. Thomas, Harold A., Jr., R. Stevens Kleinschmidt, Frank L. Parker, and Carlos G. Bell, Jr. Radioactive Fallout in Massachusetts Surface Waters. Jour., A. W. W. A., *45*, 562 (June 1953).

3. Bell, Carlos G., Jr., Harold A. Thomas, Jr., and Barnet L. Rosenthal. Passage of Nuclear Detonation Debris Through Water Treatment Plants. Jour., A. W. W. A., *46:* 10, 973 (October 1954).

4. Nader, J. S., A. S. Goldin, and L. R. Setter. Radioactive Fallout in Cincinnati Area. Jour., A. W. W. A., *46:* 1096 (November 1954).

5. Straub, Conrad P., Roy J. Morton, and Oliver R. Placak. Studies on the Removal of Radioactive Contaminants from Water. Jour., A. W. W. A., *43:* 773 (October 1951).

6. Straub, Conrad P. Removal of Radioactive Waste from Water. Nucleonics, *10:* 1, 40 (January 1952).

7. Lacy, W. J., Rollins, R. R. and Lawless, L. M. "Removal of Radioactive Material From Water by Serial Coagulation, Ion Exchange and by Charcoal Adsorption", ERDL Report No. 1451–RR, 22 June 1956.

8. Lacy, W. J. "Removing Radioactive Material from Water by Coagulation" Water and Sewage Works *100*, 10, 410 (October 1953).

9. McCauley, Robert F., Robert A. Lauderdale, and Rolf Eliassen. A Study of the Lime-Soda Softening Process as a Method for Decontaminating Radioactive Waters. Report NYO–4439. Sedgwick Laboratories of Sanitary Science, Massachusetts Institute of Technology, Cambridge, Massachusetts (September 1, 1953).

10. Nesbitt, John B., Warren J. Kaufman, Robert F. McCauley, and Rolf Eliassen. The removal of Radioactive Strontium from Water by Phosphate Coagulation. Report NYO–4435. Massachusetts Institute of Technology, Cambridge, Massachusetts. (February 15, 1951).

11. Lacy, W. J., and Don C. Lindsten "Water Decontamination, An Ion Exchange Pilot Plant Study," ORNL–CF–Report No. 55–10–153 (October 1953).

12. Lacy, W. J. and D. C. Lindsten "Removal of Radioactive Contaminants from Water by Ion Exchange Slurry" I & E Chemistry *49*, 10 (October 1957).

13. Lacy, W. J., Decontamination of Radioactively Contaminated Water by Slurry with Clay. Ind. Eng. Chem., *46:* 1061 (May 1954).

14. Lacy, W. J., D. C. Lindsten, and H. N. Lowe "Removal of Radioactivity from Water by Thermocompression Distillation" ERDL Report No. 1313 (August 53).

TABLE I.—*Coagulation for removal of radioactivity*

Contaminant	Dosage, p. p. m.	Percent removal	
		$FeCl_3$-$CoCO_3$	Alum-soda ash
Ce^{144}–Pr^{144}	50	99.2	96.1
	100	99.4	96.5
Ba^{140}–La^{140}	50	67.4	58.4
	100	70.7	58.0
Zr^{95}–Nb^{95}	50	98.1	76.4
	100	98.8	78.6
I^{131}	50	45.0	26.3
	100	63.0	35.7
P^{32}	45.7	93.3	94.1
MFP-1 [1]	29–58	60–83.7	----------
MFP-2 [2]	50	70.1	72.6

[1] MFP-1—ORNL waste containing mixed fission products.
[2] MFP-2—Simulated 30-day atomic-bomb blast mixture.

TABLE II.—*Results of lime-soda ash treatment for removal of strontium*

Treatment	Percent removal of activity
Stoichiometric amounts	75.0
20 ppm excess lime-soda ash	77.0
50 ppm excess lime-soda ash	80.1
100 ppm excess lime-soda ash	85.3
150 ppm excess lime-soda ash	97.3
200 ppm excess lime-soda ash	99.4
300 ppm excess lime-soda ash	99.7

TABLE III.—*Ion exchange column for water decontamination*

Run No.	Resin*	Contaminant	Resin capacity gal./ft.^{3}	Percent removal until breakthrough
1	Cation	MFP-1	5,700	71–82
2	Mixed bed	MFP-1	3,150	93–99+
3	Cation	MFP-2	6,000	88–96
4	Mixed bed	MFP-2	2,890	96–99
5	Cation	Zr^{95}–Nb^{95}	6,750	85–88
6	Mixed bed	Zr^{95}–Nb^{95}	2,600	92–97
7	Cation	MFP-3	3,270	85–90
8	Mixed bed	MFP-3	6,150	92–99

*Cation resin was a high capacity nuclear sulfonic acid type and mixed bed was amberlite MB-3.

NOTES

MFP-1—ORNL liquid waste material.
MFP-2—Simulated 30-day atomic-bomb debris.
MFP-3—Three year old dissolved reactor fuel element.

TABLE IV.—*Removal of radioactive contaminants from water—Resin-jar test studies (stirring time, 90 minutes, samples filtered)*

Contaminant	Initial pH	Initial activity c/m/ml	Percent removal mixed ion exchange resin, p. p. m.			
			450	900	1,800	2,700
P^{32}	8.2	5,560	47.4	74.5	96.2	99.8
Cd^{115}	8.0	7,880	37.9	45.6	91.1	99.99
Cs^{137}–Ba^{137}	8.2	8,200	15.1	14.6	69.1	99.99
Zr^{95}–Nb^{95}	8.1	6,700	98.3	98.4	99.2	99.4
I^{131}	7.5	3,200	84.5	93.5	95.6	98.1
$Ce^{141, 144}$–Pr^{144}	7.9	4,150	98.7	99.2	99.8	99.98
Ba^{140}–La^{140}	7.6	3,490	85.1	94.5	98.8	99.9
FPM-4	8.3	13,600	82.7	90.5	97.3	99.2
FPM-5	2.7	3,400	38.4	----	----	----

NOTES

FPM-4—Iodine dissolver solution aged 30 days.
FPM-5—Mixed fission product waste containing mainly Cs^{137}–Ba^{137} and Ru^{106}–Rh^{106}.

TABLE V.—*Decontamination of radioactively contaminated water by slurrying with clay*

Contaminant	pH	Clay concentration, p. p. m.	
		1,000	3,000
		Percent removal	
Ru^{106}–Rh^{106}	5.2	50.5	61.5
Zr^{95}–Nb^{95}	7.5	98.0	99.4
Sr^{90}–Y^{90}	7.7	83.4	92.9
I^{131}	7.5	4.9	3.4
$Ce^{141,144}$–Pr^{144}	8.0	99.7	99.9
Ba^{140}–La^{140}	7.8	88.8	94.3
MFP–1	8.8	82.0	86.3
MFP–2	9.0	70.0	72.8
MFP–3	7.7	79.0	83.6

TABLE VI.—*Removal of radioactive material by distillation (60 gallon/hr thermocompression unit)*

Run No.	Contaminant	Activity of feed, d/m/ml	Removal of activity expressed as decontamination factor	Percent
1	MFP–1	22,060	4.10×10^3	99.98
2	MFP–2	97,400	4.97×10^3	99.98
3	MFP–3	31,150	3.59×10^3	99.97
4	MFP–4	62,400	3.52×10^2	99.72
5	Pa^{233}	41,030	2.31×10^3	99.96
6	I^{131}	60,900	7.04×10^2	99.86
7*	MFP–5	38,910	1.09×10^3	99.91
8*	MFP–4	69,700	1.00×10^4	99.99
9*	MFP–1	12,020	1.70×10^4	99.99
10*	I^{131}	45,600	1.28×10^3	99.92
11*	Pa^{233}	25,300	5.80×10^3	99.98

*Glass wool reflux condenser used.

NOTES

MFP–1 was 3-year-old fission product mixture.
MFP–2 was a 2-week-old mixture from disolution of a reactor slug.
MFP–3 was composite sample or ORNL liquid waste.
MFP–4 concentrate from ORNL liquid waste evaporator.
MFP–5 mixture to simulate the material expected 10 days after atomic detonation.

APPENDIX 11

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington 25, D. C., August 20, 1957.

Hon. CHET HOLIFIELD,
Chairman, Special Subcommittee on Radiation of the Joint Committee on Atomic Energy, House of Representatives, Congress of the United States.

DEAR MR. HOLIFIELD: At the suggestion of your Committee, the Division of Biology and Medicine, U. S. Atomic Energy Commission, invited the principal participants in the discussions involving predictions of future skeletal concentrations of strontium 90 in humans which took place at the recent Congressional Hearings on fallout to meet once again in an attempt, insofar as present information permitted, to reduce the degrees of uncertainty in these predictions.

This meeting took place on July 29, 1957 and I am pleased to transmit a summary report of the meeting based on the stenographic transcript and consultation with the principal participants. This report was prepared by Dr. Forrest Western, of the Division of Biology and Medicine. It is my opinion this report honestly and clearly reflects the views of the participant scientists with respect to this problem. This document, then, would appear to reflect the thinking of those scientists who have worked hardest and thought most on the subject of these predictions, and should, therefore, be a useful addition to the text of the very important and

informative Hearings which your Committee held in May and June of this year on the whole matter of fallout from weapons tests.

Sincerely yours,

CHARLES L. DUNHAM, M. D.,
Director, Division of Biology and Medicine.

PREDICTED SKELETAL CONCENTRATIONS OF STRONTIUM 90

INFORMAL DISCUSSION OF JULY 29, 1957

At the suggestion of the Joint Committee on Atomic Energy, the Division of Biology and Medicine, U. S. Atomic Energy Commission, invited the principal participants in discussion of this subject at the recent Congressional Hearings, May 27–June 6, 1957, to meet in Washington, D. C., July 29, to try to reduce the degrees of uncertainty involved in various predictions of future skeletal concentrations of strontium 90 in humans. These persons were: Mr. Merril Eisenbud, Dr. J. Laurence Kulp, Dr. Wright H. Langham, Dr. Willard F. Libby, Dr. Lester Machta, Dr. William F. Neuman and Dr. Walter Selove. In addition to these participants were: Dr. Charles L. Dunham who alternated with Dr. Libby as Chairman of the discussion, Dr. Lyle T. Alexander, Mr. Hal Hollister, Dr. J. Calvin Potts, Dr. Robert Reitemeier, and the author of this summary, Forrest Western. This summary is based on a stenographic transcript and has had the benefit of comments by most of the participants.

It was generally agreed that the extensive measurements by Dr. Kulp of concentrations of strontium 90 in human skeletons established one base from which one may extrapolate skeletal concentrations of strontium 90 to be expected in the future. Dr. Kulp stated that the average concentration in children of the northeastern United States in the fall of 1956 was about 0.8 micromicrocuries of strontium 90 per gram of calcium. In discussion of relationships between concentrations in the United States and other parts of the world, Dr. Kulp indicated that concentrations in children of northeastern United States are consistent with those of two widely separated areas in other parts of the world from which he has been able to obtain a considerable number of samples. Dr. Selove discussed reports of relatively high local concentrations of fallout in an Asiatic area and suggested that possible fluctuations in local patterns of tropospheric fallout in the periods shortly following tests explosions might result in considerably higher skeleton concentrations in some areas of the world. Although our world sampling program to date has failed to disclose areas in which skeletal concentrations are higher than in the United States, individual comments endorsed the desirability of continued search for such areas as a part of our world-wide study of the distribution and uptake of fallout from nuclear detonations.

It was agreed that, *even if fallout had ceased at the end of 1956*, skeletal concentrations of strontium 90 would be expected to increase until they came into equilibrium with the strontium 90 in the environment. Somewhat independent estimates of the equilibrium value may be made (1) from a knowledge of *changes* in environmental (specifically, *dietary*) concentrations during the growth of the skeletons assayed, and (2) by application of factors of discrimination between calcium and strontium in estimating the uptake of strontium 90 from existing concntrations of strontium 90 (a) the soil, (b) the over-all diet, or (c) milk. Each of these methods involves some degree of uncertainty. After discussion of the uncertainties involved, it was agreed that these various considerations make it appear probable that average skeletal concentrations to be expected in young persons of northeastern United States as a result of strontium 90 actually deposited on the earth's surface up to the end of 1956, fall between 1.5 and 2 micromicrocuries per gram of calcium.

Because some of the strontium 90 released to the stratosphere in past years has not yet been deposited on the earth's surface, actual skeletal concentrations from tests performed before the end of 1956 may be expected to become greater than the values estimated under the conditions assumed above. The increase in concentration to be expected from additional fallout of this material depends upon a number of factors; (1) the additional activity reaching the surface of the earth, (2) the fact of delay in the appearance of additional strontium in the diet, (3) the relative importance of total soil content and rate of fallout (specifically, rate of retention on surfaces of vegetation), (4) radioactive decay and (5) decrease, with time after fallout, in the percentage of strontium 90 in the soil which is available for uptake by plants. It appeared from the discussion that the greatest uncertainty involved here is in predicting the distribution and time of fallout to be expected from residual stratospheric content.

It was estimated that, in addition to the range of uncertainty by a factor of 1.25 represented in the above estimate of the average skeletal concentrations to be expected from strontium 90 actually deposited on the earth's surface by the fall of 1956, there might be an additional range of uncertainty, by a factor as large as two, in estimates involving the ratio of the quantity of strontium 90 in the stratosphere to that on the ground. In estimating total skeletal concentrations in the northeastern United States which might be expected when fallout of strontium 90 produced prior to 1957 is essentially complete, these two uncertainties alone would result in a range of a factor of 2.5 between minimum and maximum estimates.

It was agreed that the first effort of the group would be to estimate average skeletal concentrations to be expected in the age group of maximum concentration in 1975, assuming that there were no nuclear detonations after 1956. Dr. Kulp estimated that, because of radioactive decay and decrease in availability, additional fallout of strontium 90 produced before 1957 would not make environmental concentrations of strontium 90 in the northeastern United States in 1957 significantly greater than in 1956. After discussion, this led to the estimate that, if the residual stratospheric content were to be deposited with uniform distribution over the surface of the earth, skeletal concentrations in the age group of maximum concentration in 1975 would fall in the range of from 1.5 to 3.5 micromicrocuries of strontium 90 per gram of calcium.

Mr. Eisenbud, taking a different approach, first estimated quantities of strontium 90 on the surface in the northeastern United States at the end of 1956 due to tropospheric and stratospheric fallout, respectively, and the fraction of strontium 90 previously injected into the stratosphere which had fallen out by the end of 1956. Maximum future environmental levels were then related to those of 1956, using the assumption that fallout of the residual (1956) stratospheric content would have the same geographic distribution as the previous stratospheric fallout. Discussion of this approach led to an estimate of skeletal concentrations, in 1975, in the range from 2 to 5 micromicrocuries of strontium 90 per gram of calcium.

Dr. Machta discussed the possibility that strontium 90 injected into the stratosphere near the equator may be moving northward and entering the troposphere preferentially above 30° N., in such a manner that the fraction of the stratospheric content falling out in these latitudes is increasing with time. It was estimated that such "banding" of fallout might result in skeletal concentrations two times as high as those estimated on the basis of Mr. Eisenbud's assumption of a constant stratospheric fallout pattern; i. e., an average skeletal concentration in children of from 4 to 10 micromicrocuries per gram of calcium.

It was agreed that at this time our knowledge of atmospheric transport is too limited to reduce the range of uncertainty on this point represented by the ranges of estimated concentrations described in the preceding three paragraphs.

It was estimated that, if testing of weapons were continued up to about 1965 in such a manner as to produce strontium 90 in the same total quantity and with the same distribution (i. e., with tests held at the same geographical locations and with the same distributions of tropospheric and stratospheric fallout) as were produced prior to 1957, the average total skeletal content in young persons in about 1975 might be approximately 2.5 times that to be expected if there were no further testing. This factor is considered to apply equally to the three estimates discussed in the preceding paragraphs.

The estimates described above are summarized in the following table:

Predicted average skeletal levels of strontium to be expected in young persons in northeastern United States under various conditions of testing of nuclear weapons

[All levels are in micromicrocuries of strontium 90 per gram of calcium]

Concentrations in children, fall 1956	Predicted future concentrations from strontium 90 on ground before 1957	Predicted concentrations in 1975 from all strontium 90 produced before 1957	Predicted concentrations in 1975 if past tests or equivalent were repeated before 1965
0.8	1.5 to 2	1.5 to 3.5*	3.5 to 9.*
		2 to 5	5 to 12.
		4 to 10	10 to 25.

*The considerations leading to each of these 3 estimates are discussed in the text of this summary.

It was the consensus of the group that within one or two years the confidence with which predictions of future concentrations can be made will be so greatly increased that it is unprofitable for the present purpose to extend predictions beyond those given in the above table.

APPENDIX 12

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington 25, D. C., June 1, 1957.

Hon. CHET HOLIFIELD,
Chairman, Special Subcommittee on Radiation, Joint Committee on Atomic Energy, Congress of the United States.

DEAR MR. HOLIFIELD: Your letter of 31 May 1957 requests a letter on an unclassified basis giving: (a) an estimate as to year-to-year fission yield and total yield put into the atmosphere by atomic weapons from all sources; (b) a list of weapons test series conducted by each country; and (c) a listing of the number of explosions which have taken place, country by country and year by year.

As to total yield and total fission yield released by tests to date either in gross or by nation, this information has security implications which preclude my giving it as unclassified data. Various unclassified estimates which do not reveal such totals have been made as to the amount of fission debris making its way into the stratosphere. One such estimate is that given on pages 952 and 954 of the attached report, "Current Research Findings on Radioactive Fallout," by Dr. Willard F. Libby.

Information as to U. S. test series and the number of shots in each is contained in the following table:

Name of series	Date	Number of shots
Trinity	1945	1
Crossroads	1946	2
Sandstone	1948	3
Greenhouse	1951	4
Ranger	1951	5
Buster/Jangle	1951	7
Tumbler/Snapper	1952	8
Ivy	1952	2
Upshot/Knothole	1953	11
Castle	1954	(1)
Teapot	1955	14
Wigwam	1955	1
Redwing	1956	(1)
Plumbbob	1957	[2] 1

[1] The number of shots in Castle and Redwing is still classified.
[2] Only 1 shot to date.

The above list is exclusive of the two weapons detonated in combat during World War II.

With respect to the British series, the United Kingdom has announced the following series and number of shots:

Location	Date	Number of shots
Australia	1952	1
Do	1953	2
Do	1956	2
Do	1956	4
Christmas Island	1957	2

Of several Soviet references to their tests, they have: announced one on 20 August 1953, shortly before the U. S. announcement was issued; made a general statement on 18 September 1953 to the effect that they had conducted several tests in recent weeks which constituted confirmation of the U. S. announcement of 31 August 1953; and announced a test on 10 September 1956 about which the U. S.

made no comment. The United States has from time to time announced certain of the tests. Copies of these announcements are attached. From a review of these announcements it can be noted that the U. S. has announced 23 specific USSR detonations. A complete list of all detected shots cannot be provided on an unclassified basis. In this respect on August 26, 1956, the Chairman of the AEC stated: "Although there have been but 13 announcements by the U. S. regarding Soviet testing, several have noted a series of detonations and the actual number of Soviet detonations is significently higher than 13."

Should you desire a classified summarization of the information requested which cannot be given on an unclassified basis, I would be most pleased to provide this separately.

Sincerely yours,

K. E. FIELDS,
General Manager.

[From Congressional Record, June 18, 1957]

NUCLEAR WEAPONS EXPLOSIONS

The following table has been compiled principally from press releases of the United States Atomic Energy Commission. However, reports in the press as to the size and nature of various explosions have been included when available.

On April 12, the United States had announced 19 Soviet tests. The AEC has pointed out that this country does not disclose all of the U. S. S. R. shots of which it has knowledge but limits itself to statements about explosions of special interest. The actual number of Soviet detonations is therefore significantly higher than those announced.

As of June 17 the AEC had announced 68 tests by the United States. However, the total number of detonations made by this country has never been announced.

The United Kingdom has announced 11 tests to date, June 18, which is understood to be the total number of tests made by that country.

Year	United States		U. S. S. R.		United Kingdom		Remarks
	Date	Place	Date	Place	Date	Place	
1945	July 16	Alamogordo					1st test of A-bomb.
	Aug. 6	Hiroshima					Air burst, energy release about 20,000 tons of TNT.
	Aug. 9	Nagasaki					Do.
1946	July 1	Operation Crossroads, Bikini Lagoon.					Air burst, Nagasaki-type bomb.
	July 25						Underwater detonation.
1948 (spring)		Operation Sandstone, Eniwetok Atoll.					3 explosions announced May 17, no details given.
1949			Sept. 23	Soviet territory			1st atomic explosion in U. S. S. R. announced by President Truman.
1951 (winter)	Jan. 27 Jan. 28 Feb 1 Feb. 2 Feb. 6	Operation Ranger, Nevada Flats.					5 tests primarily for tactical information, Air bursts.
1951	April and May	Operation Greenhouse, Eniwetok.					4 tests described as "experiments contributing to thermonuclear research."
			Oct. 3	Soviet territory			2d explosion. Reported by the United States, later confirmed by the Kremlin.
			Oct. 22	do			President Truman announced evidence of a 3d nuclear explosion by the U. S. S. R.
1951	Oct. 22 Oct. 28 Oct. 30 Nov. 1 Nov. 5 Nov. 19 Nov. 29	Operation Buster Jangle, Nevada Flats.					7 air, tower, and surface or underground bursts. Low yield. Tactical.
1952	Spring and summer Apr. 1 Apr. 15 Apr. 22 May 1 May . May 25 June 1 June 5	Operation Tumbler Snapper, Nevada Flats.					8 air and tower bursts. Troop participation in some tests.
	Fall	Operation Ivy, Eniwetok					2 tests. 1 was the 1st hydrogen bomb ever exploded. Yield 5 megatons.
					Oct. 3	Monte Bello Islands.	Nature of bomb not disclosed. Probably of Hiroshima type.

Year	United States		U. S. S. R.		United Kingdom		Remarks
	Date	Place	Date	Place	Date	Place	
1953	Mar. 17						
	Mar. 24						
	Mar. 31						
	Apr. 6						
	Apr. 11	Operation Upshot Knothole, Nevada Flats.					11 tests: Air, tower, and 1 shot from a 280-millimeter gun. The largest explosion, that of Apr. 18, was reportedly equal to 30,000 to 40,000 tons of TNT.
	Apr. 16						
	Apr. 25						
	May 8						
	May 19						
	May 29						
	June 4						
			Aug. 12	Soviet territory			Initial hydrogen bomb (nondeliverable).
			Aug. 31	[1] do			Fission explosion in same range of energy release as Nevada Flats tests.
1954	Mar. 1	Operation Castle, Bikini					Hydrogen bomb reported to have had a yield of 15 megatons. Radioactive fallout over 7,000 square miles.
	Mar. 26						No information released concerning this test.
	Apr. 6	Eniwetok					3d test in the series. Presumably an H-bomb.
			Oct. 26	Soviet territory			Announcement made by the AEC of a series beginning in September.
1955	Feb. 18						
	Feb. 22						
	Mar. 1						
	Mar. 7						
	Mar. 12						
	Mar. 22						
	Mar. 23	Operation Teapot, Nevada Flats.					14 air, tower, and underground tests. Included atomic trigger for detonating H-bombs, also civil defense test and "air-killer" test.
	Mar. 29 (2 tests)						
	Apr. 6						
	Apr. 9						
	Apr. 15						
	May 5						
	May 15						
	May 17	Operation Wigwam, off west coast.					Small underwater atomic weapon exploded, probable yield 1,000 to 5,000 tons of TNT.
			Aug. 4	Soviet territory			AEC announced that the Soviets had resumed testing of nuclear weapons.
			Sept. 24	do			Announcement by AEC of another Soviet nuclear explosion.
					Oct. 15	Woomera Rocket Range, Australia.	2 tests, the 1st equivalent to 20,000 tons of TNT. No data for the 2d test.
			Nov. 10	Soviet territory			Announcement by AEC of a further test in the 1955 series.

			Nov. 23	do			Hydrogen bomb in the range of megatons. Khrushchev announced on Nov. 27 that it was a hydrogen bomb and that it had been dropped at a great height from an airplane.
1956			Mar. 21	Soviet territory, probably central Asia.			AEC announced another Soviet nuclear explosion.
			Apr. 2				Another explosion in current series announced by AEC.
	May 5	Operation Red Wing, Eniwetok.					Relatively small device exploded, about 10 kilotons.
					May 16	Monte Bello Islands.	Test expected to supply data for triggering of H-bomb.
	May 21	Bikini					1st H-bomb dropped by U. S. Air Force. Yield at least 15 megatons.
			Aug. 24	Southwestern Siberia.			A series of small explosions.
			Aug. 30				
			Sept. 2				
					Sept. 27	Maralinga Proving Ground, South Australia.	4 tests, tower and air; new and cheaper kinds of A-bombs tested.
			Nov. 17	Southwestern Siberia.			Small explosion.
			Jan. 19	do			Moscow announced these tests were designed to perfect atomic warheads for tactical purposes.
			Mar. 8				
			Apr. 3				
			Apr. 6				
			Apr. 10				
			Apr. 12				
			Apr. 16				
					May 15	Christmas Islands.	H-bomb dropped from Vikers Valiant jet bomber. Energy release 1,000,000 tons of TNT.
	May 28	Beginning of Operation Plum Bob, atomic test site, Nevada. The tests are scheduled to continue into September.					Probably a warhead for small rockets or missiles. Yield about 10,000 tons of TNT.
					May 31	Christmas Islands.	2d hydrogen bomb exploded by the British.

[1] 1 of a series.

Prepared for the use of the Subcommittee on Disarmament by Janie E. Mason, of the subcommittee staff, on loan from the Library of Congress, June 6, 1957.

X